CHAPTER 1

Introduction and Overview

Posttraumatic stress disorder (PTSD) was officially introduced into the psychiatric nomenclature in 1980, with the publication of the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition* (*DSM-III*) by the American Psychiatric Association (APA). Over the past 30 years, this disorder has changed the landscape of trauma and general stress studies, and it has contributed to the development of a wide range of sociopolitical, conceptual, and clinical issues and questions. The concept of posttraumatic reactions has been widely absorbed by the general public with regard to psychological adjustment after major traumatic events, as well as a wide range of other life stressors. Public awareness of PTSD has been furthered by extensive news coverage of recent national and international news events, including the 9/11 atrocities; the wars in Iraq and Afghanistan; terrorist attacks in London and Madrid; hurricanes, earthquakes, tsunamis, and other natural disasters; widely publicized cases of child sexual abuse among church officials and national sports heroes; and genocides in Africa and eastern Europe. Mainstream media publicity and films since the Vietnam War have also likely played a role in the layman's understanding of PTSD. A now common portrayal of returning combat veterans is that of the psychologically impaired victim-hero, found in Hollywood movies too numerous to count, such as *Taxi Driver* (1976), *Coming Home* (1978), *The Deer Hunter* (1979), *Born on the Fourth of July* (1989), and *In the Valley of Elah* (2007).

Undoubtedly, research on every nook and corner of this disorder has abounded in the past few decades, providing patients and their families,

clinicians, health care administrators, and policy makers with a vastly better understanding of posttraumatic reactions—from both a psychopathological and a resilient standpoint—than we had before. Despite this expansion of our knowledge, there remain numerous unanswered questions and controversies as to how clinicians should evaluate, define, and treat psychiatric symptoms in the aftermath of trauma events. Clinicians are left in their practices with questions that lack practical directions based on solid empirical evidence. A good deal of pseudoscience and poor science in the field of traumatology, as well as a proliferation of clinical myths, misconceptions, and fads about traumatic reactions, has further muddied the water for clinicians. Toward this end, we have produced a volume intended to help the dedicated evidence-based mental health practitioner with clinical assessment and treatment planning for people—patients, clients, consumers, however one wishes to term or define them—suffering from posttraumatic reactions.

This opening chapter begins with a historical perspective on posttraumatic reactions, segues into a brief review of important conceptual questions and issues with regard to the diagnosis of PTSD, and concludes with a general overview of evidence-based practice in mental health care and a discussion of why such practice is so important for people suffering posttraumatic reactions.

Historical and Societal Perspectives

The general notion that people are significantly affected, perhaps changed forever, by violent and horrific ordeals is not new. The postcombat reactions of warriors have been noted since ancient times throughout mythology and literature and in a variety of cultures (e.g., "Epic of Gilgamesh," writings of Homer and Shakespeare). Since the 19th century, different terms have been used to describe posttraumatic reactions for a variety of dangerous and frightening experiences. Frequently, these terms provided clues as to how the etiology or nature of the symptoms was viewed at the time. In the years following the U.S. Civil War, it was noted that many veterans reported symptoms of chronic chest pain, as well as fatigue, shortness of breath, and heart palpitations—yet physical abnormalities to explain these symptoms were often not to be found. Physicians

and caretakers were puzzled by the syndrome, which, while somewhat common, had no obvious explanation. The observed functional syndrome became known as *soldier's heart* or *Da Costa's syndrome* for the surgeon who noted it in a series of case reports (Barnes, 1870). During World War I, the term *shell shock* was used to refer to a constellation of symptoms believed to be a neurological disorder caused by the sound of explosions and bright flashes of light from bursting artillery shells on the Western Front. *Combat fatigue* was a term used during World War II, when it was believed that combat reactions were caused by exposure to extreme stress and fatigue. During the 1970s, victims of sexual assault were often identified as suffering from a "rape trauma syndrome" (Burgess & Holmstrom, 1974) or "battered woman syndrome" (Walker, 1977).

In organized psychiatry, the concept of a specific category of life events causing a psychiatric disorder was first formalized in 1952, with the first edition of the *DSM*. Gross stress reaction (GSR) was defined as a "transient situational personality disorder" that could occur when essentially "normal" individuals experienced severe physical demands or extreme emotional stress. GSR was soon dropped from psychiatry's nosology in 1968, with the second edition of the *DSM*. Twelve years after the *DSM-II*'s publication, a more narrow class of events—that is, trauma—was linked as the causative agent for a new and specific constellation of symptoms, and posttraumatic stress disorder was formally defined and included in the psychiatric nosology.

The diagnosis of PTSD, added to the *DSM* in 1980, was largely the result of attempts to account for the challenging impairment presented by Vietnam veterans at the time of their homecoming (Satel & Frueh, 2009; Shephard, 2001, 2004; Wessely & Jones, 2004). In the immediate post-Vietnam era, compensation for significant functional impairment was difficult to obtain other than for observable physical injuries and access to Veterans Administration (VA) medical services were possible only via a "war-related" disorder (Wessely & Jones, 2004). Veterans' advocates and antiwar activities were at the forefront of efforts to define and codify a "Vietnam syndrome," a psychiatric response to war unique among Vietnam veterans. At about the same time in the 1970s, the feminist movement was politically strong and gaining momentum. They successfully began to expose the violence that was common against women, which led to the

development of a "rape trauma syndrome" (Burgess & Holmstrom, 1974). These two activist groups soon joined forces to make a common cause with mental health clinicians and researchers. Together, they worked to influence the development of the new diagnostic addition to the *DSM-III*. Thus, along with medical and psychological science, the development of the clinical conceptualization of PTSD was heavily influenced by socio-economic and political forces (Mezey & Robbins, 2001; Shephard, 2001). For a more in-depth discussion on the origins of PTSD, see Shephard (2001), Summerfield (2001), Jones and Wessely (2007), Rosen and Frueh (2010), and Satel and Frueh (2009).

A Unique Psychiatric Disorder

PTSD is unique from the vast majority of psychiatric disorders in the *DSM* in that it is the rare disorder to include an etiological explanation (trauma)—and one that is actually part of its diagnostic criteria (Criterion A). Otherwise, for the most part, the *DSM* takes an atheoretical approach to quantifying and classifying mental disorders. The fact that PTSD cannot be diagnosed without the occurrence of a Criterion A event not only makes PTSD distinct from other psychiatric diagnoses, but it also renders it unique in the general field of stress studies (Breslau & Davis, 1987). As Rosen, Frueh, Elhai, Grubaugh, and Ford (2010) noted:

> Rather than all stressors creating an increased risk for a wide range of established conditions, there now was a distinct class of stressors that led to its own form of psychopathology. Thus, while any type of high stress could lead to increased risk of headaches, high blood pressure, or depression, only a Criterion A event such as combat, rape, or a life-threatening accident could lead to the distinct syndrome of PTSD. This assumption of a specific etiology, associated with a distinct clinical syndrome, provided the justification for a new field of "traumatology" to be carved out of general stress studies. (p. 7)

An Evolving Disorder

In the 30-plus years since its creation, the definition of PTSD has evolved steadily with each new revision of the *DSM*. Changes have been made to

the definition of Criterion A, new symptoms have been added, and requirements regarding symptom onset and duration (Criterion E) have been modified.

In *DSM-III* (APA, 1980) the trauma criterion (Criterion A) was defined as: "Existence of a recognizable stressor that would evoke significant symptoms of distress in almost everyone" (p. 238). This stressor was described it as being outside the range of normal human experience. In *DSM-IV* (APA, 1994), Criterion A events are defined much more specifically:

> The person has been exposed to a traumatic event in which both of the following were present: (1) 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; (2) the person's response involved intense fear, helplessness, or horror. Note: in children, this may be expressed instead by disorganized or agitated behavior. (pp. 427–428)

Moreover, by 1994, empirical research showed that traumatic events were actually quite common to the human experience, so that aspect of the *DSM-III* definition was dropped from the definition.

The number of possible PTSD symptoms expanded from 12 in *DSM-III* to 17 in *DSM-IV* and included the addition of avoidance behaviors. Also added was the caveat in Criterion E that duration of the disturbance had to exceed 1 month. This revision to Criterion E in *DSM-IV* was paired with a new (but related) diagnosis: acute stress disorder (ASD). Like PTSD, ASD requires a Criterion A traumatic event and includes symptom criteria very similar to PTSD (Criteria B through D). ASD cannot be diagnosed unless symptoms and associated impairment lasts at least 2 days (so as to exclude those with immediate peritraumatic reactions, which are very common) and may not last more than 4 weeks past exposure to the traumatic stressor. In this way, ASD serves as a diagnosis for those suffering extreme traumatic stress reactions that occur too soon after trauma exposure to be classified as PTSD.

PTSD's defining criteria continue to be subject to much debate and discussion, and are likely to be revised in future editions of the *DSM*. We will return to further discussion of the future of PTSD in Chapter 12.

Important Conceptual Questions and Issues

Despite the proliferation of research on and expansion of clinical services for PTSD, much about the disorder remains misunderstood or unknown. In the sections that follow, we briefly introduce some of the important conceptual questions and issues regarding the diagnosis of PTSD that have implications for clinical assessment and treatment planning for people with significant traumatic event exposure.

Epidemiology of PTSD: Posttraumatic Morbidity Versus Resilience

There is now a widespread assumption on the part of many laypersons, journalists, and even clinicians that the majority of people who endure a traumatic experience, such as sexual assault, childhood abuse, a natural disaster, or wartime combat, will develop a psychiatric disorder as a direct result of the experience. However, the data actually tell a very different story. Most individuals will experience some short-term distress and may be affected in a variety of psychological ways. However, the majority of people who survive even the most horrific traumatic experiences do not develop PTSD or any other full-blown psychiatric disorder. That is, only a small minority of people will develop distress and functional impairment that rises to the level of a psychiatric disorder. Instead, long-term resilience is actually the norm rather than the exception for people after trauma (Bonanno, Westphal, & Mancini, 2011).

We now have a large body of epidemiological studies, conducted across a variety of populations, to inform our understanding of PTSD prevalence estimates (Breslau, Davis, Andreski, & Peterson, 1991; Davidson, Hughes, Blazer, & George, 1991; Dohrenwend et al., 2006; Norris, 1992; Smith et al., 2008). Data consistently show that exposure to potentially traumatic events (i.e., Criterion A) is quite common in the general population, with 60% to 80% of the population reporting exposure to various types of traumatic events (Breslau et al., 1991; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). PTSD rates (point-prevalence) are consistently found to be in the 6% to 9% range for both civilians and military veterans. For those who develop PTSD, about 50% will remit within 3 months without

treatment (Galea et al., 2002; Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992). This is a robust finding that is specifically noted in *DSM-IV.* Individuals who are diagnosed with PTSD are at 3 times greater risk of meeting criteria again if exposed to a later traumatic stressor (Breslau, Peterson, & Schultz, 2008). In this way, PTSD can be a recurrent disorder after a first episode.

Psychiatric Comorbidity

Epidemiological studies also indicate that PTSD is not the only, or even the most likely, psychiatric reaction to follow trauma exposure. Fear, anxiety, sadness, anger, and guilt (among others) are common reactions to traumatic experiences. Other common reactions include physical or somatic complaints, such as insomnia, gastrointestinal symptoms, headaches, or sleep problems; social and relationship difficulties; and substance use, including alcohol and nicotine (Breslau et al., 1991; Bryant, 2010; Kessler et al., 1995). Major depression is probably the most common form of posttraumatic psychopathology, even more prevalent than PTSD. Moreover, among individuals who meet the diagnostic criteria for PTSD, a majority has additional psychiatric problems. Common co-occurring disorders with PTSD include depression, substance use disorders, panic attacks, and other anxiety disorders. Adding to the illness burden, people diagnosed with PTSD often present with significant medical comorbidity (e.g., chronic pain, cardiac difficulties) that is associated with increased use of health care services (Elhai, North, & Frueh, 2005; Schnurr & Green, 2004).

Individual Risk Factors

All of this raises the question: If only a small percentage (6% to 9%) of trauma-exposed people have PTSD, who is most likely to have it? A number of individual vulnerabilities and risk factors have been shown to be strong predictors of PTSD. Social support plays perhaps one of the most important buffering roles against psychiatric illness in the aftermath of trauma and stress. The converse of this is also true in that lower social support is associated with increased risk for PTSD (Andrews, Brewin, & Rose, 2003). Gender is another consistent risk factor. Females are at markedly increased risk of developing PTSD relative to men by a ratio of

approximately two to one (Breslau et al., 1991, 1998; Tolin & Foa, 2006). Other significant PTSD risk factors are lower intelligence, lower education, lower socioeconomic status, prior history of poor social adjustment or psychiatric disorders, and substance abuse. Recent research also suggests that certain genetic phenotypes may interact with environmental stressors to affect the likelihood of PTSD after trauma (Koenen, 2007). At this point, genetic research on PTSD and other posttraumatic reactions is quite underdeveloped. This will undoubtedly change over the next 5 to 10 years, but it is unlikely that genetic variations will ever explain more than 35% of the risk for PTSD development.

The Etiological Nature of the Trauma

Inherent in the internal logic of the PTSD diagnosis is that trauma (Criterion A) causes the symptoms of the disorder. The belief in a specific etiology was fundamental to the very origins of PTSD, and provided the rationale to create a new psychiatric disorder that formed a unique class of stressors from more general life stressors. Accordingly, individuals who do not experience a traumatic event should not develop PTSD. However, empirical data has consistently shown otherwise—that non–Criterion A stressors (i.e., events not considered to be traumatic) can result in similar rates of PTSD (reviewed in Long & Elhai, 2009; Rosen & Lilienfeld, 2008). In fact, the assumption of a specific etiology for PTSD is so problematic, from a theoretical and empirical standpoint, that some investigators are starting to write about the "the Criterion A problem" (Weathers & Keane, 2007). This obviously has implications for our understanding and definition of the disorder, including how the field will characterize it in future editions of the *DSM*. Recent proposals have suggested everything from tighter definitions of what constitutes a traumatic event (Kilpatrick, Resnick, & Acierno, 2009) to the extreme of doing away with the gatekeeper function of Criterion A altogether (Brewin, Lanius, Novac, Schnyder, & Galea, 2009). At this point, it is somewhat of an open question as to whether "traumatic" stressors are in fact significantly unique and different from other life stressors (divorce, job loss, financial difficulty), or are better understood as points along a

continuum of stressors (Dohrenwend, 2010). In the future, the field of trauma may converge with that of general stress studies.

PTSD Symptom Criteria: Symptom Overlap and Factor Structure

Another important question in the trauma field is: To what extent is PTSD a unique psychiatric disorder, and to what extent does it simply duplicate other diagnoses in the mood and anxiety disorders categories (McHugh & Treisman, 2007; Spitzer, First, & Wakefield, 2007)? This is not simply a lofty philosophical question. The answer has practical implications for our ability to understand and treat the syndrome of clinical symptoms that currently make up the disorder. Not only is PTSD highly comorbid with other *DSM* psychiatric disorders, but it also shares many symptoms with these other disorders. For example, criteria for diagnosing PTSD can be fully met with the right combination of symptoms pulled from the combined diagnoses of depression and specific phobia. See Rosen, Lilienfeld, Frueh, McHugh, and Spitzer (2010) for a detailed explanation for how this can be achieved. An additional related concern is that the core set of PTSD's symptoms have been demonstrated to be best conceptualized as general dysphoria or distress, common to other mood and anxiety disorders (Simms, Watson, & Doebbeling, 2002; Watson, 2005). Although initial studies on criteria sets that remove PTSD's overlapping symptoms find similar rates of prevalence and comorbidity (Elhai, Grubaugh, Kashdan, & Frueh, 2008; Ford, Elhai, Ruggiero, & Frueh, 2009; Grubaugh, Long, Elhai, Frueh, & Magruder, 2010), these findings do not resolve all concerns.

The factor structure of a disorder can provide us with information about how some symptoms are endorsed in a similar manner to each other and different from how other symptoms are endorsed. Factor analytic studies over the past 15 years have consistently shown that *DSM-IV*'s tripartite PTSD model (reexperiencing, avoidance/numbing, hyperarousal) does not adequately account for PTSD's factor structure (Elhai et al., 2008; Elhai & Palmieri, 2011; Yufik & Simms, 2010). This body of research reveals that two particular four-factor models best represent the PTSD construct across studies: (1) King, Leskin, King, and Weathers's (1998)

model consisting of reexperiencing, effortful avoidance, emotional numbing, and hyperarousal factors; and (2) Simms, et al.'s (2002) model consisting of reexperiencing, effortful avoidance, dysphoria, and hyperarousal. These two models differ only in the placement of three PTSD symptoms: difficulty sleeping (PTSD's symptom D1), irritability (D2), and difficulty concentrating (D3). Symptoms D1 through D3 are part of the King et al. model's hyperarousal factor, but part of the Simms et al. model's dysphoria factor. Very recent confirmatory factor analytic findings demonstrated that separating the three symptoms into a separate factor significantly enhanced model fit for the two models, suggesting that these three symptoms represent a unique latent construct (Elhai et al. 2011).

Memory and Trauma

A common notion in the past was that memory for traumatic events functioned very differently from memory for ordinary experiences. Many clinicians also believed that major traumatic experiences were highly susceptible to being repressed. The explanation for this is that certain events are so horrific that the human mind cannot tolerate them and, therefore, represses them to the unconscious mind. From there, these unconscious traumatic memories cause havoc in the form of severe psychopathology, including symptoms of PTSD, "multiple personalities," borderline personality disorder, and other forms of psychiatric disturbance. This concept dates back to Sigmund Freud, has been the subject of several Hollywood movies (e.g., *Sybil*), and continues to exist today in some quarters. It has been the cornerstone of many high-profile lawsuits over the years, including those brought against the Catholic Church and day care centers. In fact, *DSM-IV* somewhat legitimizes this concept by including it as one of the 17 clinical symptoms of PTSD: "inability to recall an important aspect of the trauma." However, a large body of research from the fields of memory, learning, and cognition has failed to provide any support for this concept of a special mechanism for traumatic memory (McNally, 2003). This understanding has led to several dramatic revelations. For example, a very recent book by an investigative journalist

(Nathan, 2011) thoroughly discredited the case of Sybil, the famous patient with purported multiple personalities caused by childhood trauma (played by Sally Field in the movie). The evidence portrays a patient with other psychiatric disorders who was manipulated by her psychiatrist into reporting and playing out a series of symptoms that she did not actually experience.

Delayed-Onset PTSD

A feature of PTSD that has been included in the *DSM* is the concept of "delayed-onset" PTSD. According to this commonly held view, people can appear to be resilient after trauma but many years later can suffer a sudden onset of PTSD symptoms that is not attributable to any current stressor or illness. The stereotypical example of this is the combat veteran who is overcome suddenly with PTSD symptoms 20 or 30 years after the war is over. However, there is little empirical data to support the existence of delayed-onset PTSD. Several large epidemiological studies have reported zero or extremely low rates of delayed-onset PTSD in civilians and veterans (Breslau et al., 1991; Frueh, Grubaugh, Yeager, & Magruder, 2009). If, however, "delayed onset" is reconceptualized as a delay in seeking treatment, or subsequent exacerbation of prior symptoms by recent stressors occurring years after the original traumatic event, then the phenomenon may be somewhat more common (Andrews, Brewin, Philpott, & Stewart, 2007). One implication of this is that clinicians should be sure to take careful histories regarding a patient's course of symptoms and possible delays in seeking treatment, before applying the diagnostic qualifier of delayed onset.

The Forensic Aspect of PTSD

As indicated earlier, PTSD is one of the very few psychiatric disorders in the *DSM* that by definition includes an etiological criterion: traumatic event exposure. Because of this assumption of a specific cause for psychological suffering and impaired role functioning, the diagnosis of PTSD is commonly invoked among patients with claims for worker's compensation or disability or personal injury lawsuits (Taylor, Frueh, &

Asmundson, 2007). For example, the vast majority (> 90%) of veterans seeking treatment services within the Veterans Affairs (formerly Veterans Administration) system are also applying for disability payments, which creates a wide range of assessment and treatment considerations (Frueh, Grubaugh, Elhai, & Buckley, 2007; Worthen & Moering, 2011). The diagnosis provides an opening in tort litigation for plaintiffs to argue that subjective psychiatric symptoms are the direct result of an alleged traumatic event and not from other life stressors or personal vulnerabilities. Other major psychiatric disorders, including depression, anxiety, and addictions do not easily lend themselves to this. The *DSM-IV* specifically acknowledges this reality with a cautionary guideline for clinicians: "Malingering should be ruled out in those situations in which financial remuneration, benefit eligibility, and forensic determinations play a role" (APA, 1994, p. 467). This is a clinical assessment issue that we will address in a variety of ways in later chapters.

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