Chapter 21
Posttraumatic Stress Disorder

The posttraumatic stress syndrome has been recognized for decades (Freud, 1921), and systematic empirical inquiry dates back to the 1940s (Kardiner, 1941). Yet, it was not until 1980 that the now highly recognizable posttraumatic stress syndrome was officially catalogued within the official nosological compendium of the American Psychiatric Association, the *Diagnostic and Statistical Manual of Mental Disorders,* third edition (DSM-III; American Psychiatric Association, 1980). With this recognition of the syndrome as an official mental disorder came a surge of research efforts designed to lead to better diagnostic refinement as well as improved treatment.

Whereas once the syndrome was viewed almost exclusively as a result of armed combat, now posttraumatic stress disorder (PTSD) has been found to result from not only war-related situations but also a host of non-combat-related experiences as well. In 1987, and again in 1994 and 2000, the American Psychiatric Association revised its official nosology (1987, 1994, 2000). Are we coming closer to a comprehensive understanding of PTSD, or are we just beginning to scratch the surface of what may be a uniquely complex interaction of pathophysiological and psychopathological constituents? The purpose of this chapter is to review current evidence on the nature of PTSD as well to as offer an integrating phenomenological hypothesis regarding this disorder, which appears to be playing more and more a role in Western society.

The Prevalence of Trauma as a Public Health Problem

What is the magnitude of risk for experiencing a significant psychological trauma that might yield a significantly adverse impact upon one's mental health? Is the risk minimal, or does it represent a significant public health issue? To review Chap. 1, the reader will recall:

- Recent evidence suggests that 82.8% of adults in the USA will be exposed to a traumatic event during their lifetime (Breslau, 2009).
- Suicide rates in the military seem to be increasing (Kang & Bullman, 2009).

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- Twelve-month DSM-IV disorders are highly prevalent in the USA, with 14% experiencing moderate to severe cases (Kessler, Chiu, Demler, Merikangas, & Walters, 2005).
- Suicide was the tenth leading cause of death in the USA in 2007, and an estimated 11 attempted suicides occur per every suicide death.
- An elevated rate of major depression was equal to the rate of PTSD in New York City residents several months after the attacks on the World Trade Center of September 11, 2001 (Galea et al., 2002).
- Rates of trauma occurrence related to violence, injury/shock trauma, trauma to others, and unexpected death peaked sharply at age 16–20 years (Breslau, 2009).
- The lifetime prevalence of criminal victimization was assessed among female health management organization patients and found to be about 57%.
- In 2001, the terrorist attacks against the World Trade Center and the Pentagon focus terrorism against the USA.
- Of 2050 American Airlines (AA) flight attendants, 18.2% reported symptoms consistent with probable PTSD in the aftermath of the September 11 attacks (Lating, Sherman, Everly, Lowry, & Peragine, 2004).
- Clearly, trauma and stress are at epidemic proportions in the USA. It seems clear that such conditions represent a "clear and present danger" to the psychological health of American society.
- Perhaps of greatest concern, from a public health perspective is the realization that veterans returning from military service in Iraq and Afghanistan are returning home with a high prevalence of PTSD and PTSD-like syndromes. A recent review of 29 published studies revealed varying estimates of PTSD. "Among previously deployed personnel not seeking treatment, most prevalence estimates range from 5 to 20%. Prevalence estimates are generally higher among those seeking treatment: As many as 50% of veterans seeking treatment screen positive for PTSD…Combat exposure is the only correlate consistently associated with PTSD" (Ramchand et al., 2010, p. 59).
- The Veterans Affairs (VA) estimate that about 26% of veterans seeking treatment at VA facilities meet criteria for PTSD (U.S. Department of Veteran Affairs, Veterans Health Administration, Office of Public Health and Environmental Hazards, 2010).

Clearly, trauma has reached epidemic proportions in the USA! It seems clear that such crisis events represent a "clear and present danger" to the psychological health of Americans.

Diagnostic Symptomatology

In 1941, Kardiner (1941) described five consistent clinical features of the syndrome now referred to as PTSD:

1. Constriction of personality functioning
2. Exaggerated startle reflex and irritability

3. Psychic fixation upon the trauma
4. Atypical dream experiences
5. A propensity for explosive and aggressive reactions

In 1942, Gillespie described an acute "war neurosis" as having as an important clinical feature an increased startle reaction characterized by increased and generalized muscular tension, palpitations, and a "sinking feeling," thus emphasizing a distinct autonomic nervous system (ANS) component to this posttrauma syndrome.

In 1980, the American Psychiatric Association described PTSD as a form of anxiety disorder:

> The essential feature is the development of characteristic symptoms following a psychologically traumatic event that is generally outside the range of usual human experience. … The characteristic symptoms involve re-experiencing the traumatic event; numbing of responsiveness to, or reduced involvement with, the external world; and a variety of autonomic, dysphoric, or cognitive symptoms. (p. 236)

The specific criteria are listed in Table 21.1. PTSD was described in subvariations as well:

1. "Acute," in which the onset of symptoms occurred within 6 months of the trauma and lasted less than 6 months.
2. "Chronic or delayed," in which either or both of the following applied: duration of the symptoms for 6 months or more (chronic) and/or the onset of symptoms at least 6 months after the trauma (delayed).

In 1987, the American Psychiatric Association revised its criteria for PTSD (American Psychiatric Association, 1987). In doing so, the traumata giving rise to PTSD were somewhat better defined. Once again, the notion of a psychologically distressing event outside the normal range of human experience was emphasized. Yet, specific instances were cited:

> a serious threat to one's life or physical integrity; a serious threat or harm to one's children, spouse, or other close relatives and friends; sudden destruction of one's home or community; or seeing another person who has recently been, or is being, seriously injured or killed as a result of an accident or physical violence. In some cases the trauma may be learning about a serious threat or harm to a close friend or relative. (pp. 247–248)

Table 21.2. describes the specific criteria requisite for the PTSD diagnosis.

In 1994, the American Psychiatric Association once again changed the diagnostic criteria for PTSD as contained within the revised nosological compendium (DSM-IV; American Psychiatric Association, 1994). The DSM-IV criteria (see Table 21.3) represented major alterations in the official criteria for PTSD, and even recognized a more acute variant of the posttraumatic syndrome, acute stress disorder (ASD; see Table 21.4).

The major changes in the DSM-IV formulation of PTSD reside in the definition of the traumatic event. While DSM-III and DSM-III-R defined the traumatic stressor as an unusually distressing event, the DSM-IV actually restricted the nature of the stressor by limiting it to events that involve actual or threatened death or serious injury to oneself or others. The DSM-IV-R stressor of the sudden destruction to one's home or community, in the absence of injury or death, was now omitted. This

Table 21.1 Diagnostic criteria for posttraumatic stress disorder, DSM-III

A. Existence of a recognizable stressor that would evoke significant symptoms of distress in almost everyone
B. Reexperiencing the trauma as evidenced by at least one of the following:
   1. Recurrent and instrusive recollections of the event
   2. Recurrent dreams of the event
   3. Suddenly acting or feeling as if the traumatic event were reoccurring, because of an association with an environmental or ideational stimulus
C. Numbing of responsiveness to or reduced involvement with the external world, beginning some time after the trauma, as shown by at least one of the following:
   1. Markedly diminished interest in one or more significant activities
   2. Feeling of detachment or estrangement from others
   3. Constricted affect
D. At least two of the following symptoms that were not present before the trauma:
   1. Hyperalertness or exaggerated startle response
   2. Sleep disturbance
   3. Guilt about surviving when others have not, or about behavior required for survival
   4. Memory impairment or trouble concentrating
   5. Avoidance of activities that arouse recollection of the traumatic event
   6. Intensification of symptoms by exposure to events that symbolize or resemble the traumatic event

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restriction in the nature of the traumatic stressor was not well received by many individuals who work in mass disaster venues.

While restricting one aspect of the traumatic criterion (Criterion A), the DSM-IV actually broadened another aspect of the traumatic stressor by including a subjective distress criterion.

As this volume is being written, the American Psychiatric Association is in the process of revising the criteria for PTSD once again through the publication of the DSM-5. The changes in the diagnostic formulation are proposed at this point, but are nevertheless worth mentioning. Simply stated, the A-2 criterion (fear, helplessness, and horror) would be dropped and a fourth cluster of signs and symptoms would be added ("depression"). More specifically a "depression" cluster consisting of "negative alterations in cognition and mood that are associated with the traumatic event (s)" would be added to the existing three clusters of re-experiencing, avoidance, and numbing, as well as, increased stress arousal. The "depression" cluster consists of psychogenic amnesia, negative expectations about self and the world, self-blame, negative affect, diminished interest in important activities, interpersonal estrangement, and anhedonia.

Are these diagnostic criteria of equal phenomenological importance, or are certain elements more important than others? Let us take a closer look at the posttraumatic stress concept with an appreciation for reformulation. Figure 21.1 presents a phenomenological algorithm that provides a hierarchical structure to the constituent

Table 21.2 Diagnostic criteria for posttraumatic stress disorder, DSM-III-R

A. The person has experienced an event that is outside the range of usual human experience and that would be markedly distressing to almost anyone (e.g., serious threat to one's life or physical integrity; serious threat or harm to one's children, spouse, or other close relatives and friends; sudden destruction of one's home or community; or seeing another person who has recently been, or is being, seriously injured or killed as the result of an accident or physical violence)

B. The traumatic event is persistently reexperienced in at least one of the following ways:
   1. Recurrent and intrusive distressing recollections of the event (in young children, repetitive play in which themes or aspects of the trauma are expressed)
   2. Recurrent distressing dreams of the event
   3. Suddenly acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative [flashback] episodes, even those that occur upon awakening or when intoxicated)
   4. Intense psychological distress at exposure to events that symbolize or resemble an aspect of the traumatic event, including anniversaries of the trauma

C. Persistent avoidance of stimuli associated with the trauma or numbing of general responsiveness (not present before the trauma), as indicated by at least three of the following:
   1. Efforts to avoid thoughts or feelings associated with the trauma
   2. Efforts to avoid activities or situations that arouse recollections of the traumas
   3. Inability to recall an important aspect of the trauma (psychogenic amnesia)
   4. Markedly diminished interest in significant activities (in young children, loss of recently acquired development skills such as toilet training or language skills)
   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, or children, or a long life)

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by at least two of the following:
   1. Difficulty in falling or staying asleep
   2. Irritability or outbursts of anger
   3. Difficulty in concentrating
   4. Hypervigilance
   5. Exaggerated startle response
   6. Physiological reactivity upon exposure to events that symbolize or resemble an aspect of the traumatic event (e.g., a woman who was raped in an elevator breaks out in a sweat when entering any elevator)

E. Duration of the disturbance (symptoms in B, C, and D) of at least 1 month

elements. We have combined the depression, avoidance, and withdrawal clusters as we believe they are consistent with a singular phenomenological syndrome.

As the algorithm indicates, posttraumatic stress represents a dynamic "process" rather than a monothetic formulation. Figure 21.1 emphasizes the etiological role that subjective interpretation of the traumatic stressor can play in the determination of the amplitude and chronicity of the posttraumatic stress response. This view is in concert with the model utilized throughout this text as the overarching framework for

Table 21.3 Diagnostic criteria for posttraumatic stress disorder, DSM-IV

A. The person has been exposed to a traumatic event in which both of the following were present:
   1. 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 persistency 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 3 months

Chronic: if duration of symptoms is 3 months or more

Specify if:

With Delayed Onset: if onset of symptoms is at least 6 months after the stressor

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Table 21.4 Diagnostic criteria for acute stress disorder, DSM-IV

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
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., thoughts, 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 2 days and a maximum of 4 weeks and occurs within 4 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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understanding the human stress response. At the same time, Fig. 21.1 argues that much of the depressive avoidance, numbing, and withdrawal that is replete in the posttraumatic stress constellation may, indeed, be but a second-order symptom manifestation.

While acknowledging the important role that subjective interpretation plays in the traumatic response, Fig. 21.2 is presented as a means of understanding the variable impact of subjective interpretation of the trauma spectrum.

Let us take a closer look at posttraumatic stress through the utilization of a factorial taxonomy (i.e., a two-factor model of posttraumatic stress), including the notion of subjective appraisal. The A (2) criterion of the DSM-IV notes that the individual's response to the traumatic event must involve "intense fear, helplessness, or horror." This alteration has engendered some concern from victims' advocacy groups in that acknowledgment of the subjective aspects of the traumatic stressor may lead to a "blame the victim" attitude. Yehuda (1998) raised this issue and stated, "The stipulation in DSM-IV that individuals must experience a subjective response to an event now makes the study of risk factors necessary rather than inappropriate" (p. 3).

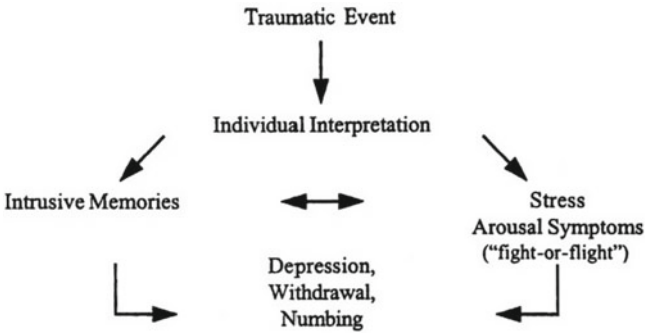

Fig. 21.1 As described by Everly and Lating (1995), the manifestation of the three symptom clusters consisting of intrusive memories, stress arousal symptoms, and withdrawal, depression, and numbing are predicated upon a complex interaction between the traumatic event and the individual experiencing the event

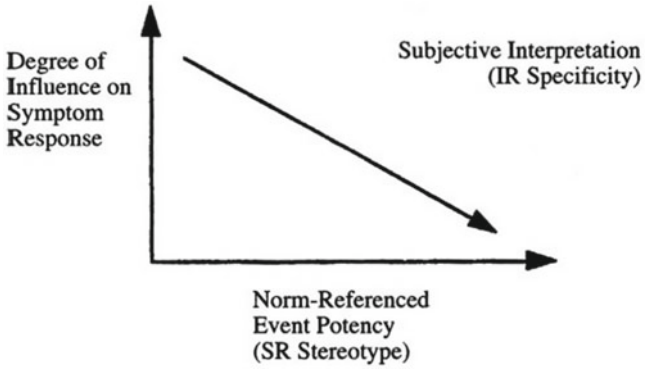

Fig. 21.2 As noted in Fig. 21.1, the nature and degree of manifest posttraumatic symptomatology is a function of the nature of the traumatic event and the individual experiencing the event. So as not to misinterpret this concept as reason to "blame the victim," the role of the victim's subjective interpretation is portrayed in overall event potency (severity). Traumatic events will vary in their normative severity, or potency. This is called stimulus–response (SR) stereotype and simply means that "mild" stressors usually engender "mild" responses, while "severe" stressors usually engender "severe" responses. Automobile accidents are less severe than torture. Thus, as the norm-referenced severity of the stressor event increases, the less a role-subjective interpretation, called individual response (IR) specificity, plays in determining the severity of the manifest symptom response. Thus, subjective interpretation plays less of a role in shaping the traumatic response to torture than it might to an automobile accident

The DSM-IV-TR also acknowledges the potential for PTSD to be associated with "… a change from the individual's previous personality characteristics" (American Psychiatric Association, 2000, p. 465). In recognizing that PTSD could alter something as concretized as personality, a new realm of psychological and biological phenomenological possibilities emerges.

A Two-Factor Theory of Posttraumatic Stress

Everly (1993; Everly & Lating, 1995, 2004) has analyzed the PTSD construct and found it to reveal two key factors, or constituents:

1. Neurological hypersensitivity
2. Psychological hypersensitivity (Everly, 1993; Everly & Lating, 1995, 2004)

Neurological Hypersensitivity

It is clear that the posttraumatic stress syndrome possesses a significant neurological constituency. Kolb (1987) has suggested that the PTSD symptoms fall within four categories (1) impaired perceptual, cognitive, and affective functions; (2) symptoms of released activation; (3) reactive affect and avoidance; and (4) restitutive symptoms and behaviors.

Yet, Kolb argues that the symptoms of released activation are the "constant" symptoms of the condition: the exaggerated startle reaction, irritability, hyperalertness, nightmares, and related psychophysiological expressions of ANS hyperfunction.

Similarly, (Foy, Sipprelle, Rueger, & Carroll 1984), in a comparison of methods for the concurrent discrimination of PTSD, found that self-report indices of anxiety and ANS arousal alone were capable of correctly identifying more than 90% of the study's participants. The investigation employed 21 Vietnam veteran PTSD patients and 22 Vietnam veterans with other psychiatric complaints.

In a review of three psychophysiological investigations into PTSD, Kolb (1984, 1987) concluded that indices of sympathetic nervous system (SNS) arousal were capable of differentiating PTSD from non-PTSD participants.

PTSD participants showed more autonomic arousal in response to trauma-related stimuli than did non-PTSD participants. Thus, Kolb (1987) concluded that "psychophysiological assessment offers strong potential not only for diagnostic identification … but also for assessment of severity of the disorder" (p. 991).

Finally, Horowitz, Wilner, Kaltreider, and Alvarez (1980) investigated the signs and symptoms of PTSD. Using a multi-inventory battery of self-report indices, they investigated the three major PTSD clusters: (1) intrusive re-experiencing of the trauma, (2) numbing/avoidance reactions, and (3) anxiety/stress reactions. The authors concluded that intrusive thinking and general symptoms of distress were of primary clinical prevalence and importance in the PTSD phenomenon. They added that the numbing and avoidance signs and symptoms are best understood as efforts of the PTSD patient to control the primary PTSD symptomatology.

From an anatomical perspective, in concert with the formulation of MacLean (1949), Gray (1982) has identified the septal–hippocampal complex as the neuroanatomical epicenter for the integration of exteroceptive as well as interoceptive, proprioceptive, and cognitive stimuli (Seifert, 1983; Van Hoesen, 1982). More

specifically, Gray argues, as do Reiman et al. (1986), that the noradrenergic system within the septal–hippocampal nuclei bears primary responsibility for integrating and responding, via hypothalamic efferent mechanisms, to novel and unpleasant stimuli, and furthermore, that stimulation of these projections results in a heightened sensitivity and reactivity within all innervated regions, including neuroendocrine effector mechanisms, to environmental cues seen in any way as novel, threatening, or otherwise aversive. Similarly, Madison and Nicoll (1982) found that noradrenergic neurons from the locus ceruleus to the hippocampus serve to impair the ability of the septal–hippocampal region to accommodate to excitatory stimuli.

Reiman et al. (1986) have demonstrated through positron emission tomography that the septal–hippocampal complex plays a major role in panic attacks. They further conclude that via the septal–amygdalar complex, the septal–hippocampal nuclei can initiate a hypothalamically mediated stress response (see Aggleton, 1992; Cullinan, Herman, Helmreich, & Watson, 1995; LeDoux, 1995).

Gloor (1986) has reported that the hippocampus plays a major role in memory and fear reactions. Electrophysiological investigations of awake patients having surgery for epilepsy found that activation of the hippocampus was capable of engendering "flashbacks," affective lability, perceptual distortions, fear, worry, and even guilt reactions (see also Post, 1986; Seifert, 1983).

In summary, to this point, a wide range of evidence indicates that residing with the confines of the septal–hippocampal–amygdalar complex are nuclei responsible for engendering all of the major symptoms of PTSD, including intrusive recollections and flashbacks (Gloor, 1986), neurological hypersensitivity, hyperstartle reactions, and inhibited stimulus accommodation (Gray, 1982; Madison & Nicoll, 1982), panic-like responses (Reiman et al., 1986), fear, rumination, worry, guilt-like reactions (Gloor, 1986), and affective lability (Post, 1986). Cooper, Bloom, and Roth (1982) have suggested that the role of the locus ceruleus is to act as a general orienting system rather than as a specific organizing epicenter for panic and related dysfunction (see also Charney, Deutch, Southwick, Krystal, & Friedman, 1995).

The amygdala has been ascribed a preeminent role in the anatomical foundations of PTSD (Charney, Deutch, Krystal, Southwick, & Davis, 1993). Consistent with the survival orientation of the "fight-or-flight" response, the amygdala appears to possess a specialized mechanism for processing emotional, especially fear-related, memories (LeDoux, 1992). LeDoux has argued that the amygdala may process emotional memories in such a way that "memories established through the amygdala are indelible" (p. 342). This may help us understand the persistence of traumatic memories; that is, the maintenance of fear-related memories may serve as a means of assuring continued survival, especially if coupled with autonomic mobilization, hypervigilance, and explosive reactivity or withdrawal and avoidance behaviors (the fight-or-flight response). A more recent review has extended our initial understanding of the neuroanatomy of PTSD, however. Sripada, Gonzalez, Phan and Liberzon's (2011) analysis and Liberzon and Sripada's (2007) review correctly implicated the importance of contextualization and interpretation in generation of PTSD. Their work underscores the medial prefrontal cortex (mPFC) as an important structure in the psychological process of contextualization and thus as a critical anatomical foundation of PTSD.

If, indeed, the anatomical basis for PTSD is in the mPFC–septal–hippocampal–amygdalar system, what extraordinary physiology serves to sustain the phenomenon? The hypersensitivity formulations of van der Kolk, Greenberg, Boyd and Krystal (1985) and Kolb (1987) as generically extended within this text and elsewhere (Everly, 1985b, 1993; Everly & Benson, 1989) seem reasonable. Using the disorders of arousal model described earlier, it may be argued that PTSD represents a limbic-system-based condition of neurological hypersensitivity, where a pathognomic propensity for limbic hyperreactivity is related to intraneuronal alterations that result from and lead to further neural hypersensitivity/hyperexcitability.

The neurological hypersensitivity proposed as a factorial constituent of PTSD may possess several pathognomic and sustaining mechanisms (see Everly & Lating, 2004, for a review):

1. Increased excitatory neurotransmitter activity within the limbic circuitry (Black et al., 1987; Post, 1985; Post & Ballenger, 1981; Post, Weiss, & Smith, 1995; Post, Rubinow & Ballenger, 1986; Sorg & Kalivas, 1995).
2. Declination of inhibitory neurotransmitters and/or receptors (Cain, 1992; see Everly, 1993).
3. Augmentation of micromorphological structures (especially amygdaloidal and hippocampal dendritic branching) (Cain, 1992; Post et al., 1995; see Everly, 1993).
4. Changes in the biochemical bases of neuronal activation, for example, augmentation of phosphoproteins and/or changes on the transduction mechanism *c-fos* so as to change the genetic message within the neuron's nucleus (Cain, 1992; Horger & Roth, 1995; Sorg & Kalivas, 1995).
5. Increased arousal of neuromuscular efferents, with resultant increased proprioceptive bombardment of the limbic system (especially amygdaloidal and hippocampal nuclei) (Gellhorn, 1964b, 1968; Malmo, 1975; Weil, 1974).
6. Repetitive cognitive excitation (Gellhorn, 1964b, 1968; Gellhorn & Loofburrow, 1963; Post et al., 1986).

While examining the physiological bases of PTSD, a more specific look at the neurochemistry seems in order. It is clear that excitatory neurotransmitter activity is an essential component of the presentation of PTSD. Specifically, central amino acids such as glutamate and aspartate are implicated in hyperarousal as well as excitotoxic effects (Everly, 1995; Bermudo-Soriano, Perez-Rodriguez, Vaquero-Lorenzo, Baca-Garcia, 2012; Nair & Ajit, 2008). Corticotropin-releasing factor (CRF), endogenous opioids, vasopressin, and oxytocin are also implicated in extreme stress arousal (Selye, 1976; Rossier, Bloom, & Guillemin, 1980; Rochefort, Rosenberger, & Saffran, 1959; Nair & Ajit, 2008). Finally, there is evidence that serotonin, dopamine, and certainly norepinephrine play significant roles in extreme stress (Kolb, 1987; Sorg & Kalivas, 1995; van der Kolk et al., 1985; Charney, 2004).

The excitatory processes inherent in PTSD are not limited to the CNS. The mobilization of neuroendocrine and endocrine pathways carries the posttraumatic stress response throughout the human body. Especially implicated are the sympathoadrenomedullary (SAM) (Everly, 1990; Everly & Lating, 2004) and the hypothalamic–pituitary–adrenal

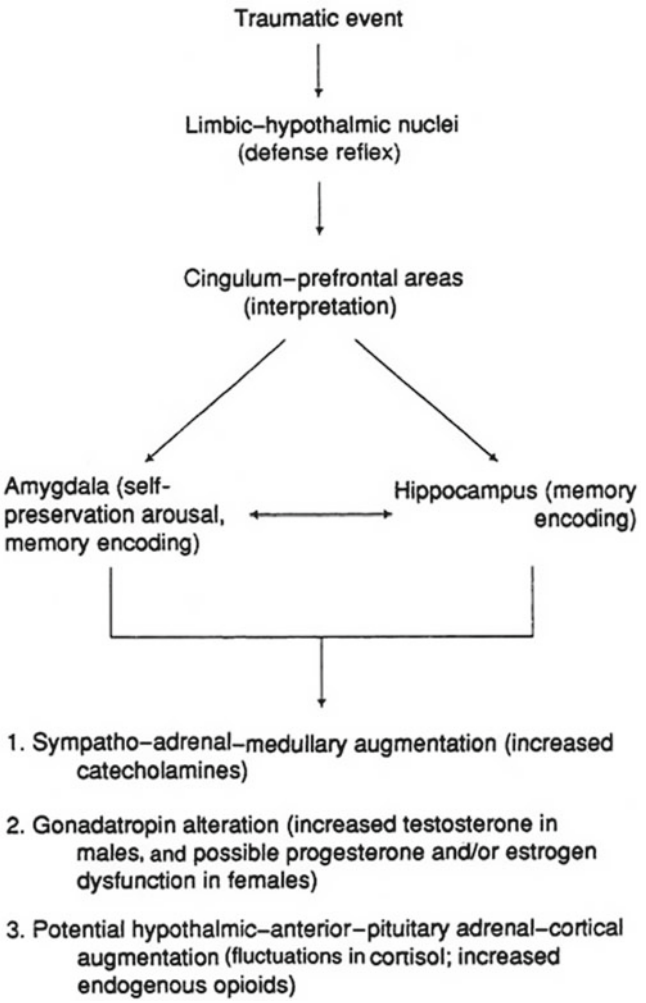

Fig. 21.3 Neurobiology of posttraumatic stress disorder

(HPA) systems (Yehuda, Giller, Levengood, Southwick, Siever, 1995; Everly & Lating). Figure 21.3 summarizes some of the key elements involved in the biology of PTSD.

Psychological Hypersensitivity

Psychological hypersensitivity is thought to arise from a violation of some deeply held belief, referred to as a worldview, or *Weltanschauung* (Everly, 1993, 1994, 1995; Everly & Lating, 2004). Thus, according to this perspective, a traumatic event

is predicated upon some situation that violates a deeply held and important world-view. Most commonly, we think of the traumatic event as a life-threatening event—a violation of the assumption of safety discussed by writers such as Maslow (1970). But there appear to be at least five universally traumatogenetic themes:

1. Violation of the belief that the world is "just" or "fair." For example, why does an infant die in a motor vehicle accident?
2. Violation of a sense of who you are by having not done something you should, or by having done something you should not have done.
3. Abandonment, betrayal, and violation of trust.
4. Violation of a universal sense of safety.
5. Disruption of a religious or spiritually based belief.

Within his construct of "psychotraumatology," Everly (1993, 1994, 1995; Everly & Lating, 2004) discusses these issues in greater detail.

The Psychological Profile of Posttraumatic Stress Disorder

The work of Keane and his colleagues has been preeminent in the search for the psychological PTSD prototype. Using patients evaluated with the Minnesota Multiphasic Personality Inventory (MMPI) and 100 patients with other psychiatric diagnoses, Keane, Malloy, and Fairbank (1984) were able to identify an MMPI profile capable of correctly classifying 74% of all patients. The MMPI decision rule was $F \geq 66$, Depression (2) ≥ 78, and Schizophrenia (8) ≥ 79 (using *T* scores). Item analysis led to the creation of a 49-item MMPI PTSD subscale, the PTSD-Keane (PK) subscale (Keane et al.), that correctly identified 82% of the patients studied. On this MMPI subscale, patients who scored 35 out of 49 had an 87% chance of possessing a valid PTSD diagnosis, whereas patients who scored above 40 had a 90% chance of a true positive PTSD diagnosis. The MMPI-2 (Butcher et al., 1989, 2001) uses 46 items for PK subscale, and in a sample of veterans, a cut-off score of 28 correctly classified 76% of the overall sample, 67% of the PTSD group and 85% of the non-PTSD comparison group (Munley, Bains, Bloem, & Busby, 1995).

In a cross-validation of the aforementioned MMPI PTSD subscale, Fairbank, McCaffrey, and Keane (1985) found that patients with a *T* score above 88 on the *F* scale were most likely to possess a factitious disorder. Thus, the *F* decision rule became $66 \leq F \leq 88$ and correctly identified 93% of the sample studied when combined with the previous (2) ≥ 78 and (8) ≥ 79. On the MMPI-2, Tolin and associates (2004) reported that the Infrequency-Psychopathogy *F(p)*scale, which consists of 27 items developed to distinguish between overreporting and accurate reporting of severe distress (Arbisi & Ben-Porath, 1995), is effective in determining over-reporting in Vietnam veterans being assessed for PTSD. In a study using the MMPI-2 to assess PTSD in a study of 90 trauma-exposed undergraduates, the MMPI-2 clinical scales of 7 (psychasthenia), 2 (depression), and 3 (hysteria), the content scales of Anxiety, Work Interference, and Low Self Esteem (LSE), and the two PTSD

subscales (PK and PTSD-Schlenger (PS) subscale (Schlenger & Kulka, 1987)) discriminated between PTSD and a well-adjusted control group (McDevitt-Murphy, Weathers, Flood, Eakin, & Benson, 2007).

The work of McDermott (1987) sought to extend the psychometric diagnosis of PTSD beyond the MMPI. Using the Millon Clinical Multiaxial Inventory (MCMI), McDermott evaluated 22 Vietnam combat veterans, 11 of whom had been diagnosed with PTSD. The results of his study indicate that PTSD patients may present elevations on the MCMI schizoid and avoidant scales ($x>80$), with a concomitant depression on the histrionic scale.

The MCMI-III contains a scale (R) that purports to assess PTSD with a 53% sensitivity and a positive predictive power of 73%. But the MCMI-III aggregate configural profile may take several forms:

1. Aggregate elevations on Schizoid, Avoidant, and Negativistic (passive–aggressive) scales are often viewed as the withdrawing "flight" variant of the MCMI posttraumatic stress profile.
2. Aggregate elevations on the Narcissistic, Aggressive, and Antisocial scales may be viewed as the aggressive "fight" variant of the MCMI posttraumatic stress profile.
3. Aggregate elevations on the Negativistic, Self-Defeating, Schizoid/Avoidant, Aggressive, and Borderline scales may be viewed as the affectively labile profile that is often characteristic of "complex PTSD" (i.e., indicative of early developmental trauma, abuse, and/or neglect).

Based on Kolb's (1987) hypothesis that PTSD represents a partial cognitive deficit, in combination with the belief that PTSD resides within the hippocampal complex, Everly and Horton hypothesized that there would be a short-term memory deficit among PTSD patients. Using 15- and 30-s trials of the Peterson Memory Paradigm, these authors found that 9 out of 14 (65%) non-combat-related PTSD patients failed to meet the 55% correct cutting-line criterion for the 15-s trials, and 11 out of 14 (79%) patients failed to meet the 45% correct cutting-line criterion for the 30-s trials. These data served to support the hypothesis that PTSD patients are likely to possess a cognitive deficit that manifests as an impairment to immediate and short-term memory function. Long-term memory was unimpaired in these participants. In one of the first controlled neuropsychological studies assessing active-duty Army soldiers, and after controlling for deployment-related heard injury, stress and depression, results revealed that Iraq deployment, when compared with non-deployment, was associated with reduced sustained attention, verbal learning, and visual-spatial memory, yet improved performance on a test of simple reaction time (Vasterling et al., 2006). Moreover, on self-report measures, deployment was associated with confusion and tension.

With the wars in Iraq and Afghanistan that have occurred in the last decade, there has been increased focus on the prevalence of PTSD in returning veterans. As noted earlier in this chapter, as many as 50% of Iraqi and Afghanistan veterans seeking treatment screen positive for PTSD. However, Ramchand and colleagues (2010) note in this same article that the prevalence rates in the studies they reported

were as low as 4%. The authors attribute this variability to a number of factors, including the varied and multiple methods of assessment and diagnostic criteria used to determine a diagnosis of PTSD. Although there are inherent challenges in determining an accurate assessment of a PTSD psychological profile, the International Society for Traumatic Stress Studies (ITSS; Foa, Keane, Friedman, & Cohen, 2009) suggests using the following categories of evidence-based measures to complete a comprehensive PTSD assessment (1) structured diagnostic interviews, such as the Clinician-Administered PTSD Scale (CAPS) (Blake et al., 1990) or the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Williams, & Gibbon, 2000), and (2) self-report measures, such as the MMPI-2, MCMI, or one of the many specific PTSD scales (e.g., PTSD Checklist; (PCL; Weathers, Litz, Herman, Huska, &, Keane, 1993)). According to Castro, Hayes, and Keane (2011), accurate assessment of PTSD within a military population is required to: (1) facilitate treatment planning, (2) allow for research progress, (3) provide valuable information used for policy making, and (4) to determine disability benefits.

Treatment of Posttraumatic Stress Disorder

This chapter has presented posttraumatic stress as a quintessential example of psychological and biological factors combining in an inextricable integration. Our two-factor model of PTSD implies that the recovery from posttraumatic stress is predicated upon improvement in both domains. This is not to say that every patient who suffers from PTSD requires medication, but it does suggest that the more severe the manifest symptomatology, the more psychotropic medications should be considered as an addition to the therapeutic mix. It is doubtful, however, that any PTSD patient has ever recovered on the basis of psychotropic medication alone. Because the "injury" is a psychological one, recovery will be based upon some alteration in the "psychological domain." That psychotherapeutic improvement may be greatly facilitated, indeed, by the addition of psychopharmacological agents. In instances when the amplitude of the neurological pathology has become self-sustaining, psychopharmacological agents will be mandatory. Let us take a look at current issues in the treatment of posttraumatic stress.

Psychopharmacotherapy

A wide variety of psychopharmacological agents have been used in the treatment of PTSD. As van der Kolk (1987) has stated, "Psychotherapy is rarely helpful as long as the patient continues to respond to contemporary events and situations with a continuation of physiological emergency reactions" (p. 75).

A review of the pharmacological treatment of PTSD has been offered by Platman (1999) and lists psychotropic agents for consideration by virtue of the symptoms they tend to target:

- *Learned helplessness*—clonidine, benzodiazepines, tricyclics (TCAs), and monoamine oxidase inhibitors (MAOI)
- *Hyperstartle response*—clonidine
- *Intrusive ideation*—selective serotonin reuptake inhibitors (SSRIs), TCAs, and MAOIs
- *Panic*—alprazolam and clonazepam
- *Depressed mood and avoidance*—SSRIs
- *Impulsive rage*—lithium and carbamazepine
- *Sleep disturbance*—trazodone

In 1999, the *Journal of Clinical Psychiatry* published findings from its Expert Consensus Panel for the Treatment of PTSD (Foa, Davidson, & Frances, 1999). The Panel recommended that psychopharmacological intervention either be followed or used in combination with psychotherapy for both acute and chronic PTSD. The medications of choice were the SSRIs. If no response was achieved, the Panel recommended that nefazodone or venlafaxine be initiated. If a partial positive response was achieved, it recommended a mood stabilizer in addition to the SSRI.

Given that some researchers consider severe posttraumatic stress a form of kindling, or subcortical ictus, the question of the utilization of mood stabilizers (carbamazepine and divalproex) as a primary medication becomes a relevant issue.

The UK's National Institute for Clinical Excellence (NICE, 2005) made the following recommendations: "Drug treatments for PTSD should not be used as a routine first-line treatment for adults (in general use or by specialist mental health professionals) in preference to a trauma-focused psychological therapy. Drug treatments (paroxetine or mirtazapine for general use, and amitriptyline or phenelzine for initiation only by mental health specialists) should be considered for the treatment of PTSD in adults who express a preference not to engage in trauma-focused psychological treatment" (NICE, p.5). In a review of consensus guidelines, such as NICE's, and several meta-analytic studies of pharmacotherapy in the treatment of PTSD, Stein, Ipser, & McAnda (2009) support that SSRIs, or some SSRIs and venlafaxine (a serotonin norepinephrine uptake inhibitor (SNRI)) are good first-line treatments.

Psychotherapy

The Expert Consensus Panel for the Treatment of PTSD (Foa et al., 1999) has recognized the superordinate role that psychotherapy initially plays in the therapeutic arsenal. The report notes that anxiety management, psycho-education, and cognitive therapy appear to be the safest and most acceptable psychotherapeutic

interventions. This recommendation is echoed in part by NICE (2005). The NICE guidelines state:

> "Trauma-focused cognitive behavioural therapy should be offered to those with severe post-traumatic symptoms or with severe PTSD in the first month after the traumatic event. These treatments should normally be provided on an individual outpatient basis. All people with PTSD should be offered a course of trauma-focused psychological treatment (trauma-focused cognitive behavioural therapy [CBT] or eye movement desensitisation and reprocessing [EMDR]). These treatments should normally be provided on an individual outpatient basis" (NICE, p. 4).

The work of Meichenbaum (1977, 1994) stands as a significant contribution in this regard. His treatise on the treatment of PTSD from a cognitive-behavioral perspective represents a powerful multidimensional approach to this complex and challenging disorder (Meichenbaum, 1994; also see Foy, 1992).

Other valuable resources in the area of treatment formulation for PTSD include the work of Foa, Keane, & Friedman (2000) and Flannery (1992). Wilson, Friedman, and Lindy (2001) offer an integrative perspective on PTSD treatment, and Wampold and colleagues (2010) provide a more recent review in determining what works in PTSD treatment.

Group therapy interventions have shown significant promise and have been summarized by van der Kolk (1987). The rationale for the use of group psychotherapy for PTSD includes the provision of peer support, a safe venue for therapeutic abreaction, consensual validation, and the minimization of regression and avoidance. A recent meta-analytic analysis of the efficacy of group treatment for PTSD (Sloan, Feinstein, Gallagher, & Beck, 2011) showed that while group treatment is better than no treatment for PTSD, it was not superior when compared to active treatment conditions that were used in the studies to control for nonspecific therapy effects (e.g., supportive therapy group). Moreover, the results indicate that group treatment might be less effective with men than women, and also in conditions where there is repeated trauma, such as child abuse and combat trauma, or more chronic PTSD.

Neurocognitive Strategic Therapy for Posttraumatic Stress

Everly (1993, 1994, 1995; Everly & Lating, 2004, 2005) has posited that posttraumatic stress represents a two-factor phenomenon (i.e., two inextricably intertwined factors that make up its core essence): (1) neurological hypersensitivity and (2) psychological hypersensitivity. We reviewed their respective constituencies earlier in this chapter, so we shall not reiterate them here. It may be argued that treatment should be the natural corollary of phenomenology. If so, then treatment formulations for posttraumatic stress reactions, including ASD and PTSD, should parallel, or match, their phenomenology. To put it another way, the treatment of posttraumatic stress reactions, including ASD and PTSD, should possess a two-factor constituency so as to match the two-factor phenomenology of the disorder.

Everly (1994, 1995) has proposed that a neurocognitive strategic treatment formulation for posttraumatic stress is likely to prove the most effective and is clearly

the most theoretically sound. By way of explanation, it is clear that numerous therapies are effective for posttraumatic stress. In that it is unclear that any given "brand name" tactic is always superior to any other given tactic, Everly offers a strategic formulation for the treatment of posttraumatic stress rather than recommending a specific tactical approach. This strategic formulation recommends a phenomenologically driven *approach* to therapy rather than a specific *technique* for therapy. Thus, Everly suggests that *neurological desensitization* techniques should be used to address the neurological sensitivity of posttraumatic stress and be combined with techniques that address the *cognitive schemas* that have been threatened or destroyed by the traumatic event. Techniques for neurological desensitization might include meditation, Yoga, physical exercise, massage, neuromuscular relaxation techniques, hypnosis, psychotropic medications, imagery, and so on. Techniques to address the endangered cognitive schemas might include cognitive therapy, cognitive-behavioral therapy, dynamic therapies, group therapy, behavior therapy, and so on.

Eye Movement Desensitization and Reprocessing

Eye Movement Desensitization and Reprocessing (EMDR) may represent a unique example of an integrated neurocognitive therapy in that it may address both the neurological hypersensitivity and the cognitive schemas within the same therapeutic paradigm, virtually simultaneously. EMDR is a therapeutic method originated by Francine Shapiro in 1987, when she indiscriminately discovered that recurring, disturbing thoughts rapidly and permanently disappeared when she engaged in rapid, saccadic eye movements (Shapiro & Solomon, 1995). Shapiro first published her work in 1989 as EMD and reported on the successful controlled treatment of 22 rape/molestation victims and Vietnam veterans, using a one-session application that included as part of the protocol having the participant follow the repeated side-to-side movement of her fingers (Shapiro, 1989). The impressive treatment gains were maintained at a 3-month follow-up. The results of Shapiro's initial work generated tremendous excitement in area of PTSD treatment; however, it also raised considerable skepticism because of the lack of validated PTSD measures employed and the possibility of placebo effects, including demand characteristics (Feske, 1998).

The intense research scrutiny that resulted from the introduction of EMDR led in a relatively short period of time to numerous applied studies. However, the overall results of early studies of EMDR were largely equivocal due primarily to flawed methodology, poor experimental design, and inadequate treatment delivery (i.e., inexperienced or minimally trained therapists providing the treatment) (Shapiro, 1999). According to Shapiro (1999), it is important to acknowledge that EMDR is "an integrated form of therapy incorporating aspects of many traditional psychological orientations and one that makes use of a variety of bilateral stimuli besides eye movement" (p. 37). In fact, treatment effectiveness has been reported for bilateral auditory stimulation therapist (e.g., snaps fingers nearer one ear of the patient than the other) and bilateral tactile stimulation (e.g., participant rests palms on his or her knees and therapist alternatively taps the palms) (Lipke, 2000).

Therefore, the emphasis on eye movements is actually a misconception, but one that is certainly understandable given the name of the process. Shapiro is also quick to emphasize that other quite salient, nonspecific elements account for therapeutic success (Shapiro, 1995, 1999). She proposes that the general model of EMDR is predicated on the notion of accelerated information processing, which states that "there is an innate physiological system that is designed to transform disturbing input into an adaptive resolution and a psychologically healthy integration" (Shapiro, 1995, p. 53).

Within the past decade there have been numerous studies assessing the treatment efficacy of EMDR. Seidler and Wagner (2006) in a meta-analysis compared seven studies of "trauma-focused" cognitive behavioral therapy (CBT) to EMDR, and the results showed that both treatments were effective and that there were no differences between the two. A systematic review and meta-analysis of 38 randomized controlled trials of psychological treatments for chronic PTSD (participants need to have PTSD symptoms for at least 3 months) from search engine databases as well as the Cochrane Library, revealed that the treatments most supported were individually trauma-focused cognitive-behavioural therapy (TFCBT) and EMDR (Bisson et al., 2007). A meta-analysis of the efficacy of using EMDR in children with PTSD revealed an overall medium effect size ($d = 0.56$), suggesting that children benefited from EMDR treatment when compared to non-established trauma treatment or no-treatment control groups (Rodenburg, Benjamin, Roos, Meijer & Stams, 2009). Moreover, when compared to children receiving CBT, EMDR was shown to add small, but significant incremental efficacy. Overall, these results are supportive of EMDR as a treatment approach for PTSD, and also are clearly consistent with a neurocognitive treatment strategy for PTSD.

One of the most supported empirically based treatments for PTSD in the past two decades has been prolonged exposure (PE) therapy, a manualized 9–12 session treatment which emphasizes reduction of avoidance through repeated imaginal and in-vivo exposure (Foa, Rothbaum, Riggs, & Murdock, 1991; Foa et al., 1999; Nemeroff et al., 2006). The recent and impressive advances in virtual reality techniques have allowed exposure therapy to create much more realistic and somatosensory salient (i.e., sights, sounds, and smells) treatment environments, particularly when working with recent war veterans (Rizzo et al., 2011). We envision these virtual advances to continue and to expand PE to more diagnostic domains. In a study comparing the efficacy, speed, and adverse effects of exposure therapy, EMDR, and relaxation training in a sample of 60 participants (97% of whom were diagnosed with chronic PTSD), the results revealed that all three treatments were effective, but compared with EMDR and relaxation training, exposure therapy was the most efficacious in reducing experiencing and avoidance symptoms, more rapidly reduced avoidance, and resulted in the highest proportion of participants no longer meeting PTSD diagnostic criteria (Taylor et al., 2003). In a more recent meta-analytic review of PE for PTSD, Powers, Halpern, Ferenschak, Gillihan, and Foa (2010) reported on the treatment success of PE (86% of patients receiving PE fared better than control conditions), but noted as well that PE was no more effective than other active treatments, such as EMDR, stress inoculation training (SIT), or other cognitive therapies (see Chap. 8).

In the final analysis, and as suggested by Horowitz (1974) close to 40 years ago, psychotherapy should be directed toward cognitive control, improving self-image and interpersonal relationships, decreasing stress, and working through the "meaning" of the trauma. As noted by writers such as Janoff-Bulman (1992), addressing the "meaning" of the trauma becomes a pivotal aspect of the recovery process. This is clearly consistent with the two-factor model of PTSD introduced in this chapter, and also with the overarching formulation of the human stress response as used throughout this text, in that the "interpretation," or meaning, of the stressor event serves to contribute to the intensity and chronicity of the stress response itself.

Obviously, the treatment of PTSD needs to be tailored to the specific needs of the individual patient. Not only must the clinician consider the manifest symptomatology, but he or she must also strive to understand the "meaning" of the traumatic event. Once the symptoms have been stabilized and no longer represent a barrier to psychotherapy, the focus of the therapeutic process should most likely turn to the endangered or compromised belief about the world, or oneself, that lies at the foundation of the posttraumatic response (Everly, 1993, 1994, 1995).

Summary

This chapter addressed the subject of PTSD. Historically, in its more severe forms, PTSD has led to permanent partial disabilities. In some cases, permanent total disabilities have resulted. Because of the prevalence and propensity to remain undiagnosed for protracted periods of time, this stress-related disorder has been included in the present volume. Let us review the main points:

1. PTSD is generally thought to possess four key phenomenological constituents: (a) the presence of stressful experience generally acknowledged as being outside the usual realm of human experience; (b) intrusive, recollective experiences; (c) ANS hyperactivity; and (d) avoidance and numbing symptoms.
2. Within this chapter, we have argued that the "essence" of PTSD is the intrusive, recollective experience in combination with the ANS hyperfunction. The avoidant and numbing symptoms have been reformulated as attempts by the patient to control the pathological syndrome. Exposure to a stressor remains a necessary but insufficient diagnostic criterion.
3. Once viewed in the context of a combat-related syndrome, PTSD is now recognized as having the potential to arise out of virtually any life-threatening experience. Recent evidence has even suggested that PTSD can arise out of an accumulation of stressor experiences; exposure to certain solvents, toxins, and stimulants; and the experience or observation of traumatic, but not necessarily life-threatening, events such as the loss of personal property and/or physical injury.
4. Once suggested as residing within the hindbrain, PTSD has been reformulated from a physiological perspective as residing primarily as a condition of neurological hypersensitivity within the noradrenergic projections of the septal–

amygdalar–hippocampal complexes. Potential causes of the neuronal hypersensitivity include an augmentation of tyrosine hydroxylase, an increase in beta-1 postsynaptic excitatory receptors, a decrease in alpha-2 presynaptic inhibitory receptors, and an increase in postsynaptic dendritic spines.

5. Attempts to identify the psychological profile of the PTSD patient have focused upon the use of the MMPI. The $66 \leq F \leq 88$, $(2) \geq 78$, and $(8) \geq 79$ and decision rule for the MMPI seems a useful starting point. Other research utilizing the MCMI has found elevations on the Schizoid and Avoidant subscales, coupled with a diminution of the Histrionic subscale to be useful in identifying PTSD patients. Research has also found an impairment of short-term memory among PTSD patients. Finally, it should be noted that PTSD patients may frequently be misdiagnosed as sociopathic, hypochondriacal, and/or as substance abusers.
6. From a treatment perspective, PTSD, especially in its chronic forms, may require a combination of psychotherapeutic and pharmacological efforts to be truly effective. Antidepressants and anticonvulsants continue to be promising agents for the cases wherein psychotherapy alone seems insufficient.
7. Strategically, a two-factor neuro-cognitive strategic formulation for conceptualizing the treatment of posttraumatic stress, was offered.

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