PART

I

Core Issues

CHAPTER

1

Posttraumatic Stress Disorder and General Stress Studies

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In the relatively short span of three decades, posttraumatic stress disorder (PTSD) has captured the attention of mental health professionals, their patients, and the public at large. First introduced into the third edition of psychiatry's Diagnostic and Statistical Manual of Mental Disorders (DSM-III; APA, 1980), the diagnosis of PTSD has served as the focus of more than 12,000 studies in peer-review journals. Clinicians have found the diagnosis useful when conceptualizing patients' reactions to horrific and life-threatening events. Finding PTSD of benefit, clinicians have expanded its application in an effort to help patients with a variety of stress issues.

The general public has increasingly applied the "PTSD model" to their understanding of adjustment in the aftermath of trauma. Public awareness of psychiatric posttrauma tic issues has been furthered by extensive news coverage of events around the globe, including terrorist attacks in New York, London, and Madrid; Hurricane Katrina, earthquakes, and other natural disasters; widely publicized cases in America

of child sexual abuse and international stories of child trafficking; mass genocides and other atrocities; and reports on the psychiatric casualties of war, including America's veterans who have fought in Iraq and Afghanistan.

To appreciate why PTSD was introduced in the DSM-III, and to understand the spiraling growth of research and clinical interest, it is instructive to step back and consider the origins from which the diagnosis emerged. By looking at PTSD's origins, its underlying assumptions, and the fruits of three decades of research, clinicians will better understand posttraumatic morbidity and issues surrounding patient care.

HISTORICAL AND SOCIETAL PERSPECTIVES

The field of general stress studies was greatly influenced by the early work of Walter Cannon (e.g., Cannon, 1929) and his proposal that "critical stress" can disrupt the body's homeostatic mechanisms. Later, Hans Selye proposed a General Adaptation Syndrome (Selye, 1936), which conceived of stressors as "etiologically nonspecific." Selye's model held the view that any event of sufficient intensity (i.e., the stressor) was capable of producing a physiological adaptation response (i.e., the syndrome) whose features were constant regardless of event type.

By the mid-1970s, interest in the field of stress studies had grown substantially. This growth was demonstrated by Selye's (1975) estimate that he had more than 100,000 publications in his stress library. At that point in time, the literature had yielded several insights into the nature and effects of stressful life events (B. S. Dohrenwend & B. P. Dohrenwend, 1974a). Research demonstrated that "stressors" created a risk for subsequent illness, both physical and psychiatric. It also had been shown that severe stressors were more likely than mild ones to produce maladaptive responses (Brown, Sklair, Harris, & Birley, 1973; Wyler, Masuda, & Holmes, 1971), although the magnitude or severity of a stressful event was influenced by an individual's subjective appraisals (Lazarus & Alfert, 1964; Lazarus & Folkman, 1984). Research also suggested that the likelihood of a stressor producing psychopathological reactions was influenced

by pre-incident risk factors, such as personality traits, as well as the buffering effects of social support (Andrews, Tennant, Hewson, & Vaillant, 1978; Cobb, 1976; Rabkin & Struening, 1976).

One issue long debated in the stress field concerned the specificity of effects. Selye's model of adaptation was non-specific: It postulated a general physiological response to a diverse set of events. In contrast, others believed that experimental findings brought into question the nonspecificity concept. B. S. Dohrenwend and B. P. Dohrenwend (1974b) stated this alternative view:

> [The] question still to be answered is whether limited domains of possibly stressful life events will be found for some types of disorder, or whether the domain of possibly stressful life events encompasses all life changes for all or nearly all outcomes. The prospect of finding that relatively narrow domains of life events are related to specific disorders is an attractive one, either from a theoretical or a practical perspective that deserves systematic investigation (p. 321).

Traumatic Stressors

The notion that a "narrow domain" of life events could be related to specific disorders is certainly not novel. Warriors' post-combat reactions have been noted throughout literature (e.g., "Epic of Gilgamesh;" writings of Homer and Shakespeare). Nineteenth century concepts of "railway spine" and "traumatic neuroses" were thought to result from high-impact accidents. Oftentimes, a term provided descriptive or explanatory elements for the noted reactions and behaviors. For example, after the U.S. Civil War, it was noted that many military veterans reported somatic symptoms related to chest pain and cardiac functioning. These reactions included *fatigue*, *shortness of breath*, *heart palpitations*, *sweating*, and *chest pain*—yet physical examination revealed no physical abnormalities to explain the symptoms. The observed syndrome was known as "soldier's heart." During and shortly after World War I, "shell shock" referred to a syndrome that was thought to be a neurological disorder caused by exposure to loud booming noises and

Table 1.1 Posttraumatic Reactions: Historical Terms

Accident neurosis	Mediterranean back/disease
Accident victim syndrome	Postaccident anxiety syndrome
Aftermath neurosis	Postaccident syndrome
American disease	Posttraumatic syndrome
Attitudinal pathosis	Railway spine
Battered woman's syndrome	Rape trauma syndrome
Combat fatigue	Secondary gain neurosis
Compensation hysteria	Shell shock
Compensation/profit neurosis	Soldier's heart
Da Costa's syndrome	Traumatic hysteria
Fright neurosis	Traumatic neurasthenia
Greek disease	Traumatic neurosis
Greenback neurosis	Triggered neurosis
Gross stress reactions	Vietnam syndrome
Justice neurosis	Wharfie's back
Litigation neurosis	Whiplash neurosis

bright flashes of sudden light associated with bursting artillery shells. "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. In the 1970s, the concept of event specificity was applied to victims of sexual assault, with the creation of "rape trauma syndrome" (Burgess & Holmstrom, 1974) and "battered woman syndrome" (Walker, 1977). These historical terms and others applied to posttraumatic reactions are listed in Table 1.1. More detailed historical reviews on the precursors of what we now call PTSD have been provided elsewhere (e.g., Ford, 2008; Jones & Wessely, 2005; Satel & Frueh, 2009; Shephard, 2001).

Posttraumatic Stress Disorder

The possible linkage of a specific class of events to psychiatric disorder was raised in 1952, when "Gross stress reaction" (GSR) was introduced in the first edition of the DSM. This condition was defined as a "transient situational personality disorder" that could occur when essentially "normal" individuals experienced severe physical demands or extreme emotional stress, such as in combat or civilian catastrophe. GSR had

a relatively short life span: it was dropped from psychiatry's nosology in 1968, with publication of the DSM's second edition. It was 12 years later, in 1980, that the linkage of a specific class of events to a specific constellation of symptoms was formalized with the introduction of Posttraumatic Stress Disorder (PTSD).

The DSM-III defined traumatic events by Criterion A, and this criterion served a "gatekeeper" role for the diagnosis of PTSD. In other words, PTSD could not be diagnosed without the occurrence of a Criterion A event. Breslau and Davis (1987) observed how this conceptualization rendered PTSD distinct from other psychiatric diagnoses and from the general field of stress studies. 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.

Changing Criteria and Acute Stress Disorder

Criteria that defined PTSD were determined by a DSM-III subcommittee, who were influenced more by theory than empirical data. Committee members considered the observations of Horowitz (1978) on stress response syndromes, the writings of a self-described "psychohistorian" (Lifton, 1961), Kardiner's (1941) construct of a physioneurosis, and issues raised on behalf of the mental health needs of Vietnam veterans (see Scott, 1990; Young, 1995). Appreciating the origins of PTSD, Yehuda and McFarlane (1995) observed how the formulation of the diagnosis "addressed a social and political issue as well as a mental health one" (p. 1706).

With experience, and a growing empirical basis for defining PTSD, multiple changes have occurred in subsequent editions of the DSM (DSM-III-R, APA, 1987; DSM-IV, APA, 1994). For example, the original definition of Criterion A as provided in the DSM-III (APA, 1980)

was a single sentence: "Existence of a recognizable stressor that would evoke significant symptoms of distress in almost everyone" (p. 238). By the time the DSM-IV was published (APA, 1994), Criterion A events were more clearly defined:

> 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 (p. 467).

Symptom criteria that defined the PTSD clinical syndrome also were revised in subsequent editions of the DSM. In the DSM-III, 12 symptom criteria were grouped into 3 clusters (Criteria B through D), representing reexperiencing, numbing of responsiveness, and hyperarousal reactions. With publication of the DSM-IV, 17 symptom criteria were specified, now covering reexperiencing, avoidance and numbing symptoms, and hyperarousal (see Table 1.2).

Table 1.2 DSM-IV Diagnostic Criteria for Posttraumatic Stress Disorder

A. 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.

B. The traumatic event is persistently reexperienced in one (or more) of the following ways:
   1. Recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions. Note: In young children, repetitive play may occur in which themes or aspects of the trauma are expressed.
   2. Recurrent distressing dreams of the event. Note: In children, there may be frightening dreams without recognizable content.
   3. Acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated). Note: In young children, trauma-specific reenactment may occur.

4. Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.
5. Physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:
1. Efforts to avoid thoughts, feelings, or conversations associated with the trauma.
2. Efforts to avoid activities, places, or people that arouse recollections of the trauma.
3. Inability to recall an important aspect of the trauma.
4. Markedly diminished interest or participation in significant activities.
5. Feeling of detachment or estrangement from others.
6. Restricted range of affect (e.g., unable to have loving feelings).
7. Sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span).

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:
1. Difficulty falling or staying asleep
2. Irritability or outbursts of anger
3. Difficulty concentrating
4. Hypervigilance
5. Exaggerated startle response

E. Duration of the disturbance (symptoms in Criteria B, C, and D) is more than 1 month.

F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify if
Acute: if duration of symptoms is less than three months.
Chronic: if duration of symptoms is three months or more.
With Delayed Onset: if onset of symptoms is at least six months after the Stressor.

Source: Reprinted with permission from the *American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders*, 4th Edition, Text Revision (DSM-IV-TR). American Psychiatrics Association, 2000, pp. 467–468.

In DSM-III, a diagnosis of PTSD included Criterion E, which specified the course of posttraumatic reactions. The original form of Criterion E for acute PTSD stated: "Onset of symptoms within six months of the trauma" (p. 238). Over time, clinicians realized that this provision was problematic, because most people have significant reactions in the aftermath of trauma, even in the absence of any psychiatric disorder. To avoid widespread confusion between essentially normal reactions to adversity, and symptoms of psychiatric disorder, Criterion E was modified in

the 1987 revision of the DSM (DSM-III-R; APA, 1987). At that time Criterion E specified, "Duration of the disturbance (symptoms B, C, and D) of at least one month" (p. 251).

Yet, the requirement that symptoms had to persist for at least one month raised its own concerns. This new statement of Criterion E left open the question of how to characterize individuals with unusually severe symptoms in the immediate aftermath of trauma. To address this concern, the fourth edition of the DSM introduced the diagnosis of Acute Stress Disorder (ASD; DSM-IV; APA, 1994). Like PTSD, the diagnosis of ASD required a Criterion A event, and it contained symptom criteria similar to those of PTSD. However, ASD included a separate criteria groupings for symptoms of dissociation (which were not included in PTSD) and emotional numbing (which was grouped with avoidance symptoms in PTSD's Criterion C). ASD cannot be diagnosed unless the symptoms and impairment last at least two days (to exclude immediate "peritraumatic" reactions which are relatively normative) and may not last beyond four weeks following exposure to a traumatic stressor (see Table 1.3). Thus, ASD serves as a means of identifying extreme traumatic stress reactions that occur too soon after trauma to be diagnosed as PTSD.

Changes in PTSD's defining criteria illustrate how various issues regarding posttraumatic reactions and psychiatric diagnoses remain in flux. Even now, there are numerous debates about how PTSD should be defined in the fifth edition of the DSM, whose publication is expected in or around 2013. There also are debates about whether ASD should be dropped in the DSM-V, because of empirical findings that fail to support its underlying assumptions (Bryant, 2004). That these kinds of debates continue should not be unexpected, as traumatology is a young field that emerged only three decades ago. Nevertheless, changes in PTSD criteria raise important issues that we will return to later.

EPIDEMIOLOGY OF TRAUMATIC EVENTS AND POSTTRAUMATIC SYMPTOMS

Since the introduction of PTSD in the DSM-III, much has been learned about the nature and course of posttraumatic reactions. Consider that the DSM-III, back in 1980, had this to say about the important topic of

Table 1.3 DSM-IV Diagnostic Criteria for Acute Stress Disorder

A. Traumatic event exposure [As specified for PTSD]
B. Either while experiencing or after experiencing the distressing event, the individual has three (or more) of the following dissociative symptoms:
   1. A subjective sense of numbing, detachment, or absence of emotional responsiveness
   2. A reduction in awareness of his or her surroundings (e.g., "being in a daze")
   3. Derealization
   4. Depersonalization
   5. Dissociative amnesia (i.e., inability to recall an important aspect of the trauma)
C. The traumatic event is persistently reexperienced in at least one of the following ways: recurrent images, thoughts, dreams, illusions, flashback episodes, or a sense of reliving the experience; or distress on exposure to reminders of the traumatic event.
D. Marked avoidance of stimuli that arouse recollections of the trauma (e.g., thought, feelings, conversations, activities, places, people).
E. Marked symptoms of anxiety or increased arousal (e.g., difficulty sleeping, irritability, poor concentration, hypervigilance, exaggerated startle response, motor restlessness).
F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or impairs the individual's ability to pursue some necessary task, such as obtaining necessary assistance or mobilizing personal resources by telling family members about the traumatic experience.
G. The disturbance lasts for a minimum of two days and a maximum of four weeks and occurs within four weeks of the traumatic event.
H. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition, is not better accounted for by Brief Psychotic Disorder, and is not merely an exacerbation of a preexisting Axis I or Axis II disorder.

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prevalence: "No information." These two words are a striking reminder that committee members back in 1980 framed PTSD's defining criteria without the benefit of empirical data.

By 1994 and publication of the DSM-IV, a large body of literature informed clinicians on posttraumatic reactions and the prevalence of PTSD (e.g., Breslau, Davis, Andreski, & Peterson, 1991; Davidson, Hughes, Blazer, & George, 1991; Norris, 1992). This is how the issue of prevalence is discussed in the most recent edition of the DSM (DSM-IV-TR; APA, 2000).

> Community-based studies reveal a lifetime prevalence for Posttraumatic Stress Disorder of approximately 8% of the adult population in the United States. Information is not currently available with regard to the general population prevalence in other countries. Studies of at-risk individuals (i.e., groups exposed to specific traumatic incidents) yield variable findings, with the highest rates (ranging between one-third and more than half of those exposed) found among survivors of rape, military combat and captivity, and ethnically or politically motivated internment and genocide (p. 466).

Epidemiological studies also find that exposure to potentially traumatic events (Criterion A) is actually quite common, with 60 to 80% of the population reporting exposure to various types of traumatic events (e.g., Breslau et al., 1991; Kessler et al., 1995).

Studies find that people typically react in the immediate aftermath of trauma, with symptoms developing within days of the event (e.g., North, 2001). Among those individuals whose reactions are of sufficient severity and duration that they meet criteria for PTSD, upwards of 50% improve within three months without treatment (e.g., Galea et al., 2002; Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992). This finding is so robust, across a variety of trauma types, that the DSM-IV specifically notes the pattern. Consequently, chronic PTSD (defined as symptoms lasting more than six months) is more uncommon than acute presentations (Yehuda & McFarlane, 1995). Of note, individuals who receive a diagnosis of PTSD are at three times greater risk of again meeting criteria if exposed to a later traumatic stressor, as compared with those who did not develop PTSD in the first instance (Breslau, Peterson, & Schultz, 2008). Thus, PTSD can be a recurrent disorder once it has first occurred, a finding that may be indicative of individual vulnerabilities and risk factors.

Epidemiological studies also have shown that PTSD symptoms are not the only, indeed not even the most likely, form of posttraumatic reactions. General reactions of fear, anxiety, sadness, dysphoria, anger, and guilt (among others) are common reactions to traumatic experiences. Other common reactions include the following: physical or somatic

complaints (insomnia, gastrointestinal symptoms, headaches, sleep problems); social and relationship difficulties; and increased substance use (Breslau et al., 1991; Kessler et al., 1995).

Posttraumatic Morbidity Versus Resilience

One of the most important lessons obtained from research is that most people who survive even the most harrowing of traumatic experiences do not develop PTSD or any other posttraumatic psychiatric disorder. This is not to suggest that people remain unaffected by traumatic experiences. To the contrary, most everyone is likely to experience at least short-term distress. Nevertheless, only a minority of individuals develop distress and functional impairment that rise to meet the criteria for one or more of the psychiatric disorders. Therefore, it is important scientifically and clinically to identify those characteristics of trauma-exposed people, and the stressors themselves, that contribute to adverse or positive outcomes.

Individual Differences, Risk and Protective Factors

Research has demonstrated that individual vulnerabilities and risk factors serve as strong predictors of PTSD development. For example, one robust finding in both traumatic stress and general stress research is that social support can play an important buffering role: Lower social support is associated with increased risk of PTSD, and higher amounts or quality of social support is protective against the development of PTSD (Andrews, Brewin, & Rose, 2003).

Gender is an important risk factor, with females at increased risk of developing lifetime PTSD relative to men by a ratio of approximately 2:1 (Breslau et al., 1991; Breslau et al., 1998; Kilpatrick et al., 2003). In one study, 13% of women and 6.2% of men met criteria for lifetime PTSD based on a randomly selected event; and 17.7% of women and 9.5% of men met criteria for lifetime PTSD based on the study participant's self-identified worst event (Breslau et al., 1998). A gender difference in conditional risk has been observed, even when controlling for type of traumatic event (Breslau et al., 1998; Kessler et al., 1995). Possible factors contributing to the higher prevalence of PTSD among females have been reviewed by Tolin and Foa (2006).

Other factors generally associated, to varying degrees, with greater risk for PTSD are lower socioeconomic status, lower intelligence, lower educational attainment, prior history of poor social adjustment or psychiatric disorder, increased severity of initial (peritraumatic) stress reactions, increasing severity of traumatic exposure, and presence of other environmental stressors. Recent data also suggest the role of certain genetic phenotypes, which may interact with environmental variables to affect rates of PTSD in the aftermath of trauma, although no candidate genes have been identified definitively (Koenen, 2007).

Comorbidity

Most individuals who have significant problems coping with trauma, and who meet the diagnostic criteria for PTSD, have additional problems. Consequently, comorbid depression, panic attacks, substance abuse, and other psychiatric issues can be common. In fact, there is good evidence to suggest that major depression is the most common form of posttraumatic psychopathology, even more prevalent than PTSD. In addition, PTSD patients frequently present with significant medical comorbidity (e.g., chronic pain) that requires increased health care (Elhai, North, & Frueh, 2005; Schnurr & Green, 2004).

THEORIES UNDERLYING THE PTSD DIAGNOSIS

As previously discussed, the vast majority (i.e., 75 to 80%) of individuals exposed to traumatic stressors do not develop PTSD. Several theoretical models have been formulated to explain for whom, when, and why PTSD develops. Biological theories have focused on stress hormone responses (e.g., Yehuda, 2002), neuroanatomy (e.g., Gilbertson et al., 2002), neurocircuitry (e.g., Shin & Handwerger, 2009), and genetic predispositions (e.g., Koenen, 2007). Psychosocial theories focus on exposure to life threat and interpersonal violence, histories of childhood abuse or other severe childhood adversities, and recent life stressors (Brewin, Andrews, & Valentine, 2000; Ozer, Best, Lipsey, & Weiss, 2008; Schnurr, Lunney, & Sengupta, 2004). Psychosocial theories also posit beneficial or buffering effects of protective factors including socioeconomic resources, education, intelligence, and social support. Cognitive theories of PTSD

identify patterns of altered beliefs and information processing consistent with persistent fear and hypervigilance (Dalgleish, 2004; Ehlers & Clark, 2000). Janoff-Bulman (1992) has suggested that "shattered assumptions" about self and the world are the basis for PTSD. Learning theories posit fear networks (Foa & Kozak, 1986) whereby trauma will condition associations and cognitions associated with hyperreactivity to reminders of the original event and behavioral avoidance. Emotional processing theories extend earlier fear network models, with additional emphasis on cognitive processing (Foa & Riggs, 1993; Foa & Rothbaum, 1998). Finally, there are theories that account for PTSD as a function of how traumatic memories are stored and processed (e.g., Brewin, Dalgeish, & Joseph, 1996).

Anyone reading this list of theories probably wants to know which one is correct. Unfortunately, there is no simple answer (Brewin & Holmes, 2003). The current situation may be likened to the proverbial blind men, each of whom describes a part of an elephant while missing the whole. So it may be that posttraumatic morbidity is a function of multiple factors including stress hormones, altered neural activity in the brain, cognitive appraisals, shattered beliefs, fear conditioning, intrusive memories, and biological and psychosocial risk and protective factors.

ISSUES AND CONTROVERSIES

Despite PTSD's success in spurring research, assisting clinicians, and providing a framework widely understood by the public, there are multiple issues and controversies that remain unresolved. Robert Spitzer, who served as chair of the DSM-III, observed with his colleagues (Spitzer, First, & Wakefield, 2007): "Since its introduction into the DSM-III in 1980, no other DSM diagnosis, with the exception of Dissociative Identify Disorder, has generated so much controversy in the field as to the boundaries of the disorder, diagnostic criteria, central assumptions, clinical utility, and prevalence in various populations" (p. 233).

In the sections that follow, we review the major issues and controversies that have arisen with regard to the PTSD diagnosis. We will then consider the implications of these concerns for the practicing clinician.

Challenges to the Assumption of a Specific Etiology

As previously discussed, the assumption of a specific etiology was fundamental to the origins of PTSD, and provided justification to separate a particular class of stressors from the general field of stress studies. If this assumption is correct, then individuals who do not experience a Criterion A traumatic event should not suffer from PTSD symptoms. Unfortunately for the "PTSD model," this has not turned out to be the case (Long & Elhai, 2009: Rosen & Lilienfeld, 2008). Instead, multiple studies have demonstrated that non-Criterion A events (e.g., not traumatic) can result in equivalent rates of PTSD (e.g., Long et al., 2008). Case reports have documented clinical presentations of PTSD among individuals who have suffered such non-life threatening events as financial strains, loss of friendships, marital infidelity, and collapse of adoption arrangements (e.g., Scott & Stradling, 1994). When loss of livestock on a ranch was reportedly associated with PTSD, commentators asked, "What is a traumatic event?" (Elhai, Kashdan, & Frueh, 2005).

It turns out that the assumption of a specific etiology for PTSD is so fraught with difficulties that a recent paper spoke of "the Criterion A problem" (Weathers & Keane, 2007). Most recently, analysts have considered the issue with proposals ranging from tighter definitions of traumatic events (Kilpatrick, Resnick, & Acierno, 2009) to the other extreme of totally doing away with the gatekeeper function of Criterion A (Brewin, Lanius, Novac, Schnyder, & Galea, 2009). Totally abandoning the PTSD "notion" of a qualitatively unique type of stressor could be premature in light of evidence from both animal and human studies that certain stressors which threaten survival of the organism elicit biological and behavioral reactions that are particularly extreme and persistent (Magnea & Lanius, 2008; Ronan & Summers, 2008). Whether "traumatic" stressors are in fact qualitatively different than other stressors, or better understood as extreme types on a continuum of stressors, remains unclear. Also, notice that if we eliminate Criterion A completely, then the T in PTSD must logically be dropped. In effect, we will have returned to the general field of stress studies and the nature of post-stress disorders.

The Symptom Criteria

PTSD is highly comorbid with other mental disorders—especially major depressive disorder, substance use and other anxiety disorders (Kessler et al., 1995). Moreover, several of PTSD's symptoms are shared by other mood and anxiety disorders' criteria. In fact, criteria for diagnosing PTSD can be completely fulfilled with the diagnoses of depression and specific phobia. Consider, for example, the PTSD reexperiencing symptoms B4 and B5. Criterion B4 reads "intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event." Criterion B5 reads "physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event." A comparison of these definitions with Criteria A and B for specific phobia shows them to be essentially the same. Thus, Criterion A for specific phobia states, "marked and persistent fear that is excessive or unreasonable, cued by the presence or anticipation of a specific object or situation." Criterion B for specific phobia states, "exposure to the phobic stimulus almost invariably provokes an immediate anxiety response, which may take the form of a situationally bound or situationally predisposed Panic Attack." Criterion C2 for PTSD reads, "efforts to avoid activities, places, or people that arouse recollections of the trauma," while Criterion D for specific phobia reads, "The phobic situation(s) is avoided or else is endured with intense anxiety or distress." Criterion C4 for PTSD reads, "markedly diminished interest or participation in significant activities," while Criterion 2A for major depression reads, "markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day." The interested clinician can find other examples of symptom overlap by comparing the criteria of these varied disorders.

In light of the similarity in defining criteria among different disorders, some have questioned if PTSD's high rates of comorbidity with depression, panic attack, and other anxiety problems results from the artifact of overlapping symptoms. Such considerations also have raised the question of whether PTSD (as currently defined in DSM-IV) is actually a unique disorder or merely an amalgam of other mood and anxiety disorders (Rosen, Spitzer, & McHugh, 2008; Spitzer & Wakefield, 2007). Adding

to these concerns, some researchers have demonstrated that a core set of PTSD's symptoms are best conceptualized as general dysphoria or distress, most common to other mood and anxiety disorders (Simms, Watson, & Doebbelling, 2002; Watson, 2005). Further, Bodkin, Pope, Detke, and Hudson (2007) demonstrated a high incidence of PTSD symptoms (78%) among depressed patients who had not reported a Criterion A event. Such findings suggest that PTSD symptoms may have been previously ignored (e.g., nightmares) among other patient groups, even though these stress reactions are part of the alternate clinical presentations. While 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), these findings do not resolve all concerns.

A case example that illustrates the issues was presented by Rosen, Spitzer, and McHugh (2008). A captain of a fishing boat that was lost at sea, in an incident that resulted in the death of several crew members, returned home with anxiety reactions that prevented him from continuing in his usual career. As a result of these new and unfamiliar anxiety problems, loss of career, resulting financial pressures, and associated adjustment issues, the captain developed severe situationally-based depression. Prior to the DSM-III, and introduction of a PTSD diagnosis, the captain's clinical problems would have been conceptualized as a conditioned phobic reaction resulting from the maritime accident, grief reactions related to loss of crew and friends, and a reactive depression resulting from situational stress. Clinicians and researchers alike can ask how our understanding of the captain's psychiatric problems has been furthered with the diagnosis of PTSD. To the extent PTSD merely duplicates precursor diagnoses and does not lead to increasingly efficient and effective treatment strategies, its construct validity and clinical utility are called into question (McHugh & Treisman, 2007).

Criterion Creep

Application of PTSD by clinicians, and public interest in the diagnosis, has not been tempered by challenges to the construct's validity. Instead, what can be thought of as the "PTSD model," has been extended to an ever-increasing array of events and to an expanding set of stress

reactions. Consider how the concept of a specific set of stressors associated with PTSD (Criterion A), once reserved for those who experienced or witnessed a traumatic event, can now be applied to individuals who simply hear of misfortunes befalling others. This change occurred when the definition of Criterion A was changed in the DSM-IV (APA, 1994). Now, with the new definition of what constitutes trauma, peer-reviewed journal articles have reported on cases of PTSD resulting from viewing television (Ahern, Galea, Resnick, & Vlahov, 2004; Bernstein et al., 2007; Eth, 2002; Simons & Silveira, 1994). It also has been argued that non-traumatic events can lead to PTSD by creating a worry or anticipation of future trauma. For example, if an individual makes an inappropriate sexual comment in the workplace, might that create PTSD in a co-worker who becomes concerned over what other transgressions might occur. This creates the conceptual equivalent of "pre-traumatic" stress disorder, leading one commentator to observe: "Any unit of classification that simultaneously encompasses the experience of surviving Auschwitz and that of being told rude jokes at work must, by any reasonable lay standard, be a nonsense, a patent absurdity" (Shephard, 2004, p. 57).

Expansion of the PTSD model, a phenomenon referred to as "criterion creep" (Rosen, 2004a), also has occurred with the symptom criteria. Normal and even expected reactions to a traumatic event, such as anger or uncertainties about the future, are now referred to as "symptoms." This labeling is encouraged by terms such as "subsyndromal," "subthreshold," or "partial" PTSD. In these cases, individuals who exhibit *some* PTSD symptoms after an adverse event, but who do not meet full PTSD criteria, can still be said to be having symptoms of that disorder. While there is empirical evidence that "subthreshold PTSD" is associated with some psychosocial impairment (Breslau, Lucia, & Davis, 2004; Stein, Walker, Hazen, & Forde, 1997), "full PTSD" is associated with "higher magnitude" (more severe) traumatic stressors and greater impairment. Such distinctions support the construct validity of full PTSD as distinct from partial or subthreshold variants. It follows, therefore, that the notion of partial PTSD may be no more meaningful diagnostically than saying that someone with a bad cold has the symptoms of tuberculosis or lung cancer. This logical fallacy becomes more extreme if the individual is coughing only because they are in a smoky tavern (Rosen & Lilienfeld, 2008).

Once again, clinicians are reminded of the essential need to distinguish between normal reactions to a particular situation, and the symptoms of disorder (Horwitz & Wakefield, 2007).

Delayed Onset PTSD

Although included in DSM-IV as a possible subcategory of the disorder, there is little in the way of empirical data to support the existence of delayed-onset PTSD. Large-scale epidemiological studies have reported zero or extremely low rates of delayed-onset PTSD in civilians and veterans (Breslau, Davis, Andreski, & Peterson, 1991; Frueh, Grubaugh, Yeager, & Magruder, 2009; Helzer, Robins, & McEvoy, 1987). On the other hand, if "delayed-onset" is redefined and conceptualized as a delay in seeking treatment, or subsequent exacerbation of prior symptoms years after an event, then the phenomenon may be relatively common (Andrews, Brewin, Philpott, & Stewart, 2007). Such findings encourage clinicians 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.

PTSD in the Courtroom

The assumption of a specific etiology (Criterion A) that is distinctive of the PTSD diagnosis also creates special concerns when patients are involved in claims of compensation or personal injury lawsuits. Slovenko (1994) observed:

> In tort litigation, PTSD is a favored diagnosis in cases of emotional distress because it is incident specific. It tends to rule out other factors important to the determination of causation. Thus plaintiffs can argue that all of their psychological problems issue from the alleged traumatic event and not from myriad other sources encountered in life. A diagnosis of depression, in contrast, opens the issue of causation to many factors other than the stated cause of action (p. 441).

An awareness of PTSD's attractiveness in court cases likely contributed to the DSM-IV including a specific cautionary guideline for clinicians: "Malingering should be ruled out in those situations in which

financial remuneration, benefit eligibility, and forensic determinations play a role" (p. 467; APA, 1994). Unfortunately, the task of ruling out malingering is easier said than done. In one study (Hickling, Blanchard, Mundy, & Galovski, 2002), six actors presented false claims of PTSD at a clinic that specialized in the assessment of motor vehicle accident victims. After clinical assessment interviews, psychological tests, and a psychophysiologic assessment of responses to trauma relevant stimuli, all six "patients" received a diagnosis of PTSD. This study demonstrates the ability of individuals to feign both an event (the non-existent accident) and subjective symptoms (intrusive thoughts, nightmares). Other studies on malingering confirm that clinicians are not lie detectors (Slovenko, 2002)—a reality that does not change even with years of clinical experience.

Clinicians who find themselves performing a forensic role and serving as an expert for the courts, should acquaint themselves with the large literature on malingering and the proper conduct of forensic assessments (e.g., Simon, 2003). When clinicians are treating patients involved in litigation, they should remain aware of their advocacy role and reliance on self-report. A clinician who testifies at trial can tell a jury that their patient reported problems with nightmares and appropriate treatment was provided. At the same time, the clinician should not tell a jury that they independently determined the existence of nightmares or the truthfulness of their patient's reporting. Strategies for dealing with these complex issues have been discussed in several publications (e.g., Taylor, Frueh, & Asmundson, 2007; Pankratz, 1998; Rosen, 2004b).

Traumatic Memory

There has long been a wide-held belief that memory for traumatic events works differently from memory for other, more common, aspects of the human experience. Dating back to the work of Sigmund Freud right on through to contemporary writers, and popularized in Hollywood movies, there has been the claim that highly traumatic experiences, especially those occurring in childhood, are susceptible to being repressed into the unconscious mind. That is, certain events are so horrific that the human mind cannot tolerate them, and therefore banishes them to some hidden place deep in the unconscious, from which they may emerge to

cause psychopathology and interpersonal maladjustment. This phenomenon has been used to explain putative "multiple personalities," borderline personality disorder, and delayed PTSD.

As it turns out, a large body of research (McNally, 2003) has failed to support this notion of a special mechanism for traumatic memory, and there is no valid scientific evidence to support the claims of "body memory" or "recovered memory" made by a small number of adherents. A number of provocative lawsuits based on claims of "recovered memory" have made the news, and some still linger in U.S. courts. In some cases, huge awards have been obtained by patients who recanted a therapeutically "recovered" memory and then sued their therapist for improperly implanting the falsely held belief (e.g., Gustafson, 1995). Although debates continue, with an occasional call for middle ground (Ost, 2003), it is important that clinicians appreciate the dangers, clinically and legally, of attempting to help or encourage patients to recall "repressed" trauma memories. Clinicians are strongly advised to avoid the use of such "recovered memory" techniques (e.g., Brandon et al., 1997).

Failed Treatments and Exaggerated Claims

Clinicians hold to the singular purpose of helping others. A corollary to this therapeutic goal is "Primum Non Nocere," or "first do no harm." Unfortunately, in the short history of traumatology's three decades, a number of well-intentioned treatments have produced questionable, if not outright harmful results. Research indicates that early interventions in the form of critical incident stress debriefings (CISD) can impede the natural recovery and resilience that is characteristic of most individuals after trauma (e.g., Rose, Bisson, & Wessely, 2001). More damaging are previously referenced techniques that can lead to false trauma memories, unfounded accusations, and the tearing apart of families (Hagen, 1997). These controversial areas of clinical concern are discussed elsewhere in this text.

In other instances, inadequately tested therapies have been promoted with exaggerated and even extreme claims of success. In the 1990s, a group of such treatments came to be known as the "Power Therapies." When these therapies were introduced, it was claimed (without empirical support) that they were more effective, efficient, or in some other way more powerful than other empirically established procedures. Perhaps

not surprisingly, when research was conducted on the power therapies, it was found that nothing miraculous or special was occurring. Instead, known principles of behavior change appeared to operate, at a level of efficacy and efficiency already known to accepted clinical practice (Rosen, Lohr, McNally, & Herbert, 1998). Ill-conceived treatments and exaggerated claims remind clinicians that they can best serve patients, and avoid doing harm, by relying on empirically supported methods and practices (Cukor, Spitalnik, Difede, Rizzo, & Rothbaum, 2009).

PTSD, STRESS STUDIES, AND THE NEED FOR EVIDENCE-BASED PRACTICE

There is little doubt that mental health clinicians are in a stronger position to conceptualize, assess, and treat posttraumatic disorders as compared to three decades ago. When PTSD was introduced in 1980, little was known about the prevalence and course of posttraumatic symptoms, nor was there an appreciation for the important issue of comorbidity. We now know that the majority of individuals face trauma at one or more points in their lifetime. While all individuals may be expected to have reactions in the immediate aftermath of adversity, this does not necessarily constitute psychiatric disorder. In fact, most individuals demonstrate resilience and cope successfully over time. Nevertheless, a minority of those who experience a traumatic event have reactions of sufficient severity and duration to warrant psychiatric diagnosis. In such cases, the appropriate diagnosis may be PTSD, although this diagnosis should not be reflexively provided to all patients presenting in the aftermath of trauma. Instead, clinicians should take a broad view (e.g., considering all viable and evidence-supported hypotheses) when formulating a diagnosis and planning appropriate treatment interventions.

Research findings also have challenged most every assumption upon which PTSD is based. Studies have shown that symptom criteria for diagnosing PTSD can occur in the absence of traumatic events, even presenting at high rates in non-traumatized patients. Such findings raise the question of whether PTSD is a distinct disorder, or if instead it was constituted by artificially joining already extant problems (e.g., phobia and depression). Additional challenges to the construct validity of PTSD

have been raised and remain in active debate. To some extent, these issues have been reflected in changing criteria through various editions of the DSM. How the issues will be resolved, and how PTSD should be defined in future editions of the DSM, remains uncertain. Clearly, an informed clinician who works with traumatized patients has a full plate of issues on which to stay current.

It is important to note that when issues are raised concerning how best to understand and apply the PTSD diagnosis, we are not questioning the emotional pain of those who have survived traumatic events or suffered horrific loss. That pain and suffering exists no matter what label a clinician chooses to use. When we strive to apply assessment and treatment practices that are evidenced based, and when we debate controversial issues, the reality of our patients and their problems is always foremost in our minds. It is in this spirit that every clinician can and should question if the introduction of PTSD, on balance, has advanced our assessment, diagnosis, and treatment of posttraumatic psychiatric disorders. Only through this active process of questioning and exploring will we come to know if PTSD is a valid and clinically informative diagnosis that was appropriately carved out of general stress studies.

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