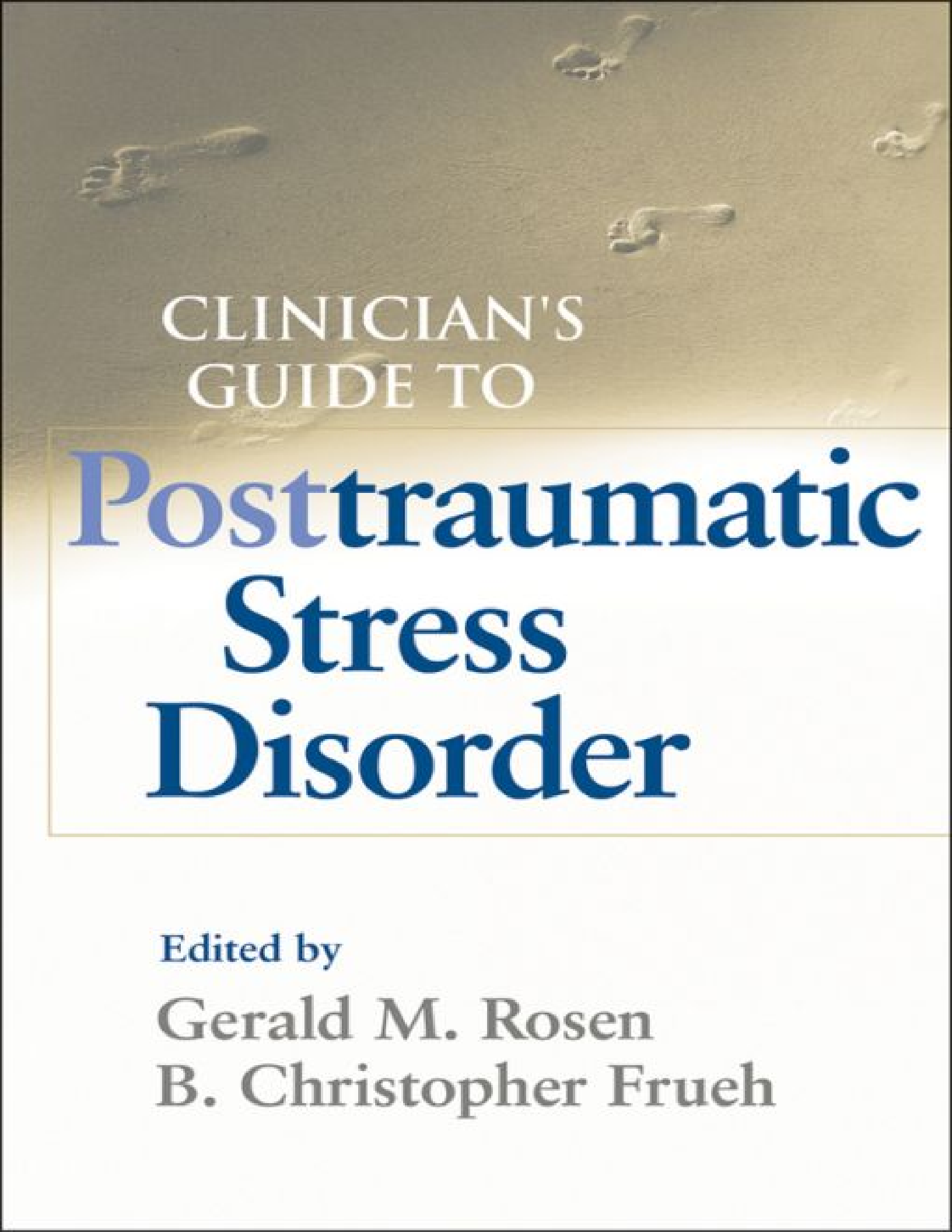
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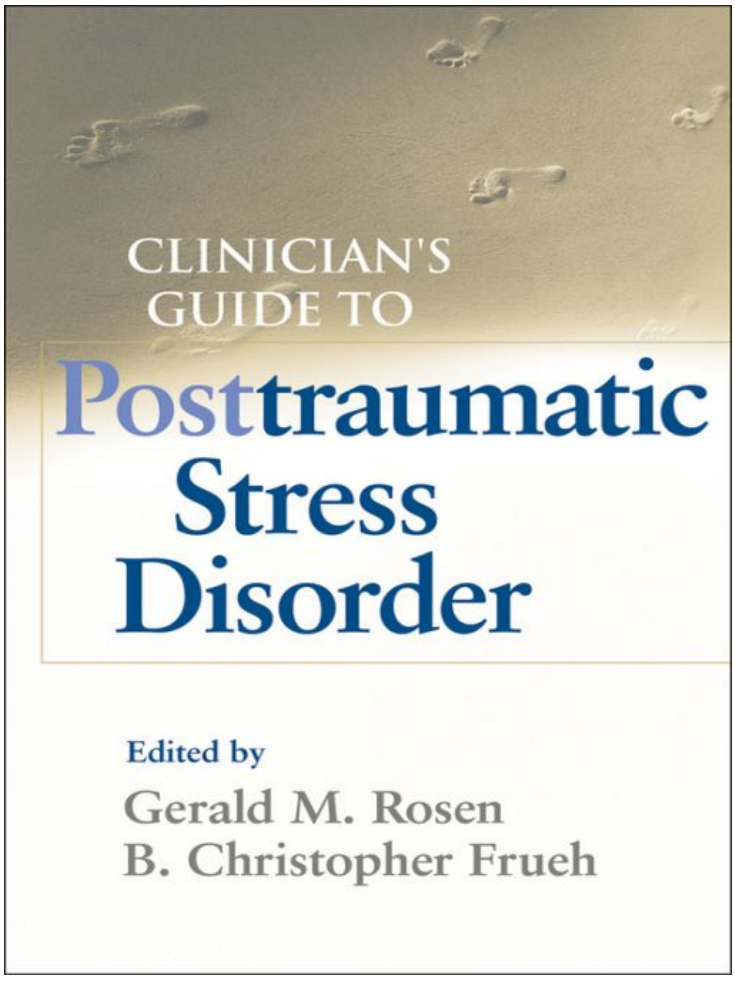
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Preface

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 post-traumatic 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

PART I

Core Issues

CHAPTER 1

Posttraumatic Stress Disorder and General Stress Studies

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B. CHRISTOPHER FRUEH
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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 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).

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

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

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.

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.

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