Managing Anger and Aggression in Patients With Posttraumatic Stress Disorder

Rachel Yehuda, Ph.D.

Posttraumatic stress disorder was categorized as a clinical entity in 1980 in response to assertions by trauma survivors (particularly Vietnam veterans) and their clinicians that existing diagnostic categories failed to adequately describe their symptoms. The diagnosis first appeared in the 1980 edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III). Prior to establishment of the new category, most individuals who developed symptoms in response to traumatic events were considered to be neurotic, and a diagnosis was sought that legitimized both the acute and chronic symptoms that occurred in response to trauma. Thus, the political consciousness of the era provided a major impetus for implementing the diagnosis of posttraumatic stress disorder.1 There are occasional discrepancies between the ideology and the reality of posttraumatic stress disorder; the disorder was modeled after Vietnam veterans and may not be representative of all trauma survivors. Nevertheless, scientific information is emerging that has resulted in important insights into the reality of posttraumatic stress disorder, and much of the information has critical treatment implications. It is now clear that posttraumatic stress disorder provides a model for a process of adjustment to and destabilization from trauma that has biological, psychological, and phenomenological dimensions. This article will discuss the evolving concept of posttraumatic stress disorder as a clinical entity, the association of anger and aggression with the disorder, and the psychopharmacologic approaches to treatment.

*(J Clin Psychiatry 1999;60[suppl 15]:33–37)*

**DIAGNOSTIC FEATURES OF POSTTRAUMATIC STRESS DISORDER**

In the DSM-III, the essential feature of posttraumatic stress disorder was the development of characteristic symptoms following exposure to a psychologically traumatic event that was generally regarded as outside the range of usual human experience.2 Since some estimates suggest that 30% to 90% of individuals are exposed to traumatic events, the definition was subsequently refined, and subjective criteria were added. As a result, the distinguishing features of the current DSM-IV definition of posttraumatic stress disorder are that characteristic symptoms occur after exposure to, witnessing of, or learning about an event that causes actual or threatened physical harm and leads to a subjective response of intense fear, helplessness, or horror.5 The lifetime prevalence rate of posttraumatic stress disorder is 7.8%,3 and the likelihood of developing the disorder may increase as the intensity of and physical proximity to the stressor increase.

Three discrete symptom clusters are associated with exposure to a traumatic event: (1) Intrusive symptoms are characterized by recurrent unwanted memories of the event, distress at reminders, reexperiencing the event, nightmares, and physiologic distress. (2) Avoidance symptoms can be active or passive. Active avoidance is the de-
liberate avoidance of thoughts, feelings, or reminders of the trauma, and may include psychogenic amnesia. Passive avoidance is characterized by diminished interest in previously enjoyed activities, detachment, a restricted emotional range, and the sense of a foreshortened future. (3) Hyperarousal symptoms include insomnia, irritability, difficulty concentrating or completing tasks, hypervigilance, and exaggerated startle response (Table 1). Symptoms usually begin within weeks or months after the traumatic event occurs, although there may be a delay of months or years before symptoms become apparent. The full symptom picture must have been present for at least 1 month, and the disturbance must cause clinically significant distress or impairments in social, occupational, or other important areas of functioning for diagnostic criteria to be fulfilled.

The majority of persons who develop posttraumatic stress disorder immediately after a traumatic event occurs will recover within a few months or years. Approximately one third of persons who develop the disorder fail to recover, and a substantial number of trauma survivors develop delayed or reactivated posttraumatic stress disorder. Trauma survivors who seek treatment for symptoms of posttraumatic stress disorder are usually individuals who have multiple problems. Posttraumatic stress disorder rarely occurs in "pure" form, and individuals who have the disorder commonly meet criteria for Axis I and Axis II disorders. The prevalence of comorbid psychiatric conditions and posttraumatic stress disorder has been investigated in a number of traumatized groups, and anywhere from 50% to 90% of persons with chronic posttraumatic stress disorder also meet diagnostic criteria for another psychiatric disorder. Comorbid Axis I disorders include mood disorders (particularly depression and bipolar disorder), substance abuse, and other anxiety disorders. Posttraumatic stress disorder, especially in the chronic state, can also be associated with numerous Axis II personality disorders.

Associated features of the disorder include anger, aggression, alexithymia, guilt feelings, and impulsiveness.

### Anger and Aggression

If anger and aggression are acknowledged features of posttraumatic stress disorder, one might ask why they were not included as diagnostic criteria. To answer this provocative question, one must recall the political milieu of 1980 when the criteria were first formulated. One purpose of the diagnosis was to avoid blaming the victim, and to have included aggression in the criteria might have been seen as critical of the victim. Indeed, trauma survivors can induce a countertransference response among therapists such that the survivors are viewed as victimizers rather than victims. Moreover, anger and irritability were included among the symptoms as an acknowledgment of these types of posttraumatic feelings. Research is now emerging that clearly supports the prevalence of aggression in posttraumatic stress disorder.

### Table 1. Posttraumatic Stress Disorder (PTSD) Symptom Clusters

<table>
<thead>
<tr>
<th>Cluster</th>
<th>Symptoms</th>
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<tbody>
<tr>
<td>Intrusive symptoms</td>
<td>Recurrent, unwanted memories of event, Nightmares, Reliving event/stress at reminders</td>
</tr>
<tr>
<td>Avoidance symptoms</td>
<td>Active (Avoiding thoughts or feelings, Reminders, Psychogenic amnesia), Passive (Diminished interest, Detachment, Restricted emotional range, Foreshortened future)</td>
</tr>
<tr>
<td>Hyperarousal symptoms</td>
<td>Insomnia, Irritability, Concentration, Hypervigilance, Startle</td>
</tr>
</tbody>
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*Adapted from reference 5.*

Although most investigations on aggression associated with a traumatic stress response have centered around Vietnam veterans, aggressive behavior was previously described in World War I and World War II veterans by Kardiner in 1941. Aggression associated with posttraumatic stress disorder has also been observed in Vietnamese refugees who resettled in Norway, and emerging data in Holocaust survivors also reveal features of anger and aggression—although less commonly than behavioral inhibition. In an investigation of interpersonal violence among Vietnam veterans, Beckham et al. reported 22 acts of aggression over a 1-year period in combat veterans with posttraumatic stress disorder compared with 0.2 acts reported by combat veterans without posttraumatic stress disorder; the reported aggression was unassociated with alcohol problems or childhood physical abuse. Other studies similarly report increased aggression associated with and without posttraumatic stress disorder. In a survey of 50 couples in which the male partner was a Vietnam veteran with symptoms of posttraumatic stress disorder, significant aggressive behavior was demonstrated against the female partners. Over a 1-year period, 34% of the veterans engaged in at least 1 act of physical violence, 92% used verbal aggression, and 100% practiced psychological aggression against their partner. Thus, the need for treatment of anger and aggression exists, and veterans are generally amenable to the idea of pharmacotherapy in these instances because it validates their symptoms as real issues.

**TREATMENT**

The approach to treatment of posttraumatic stress disorder varies with the perception of the disorder. In the
past, posttraumatic stress disorder was defined as a normal response to traumatic events. Thus, the conventional approach has focused on the traumatic event as the direct cause of symptoms, and treatment consisted of psychodynamic and cognitive-behavioral approaches. The psychodynamic approach integrates traumatic events into a meaningful structure and attempts to modify the patient’s poor defense mechanisms and coping strategies by understanding why they were present. The cognitive-behavioral approach focuses on posttraumatic stress disorder as a conditioned fear response that could be reversed through systematic desensitization, flooding, monitoring and altering responses to triggers to produce less fear, modifying mistaken perceptions—e.g., the trauma could have been anticipated or avoided—and understanding maladaptive responses.

Although these treatments are effective in treating symptoms of posttraumatic stress disorder, it is often necessary to combine them with other modalities, particularly pharmacotherapy. Many clinicians who treat posttraumatic stress disorder are nonphysicians and the use of psychopharmacology or the combination of medications with other forms of treatment has probably not been as pervasive as it could have been.16

Two independent schools of thought on the role of pharmacotherapy have evolved. One school of thought is that medication can be used as an adjunct to trauma-focused therapy by reducing abraeactions to exposure and by treating comorbid symptoms. A therapist may feel that the most important aspect of treatment is to understand the impact of the traumatic event and will therefore utilize psychodynamic and cognitive-behavioral therapy. However, if therapy techniques such as flooding or systematic desensitization cause the patient to become exceedingly aroused, a medication may be given as a calming agent. It would follow that medications such as anticonvulsants can be particularly helpful if the patient is impulsive and aggressive or is exhibiting behavior that is preventing trauma-focused therapy. The second school of thought is quite different; adherents believe that posttraumatic stress disorder is not the normal consequence of response to trauma, but rather that the disorder itself reflects a maladaptive process owing to alterations in biological systems. Therefore, adherents of the second school believe that medications directly address the maladaptive processes and reduce symptoms of the disorder.

Pharmacotherapy

Published studies, especially randomized clinical trials, on the pharmacotherapy of posttraumatic stress disorder are limited in number. Notwithstanding, pharmacotherapy of the disorder is evolving from an experimental approach—based on using medications that are effective in related and comorbid mood and anxiety disorders—to a more rational approach formulated on the basis of discrete biological alterations in posttraumatic stress disorder. Patients with the disorder undoubtedly have features of depression, anxiety, and other psychiatric disorders, but there are also circumscribed biological changes peculiar to posttraumatic stress disorder that compel the clinician to have a rationale for treatment. For example, the use of antihypertensives has been prompted by the findings of increased catecholamine activity17 and yohimbine-induced panic attacks and flashbacks6 in patients with posttraumatic stress disorder.

Most medications benefit some symptoms, but rarely does a single medication affect all symptom clusters equally or produce remission of the syndrome. Accordingly, it is reasonable to think about specific medications as beneficial for particular symptoms or clusters of posttraumatic stress disorder (Table 2). Monoamine oxidase inhibitors benefit intrusive symptoms and sleep; tricyclic antidepressants improve mood, obsessions, intrusive, and arousal symptoms; selective serotonin reuptake inhibitors (SSRIs) benefit symptoms of intrusive, sleep; antidepressants improve mood, obsessions, intrusive, and arousal symptoms; selective serotonin reuptake inhibitors (SSRIs) benefit symptoms of avoidance and hyperarousal; antihypertensives improve arousal symptoms; and anticonvulsants benefit symptoms of explosiveness, arousal, impulse control, and sleep. Symptom clusters of avoidance and hyperarousal are generally more disturbing to the patient than intrusive symptom clusters; nightmares—which are intrusive symptoms—can be devastating, but they rarely occur in the absence of avoidance and hyperarousal symptoms. Most trauma survivors, including Holocaust survivors, can withstand intrusive symptoms and may even suffer residual intrusive symptoms long after other symptoms have abated. On the other hand, avoidance symptoms—such as impairments in relationships (inability to get close to people, distancing, and numbing)—and hyperarousal symptoms (particularly hypervigilance, irritability, and inability to sleep) are symptoms that disable trauma survivors by perpetuating their isolation, weakening their social supports, and obliging them to seek treatment.

One primary goal of treatment in patients with posttraumatic stress disorder is mood stabilization. In 1984, van der Kolk et al. administered lithium to 14 combat veterans and found that hyperarousal symptoms and alcohol use improved in 8 veterans.18 Another study clearly demonstrated that lithium improved explosiveness and arousal.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Symptom Benefits</th>
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<tr>
<td>Monoamine oxidase inhibitors</td>
<td>Intrusive, sleep</td>
</tr>
<tr>
<td>Tricyclic antidepressants</td>
<td>Mood, obsessions, intrusive, arousal</td>
</tr>
<tr>
<td>Selective serotonin reuptake inhibitors</td>
<td>Avoidance, hyperarousal</td>
</tr>
<tr>
<td>Antihypertensives</td>
<td>Arousal</td>
</tr>
<tr>
<td>Anticonvulsants</td>
<td>Explosiveness, arousal, impulse control, sleep</td>
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in 4 of 5 combat veterans. Lipper et al. suggested that a kindling model or a model of a paroxysmal disorder might be applicable to posttraumatic stress disorder and that intrusive symptoms might respond to anticonvulsant treatment because they were the symptoms most likely to be considered kindled (or paroxysmal) experiences.

In a review of valproate and carbamazepine treatment of panic and posttraumatic stress disorders, Keck et al. reported that behavioral and neurobiological sensitization might be relieved by raising the neuronal threshold for arousal in limbic areas. Two open trials of carbamazepine treatment and 1 open trial of divalproex sodium indicated that the anticonvulsants were particularly effective in reducing explosiveness, which is a major problem in Vietnam combat veterans. In a 5-week open trial of carbamazepine, 7 of 10 Vietnam veterans who met DSM-III criteria for posttraumatic stress disorder manifested moderate-to-much improvement on the Clinical Global Impressions scale. Among the many symptoms of the disorder, nightmares, flashbacks, and intrusive recollections were selectively reduced in intensity and frequency. A total of 8 of 10 Vietnam veterans with posttraumatic stress disorder treated with carbamazepine showed significant improvement, especially in impulse control and preventing violent behavior or angry outbursts. In an open clinical trial of divalproex, 10 of 16 patients diagnosed with DSM-III-R combat-related posttraumatic stress disorder showed significant improvement, especially in hyperarousal/hyperactivity symptoms.

Combat veterans with posttraumatic stress disorder are complicated patients who have historically, but not always, presented as refractory to pharmacotherapy. The frequency of comorbid conditions may cloud the picture of symptom response to medication; the more depressed patients may respond to SSRIs and the more impulsive maniclike patients may respond to anticonvulsants. In combat veterans with posttraumatic stress disorder and comorbid major depressive disorder, the depression may not respond to antidepressants as expected. Even in noncombat posttraumatic stress disorder, the magnitude of response to antidepressants for depressive symptoms or of an anxious patient to anticonvulsants is not as robust as in pure depressive or anxious syndromes.

**Anger Management**

Anger management is an essential component in most specialized treatment programs for posttraumatic stress disorder—especially in veterans’ hospitals where irritability, explosiveness, and aggression are common among patients. Anger is an adaptive emotion that signals threat or harm, which fits the generalized hypervigilance pattern of posttraumatic stress disorder. Anger is also linked with arousal that may activate aggressive behavior and counter helplessness, and it stems from feeling that the world is unfair—which may foster the desire or impulse for revenge. Finally, anger is facilitated by chronic illness and may be associated with neurochemical abnormalities. Anger management is designed to identify physical, emotional, and situational cues to anger, develop anger control plans, recognize and alter destructive self-talk, utilize time out, practice conflict resolution techniques, and use groups to discuss and evaluate high-risk anger situations. A typical anger management treatment plan includes:

- identifying details or situations that incite anger,
- rating those details or situations on an anger meter or appropriate scale,
- developing plans for controlling anger,
- role-playing the coping strategies in real-life situations,
- identifying different components in the aggression cycle (e.g., how initial arousal in the escalation phase ultimately leads to negative consequences) and analyzing the most violent incident,
- talking about anger in the family of origin (pre-trauma) because angry and aggressive responses demonstrated by trauma survivors may have been learned in childhood and adolescence,
- practicing self-monitoring of statements that incite anger responses,
- relaxation training, and
- assertiveness training.

Ideally, anger management is administered in short-term group sessions, but, in the case of veterans, the basic concepts of anger management are often repeated over time in an ongoing milieu.

**CONCLUSION**

Posttraumatic stress disorder is a relatively new diagnosis that is still evolving as a clinical concept. Although not formally considered as diagnostic criteria, anger and aggression are common features of posttraumatic stress disorder, and it is timely to consider the management of patients with these associated features. Controlled data on pharmacotherapy of the disorder are limited, but patients seem to benefit most when medications target specific symptoms or symptom clusters. Preliminary data indicate a role for anticonvulsants in the treatment of the impaired impulse control and violent outbursts that often accompany posttraumatic stress disorder. Controlled studies are needed.

**Drug names:** carbamazepine (Atretol, Tegretol, and others), divalproex (Depakote), yohimbine (Yocon and others).

**REFERENCES**

2. American Psychiatric Association. Diagnostic and Statistical Manual of
Managing Anger and Aggression in PTSD


DISCLOSURE OF OFF-LABEL USAGE

The author of this article has determined that, to the best of her knowledge, no investigational information about pharmaceutical agents has been presented herein that is outside Food and Drug Administration–approved labeling.