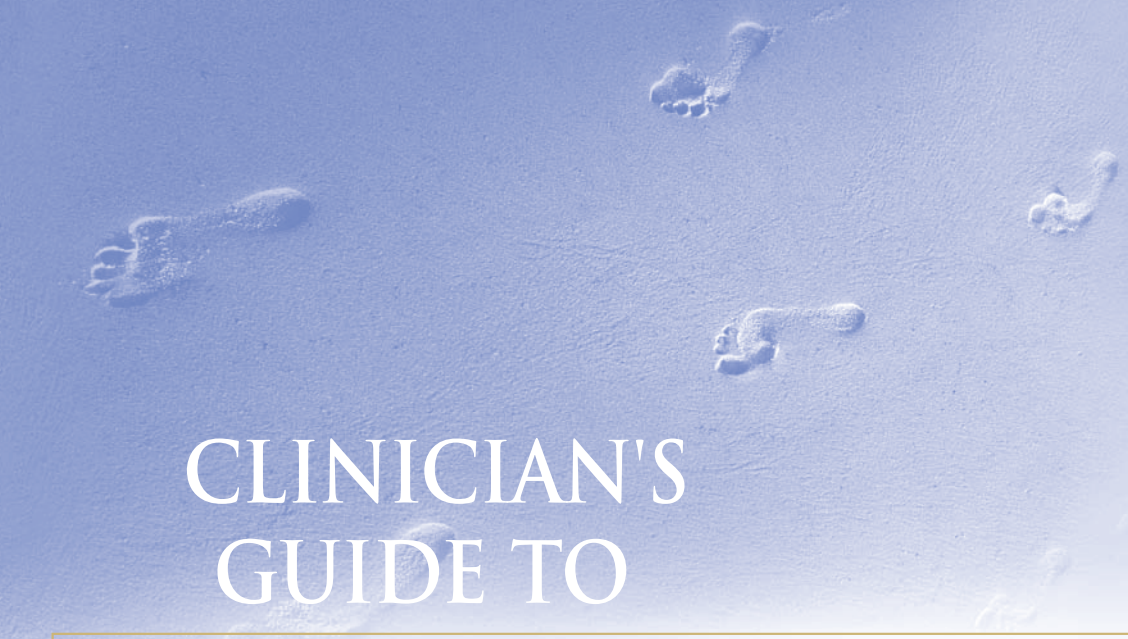

CLINICIAN'S GUIDE TO

Posttraumatic Stress Disorder

Edited by

Gerald M. Rosen
B. Christopher Frueh

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Contents

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Preface

Since its introduction in 1980, posttraumatic stress disorder (PTSD) has changed the landscape of stress studies and created an array of sociopolitical, conceptual, and clinical issues. For the better, research has grown exponentially over the years, providing clinicians, healthcare administrators, and policy makers with a better understanding of posttraumatic psychiatric morbidity. Despite this progress, controversies abound as to how clinicians should diagnose and treat psychiatric disorder in the aftermath of trauma. Further, a number of misconceptions and myths concerning PTSD have adversely influenced clinical practice and traumatic stress studies. This is of great concern, for it creates the risk of doing harm in our clinical work.

To address the core issues facing clinicians, we have brought together an international group of leading clinicians and clinical researchers. Their scholarly reviews of the literature are joined with recommendations for clinical practice, thereby providing the clinician with insights and skills based on the best available evidence. In the first section (Chapters 1 through 5), the reader is provided with an overview of stress studies and core issues that concern the PTSD construct. The second section (Chapters 6 through 10) covers issues in the assessment and treatment of posttraumatic disorders. The Clinician's Guide concludes with an Afterword that considers future definitions of PTSD, and how changes may impact how we, as clinicians, conceptualize our patient's problems.

As the reader progresses through the chapters and learns more about recent research findings, several closely held beliefs are likely to be

challenged. On those occasions, when a particular misconception or myth is examined, we ask that the reader remain open to new ideas. It is in this spirit that contributing authors have lent their time and expertise.

Gerald M. Rosen
B. Christopher Frueh

Clinician's Guide to Posttraumatic Stress Disorder

PART

I

Core Issues

CHAPTER

1

Posttraumatic Stress Disorder and General Stress Studies

Gerald M. Rosen
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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.

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.

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