Trauma and Stress: Diagnosis and Treatment

This ACADEMIC HIGHLIGHTS section of The Journal of Clinical Psychiatry *presents the highlights of the teleconference "Trauma and Stress: Diagnosis and Treatment," held August 6, 2001.*

The teleconference was chaired by ***Mark H. Pollack****, M.D., Director of the Anxiety Disorders Program at Massachusetts General Hospital and Harvard Medical School, Boston, Mass. The other participants were* ***Kathleen T. Brady****, M.D., Ph.D., Institute of Psychiatry, Medical University of South Carolina, Charleston;* ***Randall D. Marshall****, M.D., Associate Professor of Clinical Psychiatry, Columbia University, College of Physicians and Surgeons, and Director of Trauma Studies and Services, New York State Office of Mental Health; and* ***Rachel Yehuda****, M.D., Mount Sinai School of Medicine, and Department of Psychiatry, Bronx VA Medical Center, New York, N.Y.*

The teleconference and these ACADEMIC HIGHLIGHTS were independently developed by the Physicians Postgraduate Press, Inc. Office of Continuing Education pursuant to an unrestricted educational grant from GlaxoSmithKline.

Continuing Medical Education

Faculty Disclosure

In the spirit of full disclosure and in compliance with all ACCME Essential Areas and Policies, the faculty for this CME activity were asked to complete a full disclosure statement.

The information received is as follows: **Dr. Pollack** *has received honoraria from Bristol-Myers and Forest, has received honoraria and is a consultant for Lilly, Parke-Davis, Pfizer, GlaxoSmithKline, Solvay, and Wyeth-Ayerst;* **Dr. Brady** *has received research grant support and honoraria from and is a consultant and member of the speakers/advisory board for Pfizer, Abbott, Bristol-Myers, Parke-Davis, Lilly, and GlaxoSmithKline;* **Dr. Marshall** *is a consultant for Wyeth-Ayerst, has received research grant support from GlaxoSmithKline, has received honoraria and is a member of the speakers/advisory board for Pfizer, GlaxoSmithKline, and Bristol-Myers;* **Dr. Yehuda** *is a consultant for Novartis, has received research grant support from GlaxoSmithKline, Janssen, Wyeth-Ayerst, and Pfizer; has received honoraria from GlaxoSmithKline, and is a member of the speakers/advisory board for Pfizer, GlaxoSmithKline, Novartis, and Bristol-Myers.*

The opinions expressed herein are those of the authors and do not necessarily reflect the views of the CME provider and publisher or the commercial supporter.

Introduction

Recognition has recently increased that posttraumatic stress disorder (PTSD) is a condition that not only affects combat veterans but is ubiquitous in the general population as well, began Mark H. Pollack, M.D. PTSD can occur following a wide variety of incidents; clinicians may encounter PTSD in a range of patients from different walks of life. The literature on PTSD contains studies of the disorder occurring after violent crime,[1,2] sexual abuse,[3] earthquake,[4] war,[5,6] medical illness such as cancer[7,8] or heart attack,[9] automobile accidents,[10] and other traumas. PTSD is found in children and adolescents,[8,11] older adults,[12] and all ages in between, for both sexes.

Not only does PTSD have a large number of precipitants, it also has a variety of potential treatments. Treatments recommended in the literature are the newer antidepressants,[13] exposure therapy,[14] and other types of management.[15,16]

This discussion of PTSD will cover some of the current issues in diagnosis, pathophysiology, and treatment.

REFERENCES

1. Pfefferbaum B, Seale TW, McDonald NB, et al. Posttraumatic stress two years after the Oklahoma City bombing in youths geographically distant from the explosion. Psychiatry 2000;63:358–370
2. Brewin CR, Andrews B, Rose S. Fear, helplessness, and horror in posttraumatic stress disorder: investigating DSM-IV criterion A2 in victims of violent crime. J Trauma Stress 2000;13:499–509
3. Wolfsdorf BA, Zlotnick C. Affect management in group therapy for women with posttraumatic stress disorder and histories of childhood sexual abuse. J Clin Psychol 2001;57:169–181
4. Wang X, Gao L, Shinfuku N, et al. Longitudinal study of earthquake-related PTSD in a randomly selected community sample in north China. Am J Psychiatry 2000;157:1260–1266
5. Vuksic-Mihaljevic Z, Mandic N, Bensic M, et al. Posttraumatic stress disorder among Croatian veterans: a causal model. Psychiatry Clin Neurosci 2000;54:625–636
6. Carson MA, Paulus LA, Lasko NB, et al. Psychophysiologic assessment of posttraumatic stress disorder in Vietnam nurse veterans who witnessed injury or death. J Consult Clin Psychol 2000;68:890–897
7. Mundy EA, Blanchard EB, Cirenza E, et al. Posttraumatic stress disorder in breast cancer patients following autologous bone marrow transplantation or conventional cancer treatments. Behav Res Ther 2000;38:1015–1027
8. Madan-Swain A, Brown RT, Foster MA, et al. Identity in adolescent survivors of childhood cancer. J Pediatr Psychol 2000; 25:105–115
9. Alonzo AA. Acute myocardial infarction and posttraumatic stress disorder: the consequences of cumulative adversity. J Cardiovasc Nurs 1999;13:33–45
10. McDermott BM, Cvitanovich A. Posttraumatic stress disorder and emotional problems in children following motor vehicle accidents: an extended case series. Aust N Z J Psychiatry 2000;34:446–452
11. Daviss WB, Mooney D, Racusin R, et al. Predicting posttraumatic stress after hospitalization for pediatric injury. J Am Acad Child Adolesc Psychiatry 2000;39:6–83
12. Averill PM, Beck JG. Posttraumatic stress disorder in older adults: a conceptual review. J Anxiety Disord 2000;14:133–156
13. Pearlstein T. Antidepressant treatment of posttraumatic stress disorder. J Clin Psychiatry 2000;61(suppl 7):40–43
14. Ballenger JC, Davidson JR, Lecrubier Y, et al. Consensus statement on posttraumatic stress disorder from the International Consensus Group on Depression and Anxiety. J Clin Psychiatry 2000;61(suppl 5):60–66
15. Anderson PL, Rothbaum BO, Hodges L. Virtual reality: using the virtual world to improve quality of life in the real world. Bull Menninger Clin 2001;65:78–91
16. Rittenhouse J. Using eye movement desensitization and reprocessing to treat complex PTSD in a biracial client. Cultur Divers Ethni Minor Psychol 2000;6:399–408

Presentation and Epidemiology of PTSD

Kathleen T. Brady, M.D., Ph.D., described the presentation and symptoms as well as the epidemiology of PTSD.

By definition, PTSD is a characteristic set of signs and symptoms that develop after a person is exposed to a traumatic event.[1] The risk factors for experiencing traumas can differ from the risks related to developing PTSD (Table 1).[2] For instance, men are at higher risk for experiencing traumatic events, yet women are at higher risk for developing PTSD.

Symptom Clusters

To fit criteria for PTSD, symptoms must last for at least 1 month after the traumatic event and must cause considerable functional impairment.[1] The symptoms fall into 3 clusters, shown in Table 2.

The reexperiencing or intrusion cluster is marked by thoughts that an individual simply cannot put out of his or her mind—intrusive thoughts or nightmares about the event—as well as physiologic or psychological reactivity when exposed to anything that reminds him or her of the event. This reexperiencing or intrusion cluster is particularly important in the differential diagnosis because there is a marked overlap between symptoms of PTSD and symptoms of other psychiatric disorders. The symptoms in the reexperiencing or intrusion cluster are unique in that they are tied closely to the traumatic event itself. In that sense, these symptoms do not overlap with other psychiatric disorders to the extent that some of the other symptoms do.

The avoidant cluster of PTSD symptoms causes a narrowing of the person's behavioral repertoire. People with this cluster of symptoms avoid experiences with people, places, objects, or thoughts that might remind them of the event. These people have a restricted range of affect, and they lose interest in activities that were previously enjoyable to them.

Finally, the arousal symptom cluster is marked by sleep disturbance, startling easily, poor concentration, and a sense of hypervigilance—always needing to protect oneself and be watchful of one's safety.

In order to be part of the PTSD syndrome, all symptoms should have appeared only after the traumatic event.

Prevalence

PTSD is more prevalent than previously believed, Dr. Brady stated. Traditionally, PTSD has been considered to be a disorder related primarily to combat exposure. However, recent epidemiologic surveys have helped to point out that civilian traumas can and often do cause PTSD. According to the National Comorbidity Study,[3] there is a 7.8% lifetime prevalence of PTSD in the United States. PTSD will affect about 5% of men and about 10% of women in their lifetime, so it is twice as common in women as men. Green[4] reported that of people who experience 1 or more traumas, about 25% will develop PTSD, although this percentage changes depending on the severity of the event.

Risk Factors

Some types of trauma are much more likely to cause PTSD than others, such as having been wounded in combat or raped or having witnessed the sudden and unexpected death of a loved one. In the National Comorbidity Study,[3] rape was the trauma with the highest conditional probability for being associated with PTSD in both men and women (Table 3). Combat also has high conditional probability for association with PTSD.[2]

Besides being female, other preexisting risk factors for developing PTSD may include inner city residence, presence of a current psychiatric disorder, history of behavioral problems before the age of 15 years, history of childhood abuse, and history of psychiatric illness in family members.[2] A history of a previous trauma might also be associated with vulnerability to the development of PTSD after a traumatic event.

The type and the severity of the trauma are the most important determinants of developing the disorder. If the trauma is severe enough, anyone can develop PTSD whether he or she has a preexisting vulnerability or not. It is not a sign of weakness in an individual to develop PTSD, Dr. Brady noted.

Differential Diagnosis

In terms of differential diagnosis, comorbidity with other psychiatric disorders is extremely common in PTSD. Not only are there multiple symptoms of PTSD that overlap with those of different psychiatric disorders, there is often true comorbidity with other psychiatric disorders. In fact, it is more common for individuals with PTSD to have comorbid psychiatric disorders than it is to have PTSD alone. In the National Comorbidity Study,[3] 44% of women and 59% of men with PTSD met criteria for 3 or more other psychiatric diagnoses.

Depression is the most common comorbid disorder—48% of men and 49% of women who have PTSD have a lifetime major depressive disorder.[3] Other prominent disorders in individuals with PTSD include substance use disorders—both alcohol and drug abuse—as well as prescription drug abuse, and other anxiety disorders such as social phobia and panic. Somatic symptoms are also frequently comorbid with PTSD.[5] Because individuals with PTSD frequently have irritable bowel syndrome, chronic pelvic pain, fibromyalgia, or chronic headaches, they will often first present in a primary care setting.

Because symptom overlap with other disorders is so high and because PTSD can have such heterogeneous presentation, PTSD can be easily misdiagnosed or overlooked unless a clinician asks specifically about trauma and takes a careful trauma history.

Discussion

Dr. Pollack asked whether the acute stress-related symptoms a patient has immediately after a trauma differ from those that will persist and become part of PTSD. Dr. Brady replied that most

Table 1. Assessment of Risk for Exposure to Trauma and for Posttraumatic Stress Disorder (PTSD) by Sex[a]

Sex	Exposure to Trauma	PTSD
Men	At higher risk than women More physical assault and other life-threatening situations Risk factors: history of early conduct problems, neuroticism/extroversion, psychiatric disorders, parental substance abuse, parental divorce	More frequent if history of psychiatric disorder, parental psychiatric disorder At higher risk if married at the time of the trauma, lower education Leading traumas: combat, childhood neglect, physical abuse, sudden unexpected death of a loved one
Women	At lower risk than men More sexual assaults and childhood parental neglect Risk factors: history of affective, anxiety, or substance abuse; parental mental illness and substance abuse; parental aggression Lower education, younger age, being married at the time of the trauma, having been married previously, urban residence, and lower income are significant predictors	At higher risk than men More frequent if history of psychiatric disorder, parental psychiatric disorder, parental aggression Younger age and prior trauma exposure significant predictors Leading traumas: sexual assault, rape, sudden unexpected death of a loved one

[a]Adapted from Hidalgo and Davidson,[2] with permission.

Table 2. DSM-IV Criteria for the Diagnosis of Posttraumatic Stress Disorder (PTSD)[a]

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 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 1 (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. Physiologic 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 3 (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. Feelings of detachment or estrangement from others.
   6. Restricted range of affect (eg, unable to have loving feelings).
   7. Sense of foreshortened future (eg, 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 2 (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.

[a]From the American Psychiatric Association,[1] with permission.

individuals who experience a traumatic event initially have some of the symptoms of PTSD, even if they do not develop the disorder. After experiencing extreme trauma, having intrusive thoughts about that event for a few days afterward is a normal and common human experience. If these symptoms are short-lived and do not cause functional impairment for a substantial amount of time, then they are nonpathologic.

Dr. Marshall commented that the persistence of the symptomatology correlates with the severity of the trauma. Rothbaum and colleagues[6] found that 94% of women met symptomatic criteria for PTSD 2 weeks after being raped, whereas 3 months later only 47% continued to meet PTSD criteria. But, compared with rape, car accidents have less probability of resultant PTSD. Blanchard et al.[7] found that 36% of 132 victims of motor vehicle accidents met PTSD criteria 1 to 4 months after the accident, and 6 months later, half had remitted, at least in part.

Table 3. Lifetime Prevalence of Trauma Exposure and the Risk of PTSD by Sex and Type of Trauma[a]

	Lifetime Prevalence (%)		PTSD Risk (%)	
Trauma	Male	Female	Male	Female
Life-threatening accident	25.0[b]	13.8	6.3	8.8
Natural disaster	18.9[b]	15.2	3.7	5.4
Threatened with weapon	19.0[b]	6.8	1.9[b]	32.6
Physical attack	11.1[b]	6.9	1.8[b]	21.3
Rape	0.7[b]	9.2	65.0	45.9

[a]Data from Kessler et al.[3]
[b]Sex difference significant at the .05 level, 2-sided test.

Dr. Yehuda added that the fundamental problem for clinicians is often in understanding that the patient's underlying problem is not being able to control the memory and the distress associated with that memory. The normal trajectory after exposure to trauma is that gradually—over weeks or months—the emotional impact of the event fades. The event takes its place in the person's tapestry of other life experiences, and even though there is a memory of that event, which might cause emotional distress, the person will have control over both when to have the memory and how to modulate the distress it causes. However, people with PTSD feel unable to regulate the occurrence of a memory or their emotional response to it, and this inability restricts their lives because they then try to avoid being reminded.

Dr. Marshall pointed out that a useful analogy is the difference between normal mourning and pathologic mourning. After losing a loved one, it would be normal to be overwhelmed and have impaired function for a while, but, over time, those feelings fade. However, the grief symptoms become pathologic when daily functioning is impaired for a considerable amount of time.

Dr. Pollack wondered if rape accounts for the increased rate of PTSD in women. Dr. Brady responded that women are indeed more likely to experience rape, a trauma that is highly likely to cause PTSD. But she pointed out that another risk factor for PTSD is having a history of depression or current depression at the time of the trauma, and depression is twice as common in women as in men. Those factors combined probably account to some extent for the differential between the sexes.

REFERENCES

1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. Washington, DC: American Psychiatric Association; 1994
2. Hidalgo RB, Davidson JRT. Posttraumatic stress disorder: epidemiology and health-related considerations. J Clin Psychiatry 2000;61(suppl 7):5–13
3. Kessler RC, Sonnega A, Bromet E, et al. Posttraumatic stress disorder in the National Comorbidity Survey. Arch Gen Psychiatry 1995;52:1048–1060
4. Green BL. Psychological research in traumatic stress: an update. J Trauma Stress 1994;7:341–361
5. Heffernan K, Cloitre M. A comparison of posttraumatic stress disorder with and without borderline personality disorder among women with a history of childhood sexual abuse: etiological and clinical characteristics. J Nerv Ment Dis 2000;188:589–595
6. Rothbaum BO, Foa EB, Murdock T, et al. A prospective examination of post-traumatic stress disorder in rape victims. J Trauma Stress 1992;5:455–475
7. Blanchard EB, Hickling EJ, Barton KA, et al. One-year prospective follow-up of motor vehicle accident victims. Behav Res Ther 1996;34:775–786

Pathophysiology of PTSD

Rachel Yehuda, M.D., stated that the biology of depression appears to be distinct from that of PTSD, although the risk factor for increased depression in women may also be in operation in the increased risk for PTSD in women. Increased activation of the hypothalamic pituitary adrenal system has been identified as a major pathophysiologic component of depression. Major depression is associated with increased cortisol levels, while PTSD appears to be associated with normal or even low cortisol levels.[1] Dr. Yehuda and colleagues[2] have recently examined the interplay between PTSD and depression in trauma survivors with and without PTSD or depression. When people have both PTSD and depression following exposure to trauma, the biological effects can sometimes appear to cancel themselves out. That is, the individual may show neither a hypersuppression nor a nonsuppression of cortisol. These competing biological effects may result from different risk factors and competing demands on the system.

Biological Response to Stress

The response to stress is one in which many biological systems are activated so that one can cope.[3] The startle response helps alert the body to the need for a biological response. The sympathetic nervous system releases epinephrine, which is important for the formation of memories but also helps increase blood pressure and increase heart rate. The parasympathetic nervous system also is activated so that normative biological activities, such as digestion, can cease. The activation of the hypothalamic pituitary adrenal system, which releases cortisol, occurs to help contain stress-activated systems once the person has made a response to the stress and the trauma has passed.

Vulnerability to PTSD

According to Dr. Yehuda, one working hypothesis of PTSD is that biological vulnerability factors are related to early adverse experiences. Persons who were exposed to early trauma—even if they have recovered from those experiences—may have a unique biological profile before a traumatic event, and that biological profile makes it more difficult for them to recover. Specifically, it makes it more difficult for them to reduce their physiologic distress as a function of activation of their sympathetic nervous system.

People who are more likely to develop long-term PTSD have been found to show lower cortisol levels and increased heart rate in the immediate aftermath of the trauma.[4] This could theoretically increase sympathetic ner-

vous system response, which might lead to the pairing of the memory with the distress.

Discussion

Dr. Marshall commented that determining gender differences is complicated because studies need to control for the severity of the trauma and the intensity of the response to the trauma. Dr. Brady asked whether human laboratory studies in which the intensity of the stressor can be controlled yield useful data about gender differences. Dr. Yehuda pointed out that a complicating issue is that cortisol response to stress is affected by where a woman is in her menstrual cycle and where she is in her reproductive life cycle because estrogen is a major mediator of the cortisol response to stress.[5] Given that women are twice as likely to develop PTSD as men, gender variables deserve further study. One research question relates to the impact on subsequent traumatizations when girls are raped before they begin their menstrual cycles. Prepubescent girls' bodies have to mount a cortisol response in the absence of estrogen.

Dr. Pollack related that Coplan and others[6] demonstrated that early stress in animal models has permanent effects on the hypothalamic pituitary axis and stress response. He asked Dr. Yehuda what types of early stressors girls are prone to. Dr. Yehuda responded that young girls might be affected by sexual abuse, physical abuse, assault in general, a general feeling of being more vulnerable to attack than boys, or just being threatened. Witnessing domestic violence might influence boys and girls differently—a question which is being actively investigated. Dr. Marshall agreed that witnessing domestic violence early on might affect boys and girls differently, depending on who the perpetrator is and who the victim is. This experience could affect the development of role identification and sexual identity for boys and girls differently. Often the children are assaulted as well, but even if they are not assaulted and grow up seeing their mother beaten or threatened, these children grow up with the hypervigilance that is seen in children who grow up in war zones or in urban settings where the safety threat in the home or immediate environment is real.

Table 1. Current and Lifetime PTSD Among Offspring of Holocaust Survivors and Comparison Subjects[a]

	Total Sample (N = 144)			
	Comparison Subjects (N = 44)		Total Offspring Group (N = 100)	
Disorder	N	%	N	%
PTSD				
Current	1	2	15	15
Lifetime	4	9	31	31

[a]Data from Yehuda et al.[8]

Dr. Pollack asked Dr. Yehuda whether children of Holocaust survivors have similarities with children who witness their parents' domestic violence. Dr. Yehuda responded that the children of Holocaust survivors can be affected in numerous ways. Parents who survived the Holocaust might have PTSD or depression or both,[7] and living with a parent with these conditions can have a lasting impact on the child. Adult children of Holocaust survivors are 3 times more likely to develop PTSD in their lifetime compared with demographically matched peers (Table 1),[8] which suggests a certain vulnerability in children of people with PTSD. Whether the vulnerability is transmitted environmentally or genetically is unknown. Another problem these children have is that their experiences may be minimized in relation to the Holocaust, although this may not necessarily be done for malevolent reasons on the part of the parent. Adult children of Holocaust survivors complain a bit more about emotional—but not physical or sexual—abuse than their peers. Emotional abuse was recently found to be related to lower cortisol levels,[9] so the risk factor for PTSD may, in fact, be mediated by early experiences of emotional abuse. People who experience emotional abuse without other forms of abuse may not realize they have experienced any abuse, because it is subtle to be given a message that one's experiences are inferior to those of others or that one is somehow incompetent.

REFERENCES

1. Boyer P. Do anxiety and depression have a common pathophysiological mechanism? Acta Psychiatr Scand 2000;102(suppl 406): 24–29
2. Yehuda R, Halligan SL, Bierer LM. Cortisol levels in adult offspring of Holocaust survivors: relation to PTSD symptom severity in the parent and child. Psychoneuroendocrinology 2001. In press
3. Yehuda R. Biology of posttraumatic stress disorder. J Clin Psychiatry 2000;61 (suppl 7):14–21
4. Yehuda R, McFarlane AC, Shalev AY. Predicting the development of posttraumatic stress disorder from the acute response to a traumatic event. Biol Psychiatry 1998;44:1305–1313
5. Altemus M, Redwine L, Leong YM, et al. Reduced sensitivity to glucocorticoid feedback and reduced receptor mRNA expression in the luteal phase of the menstrual cycle. Neuropsychopharmacology 1997; 17:100–107
6. Coplan JD, Andrews MW, Rosenblum LA, et al. Persistent elevations of cerebrospinal fluid concentrations of corticotropin-releasing factor in adult nonhuman primates exposed to early-life stressors: implications for the pathophysiology of mood and anxiety disorders. Proc Natl Acad Sci USA 1996;93:1619–1623
7. Yehuda R, Kahana B, Southwick SM, et al. Depressive features in Holocaust survivors with post-traumatic stress disorder. J Trauma Stress 1994;7:699–704
8. Yehuda R, Schmeidler J, Wainberg M, et al. Vulnerability to posttraumatic stress disorder in adult offspring of Holocaust survivors. Am J Psychiatry 1998;155: 1163–1171
9. Yehuda R, Halligan S, Grossman R. Childhood trauma and risk for PTSD: relationship to intergenerational effects of trauma, parental PTSD and cortisol excretion. Stress Dev 2001;13:731–751

Treatment and Prevention of PTSD

Randall D. Marshall, M.D., stated that the best established treatment and prevention approach for trauma survivors and people with PTSD has been psychotherapy, of which there are several well validated types. Recent years have also brought about the approval of medication for treatment of PTSD.

Research on Psychosocial Treatment

Perhaps the best studied type of cognitive-behavioral treatment for PTSD is exposure therapy, which is based in the belief from the 1980s (when the PTSD diagnosis first was

introduced) that PTSD is a phobic disorder.[1] Exposure therapy is an amalgam of psychoeducation, support, and provision of hope—encouraging patients to face the traumatic memory and process it rather than continue to try to flee it.

Another type of treatment is stress inoculation training, which is an anxiety management program.[1] This treatment stems from the idea that people with PTSD have signs of generalized anxiety disorder.

Cognitive therapy is another variation of psychosocial treatment for PTSD sufferers, and its focus is on correcting the erroneous cognitions that PTSD patients have. These people tend to endorse negative thoughts about the world and negative thoughts about themselves, including the belief that they are incompetent or to blame for what happened to them, leading them to overgeneralize from their traumatic experience to more neutral situations.[1]

A relatively new treatment that seems effective is eye movement and desensitization reprocessing (EMDR). This treatment consists of a form of exposure therapy (asking the patient to visualize images related to the trauma) while the patient's eyes follow the rapid side-to-side movement of the therapist's finger.[1] A form of cognitive therapy is also incorporated, in which the patient is encouraged to replace negative thoughts with positive ones.

The problem with the research in psychotherapy and PTSD is that it is time limited to 10 or 15 sessions. Regardless of the treatment approach used, a substantial proportion of individuals are still symptomatic after 10 to 15 sessions, suggesting that additional treatment is needed for the majority of people with chronic PTSD.

Research on Medication Treatment

Dr. Marshall commented that it is difficult to compare earlier studies of medication for PTSD with the newer trials because the older United States trials were conducted in war veterans, while newer trials have subjects with other trauma circumstances. There has been debate over whether chronic PTSD due to combat trauma is less treatable than PTSD induced by other traumas. However, Dr. Marshall believes the difficulty of treating combat-related PTSD is associated more with the selection criteria for the clinical trials than with the difficulty of treating combat trauma with medicine.

Medication is the newest treatment to be validated for PTSD and is a major advance for the field. The selective serotonin reuptake inhibitor (SSRI) sertraline became the first medication approved for PTSD by the U.S. Food and Drug Administration. In several double-blind, placebo-controlled, multicenter trials, as well as in open-label trials, SSRIs have shown efficacy for PTSD.

Dr. Brady was involved in a placebo-controlled study of sertraline for PTSD. In this study, Baker et al.[2] studied almost 200 outpatients with PTSD for 12 weeks. They found not only significantly greater decreases on scales measuring PTSD symptoms in the sertraline-treated group than in the placebo-treated group but also improved quality of life with sertraline.

The original SSRI, fluoxetine, was compared with placebo for PTSD in a recent trial[3] in Europe and Africa. Fluoxetine was found to be well tolerated and effective for both combat veterans and civilians. The superiority of fluoxetine (N = 226) over placebo (N = 75) was significant from week 6 through the end of the study (week 12). A prior placebo-controlled trial[4] of fluoxetine in 53 civilians with PTSD had found that fluoxetine was superior to placebo by week 12 as measured by several rating scales, but subjects with a more chronic course of PTSD had a lower response rate to the drug and a higher response rate to placebo. A 5-week study[5] of fluoxetine in both combat veterans and civilians (64 men and women) had shown that fluoxetine was superior to placebo in reducing symptom scores, especially the avoidance and arousal clusters; however, there was a more robust response rate in the civilians than in the war veterans.

In a recent open trial,[6] Dr. Marshall and colleagues found paroxetine efficacious in men and women with non-combat-related, chronic PTSD. Eleven (65%) of 17 patients were rated as much or very much improved, and the mean reduction in PTSD symptom scores was 48%. Dr. Marshall and colleagues also conducted a 12-week, placebo-controlled, fixed-dose study[7] of paroxetine (either 20 mg/day or 40 mg/day) in more than 500 subjects. People taking either dose of paroxetine experienced significant decreases in the 3 symptom clusters of PTSD. In the 20-mg/day paroxetine group, 63% of subjects were defined as responders, and in the 40-mg/day paroxetine group, 57% were responders, compared with 37% of those taking placebo. Paroxetine was found as effective in men as in women. In a recent 12-week, placebo-controlled, flexible-dose (20 mg/day to 50 mg/day) study of paroxetine in 300 subjects with PTSD, Tucker et al.[8] found significantly greater improvement in those taking paroxetine than placebo on the Clinician-Administered PTSD Scale total scores. In both of these placebo-controlled studies, functional improvement was greater with paroxetine than placebo in work, social life, and family life domains, and the adverse events were consistent with the known safety profile of paroxetine.

Older studies with tricyclic antidepressants (TCAs) and monoamine oxidase inhibitors (MAOIs) also suggested efficacy of medication treatment for combat veterans and civilians. Davidson and colleagues[9] studied amitriptyline (a TCA) in an 8-week placebo-controlled trial of 46 veterans of World War II or the Vietnam conflict. The percentage of patients rated as much or very much improved was 50% with amitriptyline and 17% with placebo. Kosten and colleagues[10] examined imipramine, another TCA, and phenelzine, an MAOI, in 60 Vietnam veterans. On the Impact of Event Scale (IES), a 25% decrease in scores was found in subjects taking imipramine, a 45% reduction among those taking phenelzine, and a 5% decrease in those taking placebo.

Anticonvulsants and mood stabilizers such as carbamazepine, sodium valproate, and lithium have been studied in small trials or case series and may be effective for anger and explosive

behavior in PTSD.[11,12] Lamotrigine was examined in a 12-week placebo-controlled trial[13] with 15 subjects. The response rate for lamotrigine was 50% while the response rate for placebo was 25%. There was superior efficacy over placebo for the intrusion and avoidance symptom clusters. However, a slow dose escalation is required because of the potential for a serious skin rash.

Benzodiazepines are still frequently prescribed in PTSD. However, trials with alprazolam and clonazepam have not shown any effect on PTSD symptoms.[14,15] As Dr. Brady noted, the risk of substance abuse is high in PTSD—around 50%—and benzodiazepines are potentially addictive. Withdrawal of alprazolam resulted in homicidal ideation in 6 of 8 combat veterans.[16] Therefore, clinicians need to be careful if they decide to prescribe benzodiazepines for PTSD.

Research on Combination Treatment

The great gap in the PTSD literature is for data about combining medication with psychotherapy or even comparing psychotherapy with medications. Fundamental clinical issues have not been answered because this is a relatively new field. Data have also not yet been gathered on sequencing treatments, but many clinicians think that treating patients with medication may help the psychotherapy treatment process succeed.

Discussion

Responsivity of Veterans

Dr. Pollack commented that veterans' responsivity to treatment has been an ongoing issue in the field. In fact, in some trials, the entry of veterans has been discouraged or frankly prohibited because of the perception that they do not respond as well. There is also the issue of chronicity; veterans of the Vietnam, Korean, or World War conflicts with PTSD have been ill now for many decades and would therefore be less responsive to treatment. Dr. Marshall added that using veterans in the VA system may mean using subjects who are by definition treatment refractory; that is, the veterans who responded to treatment are eliminated from the VA system. Those who did not respond are the ones left in the system.

Dr. Yehuda asserted that in her experience of more than a decade in the VA system, veterans do want to get better. They are somewhat fearful about compensation issues, but once they are assured that the purpose of a trial is not tied into their compensation, they may be suitable research candidates. If they are treatment-refractory, then they should be excluded from a pharmaceutical trial. Also, just because the war was 20, 30, or 40 years ago does not mean the person has had PTSD that long. The natural history of PTSD is one of peaks and valleys, so there are many people who come to the VA after experiencing recent life stressors that have activated the PTSD, and they may not have sought treatment earlier. Eliminating combat veterans from pharmaceutical trials is not useful. They should be given an opportunity to be helped by a medication. Combat veterans have much to teach researchers and clinicians, because the PTSD seen in VAs is the same PTSD that is treated in non-VA settings.

Determining a Treatment Strategy

Dr. Marshall noted that a recent expert consensus[17] suggested that the first-line treatment options for PTSD are either trauma-focused psychotherapy or medication with supportive psychotherapy. Dr. Marshall added that other treatment guidelines[18] recommend that for patients with severe comorbid illness, such as debilitating panic attacks or severe major depression, medication and trauma-focused psychotherapy might be coadministered from the start. He noted that there has been debate about whether PTSD can be treated in primary care or if it is, in fact, too complicated a disorder to be treated in the brief amount of time that most primary care physicians have to spend with a patient.

Dr. Pollack mentioned that patients may feel so overpowered by their symptoms, at least initially, that the idea of exploring them in psychotherapy can be overwhelming. It may be easiest for patients to start with medication and thereby experience some reduction in their anxiety or depression, which may make them more amenable to examining issues in therapy. However, people sometimes are reluctant to take medication because of fears about side effects or dependence.

Dr. Marshall strongly recommended giving patients input into their treatment plan. Clinicians should educate patients about the different psychotherapies and medications available and should discuss with the patient his or her fears or fantasies about each of these treatments. Dr. Marshall added that if patients wonder why they should take a medication when the problem is psychological, then the clinician can describe findings about the biology of PTSD and explain that the distress response is a powerful physiologic reaction that at this point has gotten a life of its own.

Dr. Yehuda pointed out that some people shun medication because they fear losing control; the trauma made them feel out of control, and they are afraid that they will not be in control of their own emotions if they take a pill. Dr. Marshall commented that he sometimes sees the same fear when he suggests psychotherapy treatment—the fear that their emotional reaction will be out of control.

Dr. Yehuda issued a reminder that not all people with PTSD have the same symptoms, and she emphasized that clinicians must know the chief complaint before presenting the choices of psychotherapy treatment and medication treatment. The overall psychoeducation provided by the clinician must give the patient a cohesive explanation for how and why a particular treatment will be used for the chief complaint.

Treatment Nonadherence

Mentioning the fact that compliance with treatment among patients in most medical specialties is poor, Dr. Pollack emphasized that preparing patients for treatment—both medication and psychotherapy—is critical. Dr. Marshall acknowledged that with psychotherapy, clinicians can assume that few patients will adhere to the exposure homework at first because of its difficulty. Asking

the patient about noncompliance in the early stages of treatment can be simply a chance to again discuss the rationale for treatment, with the expectation that the patient has not yet been compliant. Dr. Yehuda agreed that avoidance is one of the key reasons that patients do not do homework for psychosocial therapies and may be the same reason that they do not take their medication. The pill reminds them of the PTSD and of the fact that they were traumatized. Dr. Yehuda stated that clinicians must be very conscious of the need that trauma survivors feel to be in control. Adherence with treatment might be affected by the patient's need for control. Taking medication or doing psychosocial treatment homework cannot be about trying to please the therapist or feeling that an authority figure has asked them to do something. Both Dr. Marshall and Dr. Yehuda agreed that when a clinician finds that a patient is not taking medication, it is important to distinguish between noncompliance due to intolerable side effects, in which case a change can be made in either the dose or the type of medication, and noncompliance due to the patient thinking he or she does not participate enough in treatment decisions.

Dissociative Symptoms

Dr. Pollack noted that dissociative symptoms sometimes seem less robustly responsive to treatment and asked if this clinical perception has been backed up by research. Dr. Marshall answered that most clinical trials to date measure reduction of only the core PTSD symptoms of reexperiencing, avoidance, and arousal, but in many ways the associated problems are much more debilitating. Dr. Marshall stated that when he and coworkers conducted the open trial of paroxetine,[6] dissociative symptoms were reduced about 50% with the medication. This was not surprising because dissociation is a symptom that is triggered by severe fear or anxiety or perception of threat, so when the overall level of fear or anxiety is modulated by medication, the dissociated state of mind does not get triggered as often.

Dr. Yehuda explained that if someone has to respond to a threat in the environment, being able to disengage might be very beneficial in that time of extreme fear or stress and may actually improve the person's ability to cope with the threat. So, disconnecting from the immediate environment is not necessarily pathological or negative. Certain types of dissociation skills may even be adaptive at the time of trauma. However, dissociation from one's environment long after the event can be a negative outcome. Clinicians are in a position to educate patients about which reactions are normative and which reactions are pathologic.

Acute Treatment: Identifying Pathologic Symptoms

Dr. Pollack asked which symptoms clinicians should label as normal and which are pathologic if they are called in to see a patient soon after a major trauma. The experts agreed that no one symptom may be predictive. Dr. Yehuda stated that it is important to tell survivors in the acute aftermath of the trauma to expect their level of distress to gradually decrease, and to monitor it. If, for example, the day after a traumatic event, they think about the event every hour, then after a week they should think about it only a few times a day. The people whose acute symptoms do not gradually decline are the people at most risk to have the acute symptomatology turn into a full-blown PTSD. Dr. Brady emphasized that clinicians must ask patients at intervals during the weeks and months after the event, "Has it been getting better? Are you thinking of the event less, or does it really drive your life still?" If the distress is going to resolve in a normal fashion, that progress should be evident within a month or so, generally.

Dr. Marshall pointed out that the likelihood of developing a chronic problem is highly correlated with the intensity of someone's initial reaction. If there is an initially incapacitating level of posttraumatic symptomatology, and if there is peritraumatic dissociation during the traumatic event, that individual is at much higher risk for having PTSD 3 months later. Dr. Yehuda agreed but reiterated that social support after a traumatic event can still make a difference in whether the trauma survivor develops PTSD. The tendency of trauma survivors is to want to isolate themselves, as if by not seeing anybody or talking about the event, they can erase what has happened or make the memory of it disappear. But the more support the person receives from his or her community, family, or friends, the more helpful it is, no matter how acute the distress was. Dr. Brady noted that one study of survivors of car accidents found that those who did not develop PTSD soon after the wreck but developed delayed cases after 6 months had had poorer social support than those who never developed the disorder.[19] Talking can still be beneficial in the early stages, even though it may not be sufficient, and other treatment modes such as pharmacotherapy may have to be used as well. Dr. Marshall agreed and reported that studies have shown that 4 or 5 sessions of time-limited psychotherapy within the first month can reduce rates of PTSD by at least 50%.[20–22] This works by encouraging the survivor to allow himself or herself a genuine emotional experience by reacting to the trauma, to lean on available support systems rather than to withdraw, and to overcome avoidance.

Dr. Pollack asked whether talking about what recently happened to them might retraumatize patients and how clinicians can ask patients to talk about what happened to them in a way that is useful. Dr. Marshall explained that a therapist can approach the talking therapy in ways that help the patient to cope, rather than simply making the patient relive the trauma. He agreed with Dr. Pollack that recent controversy has surrounded the debriefing model, in which immediately after the trauma, the individual is encouraged to talk through, in an intensive fashion, what has just happened. Studies have suggested that it can actually be harmful to rush people into that situation.[23–27] There are reports of individuals in exposure therapy getting worse, relapsing to substance abuse, and even becoming acutely suicidal. Dr. Marshall described a patient he recently saw who complete-

ly decompensated after a single session of EMDR because she was not prepared properly. Dr. Marshall stressed that it is important to set up the talk therapy in a supportive and safe context for the patient. In the treatment strategies that Dr. Marshall and colleagues are studying, based on Foa's work,[28,29] there are several sessions that focus only on education, forming a positive alliance with the patient, destigmatizing the disorder, and then essentially persuading the individual that psychotherapy is a good idea so that there is no sense of coercion. The therapist must convince the patient that it would be better to face this trauma and process it emotionally than to avoid it. A coerced feeling within the patient can be damaging.

Dr. Brady suggested that clinicians follow the lead of the patient in terms of how far they are comfortable going at any point in time. There may be times during exposure therapy when the therapist should take a break and use a session or two to do some different work if the individual seems to be handling the therapy poorly. Dr. Yehuda commented that clinicians may be unsure whether to follow the lead of the patient if the patient says, "I don't want to talk about it," because the therapist knows the patient should talk about it for his or her own good. Dr. Marshall responded that this point is where the therapist's artfulness comes in. Clinicians must explain to patients why they ultimately need to talk about the trauma. A patient who comes to a therapist in the aftermath of a traumatic event is there because, at some level, they do want to talk about it. If a patient tells the clinician that he or she does not want to talk about it, that is not necessarily a statement that they will not talk about it.

Dr. Marshall continued that patients with PTSD are confused about the difference between the memory of the trauma and the actual trauma. Through treatment, the patient can come to differentiate the memory from the actual experience. Then, the patient will not live in fear of having the memory as if that is the same as being retraumatized. Dr. Yehuda stated that patients with PTSD live in fear of the distress that they experience when they have the memory because they do not think that they can separate the emotional impact from the memory. In time, most people can remember terrible things that happened and remember how they felt without feeling how they felt. But patients with PTSD cannot talk about how bad they felt during the event without actually feeling how bad they felt every time they have that memory. If the clinician explains to the patient that he or she is going to help the patient have the memory without feeling the emotional distress, then the patient will want to work with the clinician, because that is exactly what they want.

Dr. Marshall commented that, initially, patients' reactions can be incredibly powerful because they have never grappled with the experience before. He described seeing patients in the first session tremble or throw up in the office. But, by the second or third or fourth session, patients learn from experience that each time they tell the memory, it gets easier. For this reason, it is important to educate and prepare patients for what they will experience early in treatment. They need to realize that therapy will be tough in the beginning and then get easier fairly quickly.

Dr. Yehuda added that, when clinicians prepare patients for therapy, it is also important to tell them that shame is a feeling that many people who are traumatized have, but there is no reason to be embarrassed or shameful when talking to the therapist. Most people who are traumatized feel shame that they were not powerful enough to be able to make an attacker stop or that they did not have enough wits to get themselves out of the situation. With the therapist, they might feel embarrassment at describing themselves as being so vulnerable and helpless. Dr. Marshall asserted his belief that medication can often make talk therapy more comfortable and can be the difference between the patient feeling able to confront the traumatic experience and process it in the course of therapy or feeling paralyzed in the face of that possibility.

Managing PTSD and Comorbid Substance Abuse

Dr. Pollack asked about managing patients with comorbid substance abuse and PTSD. Dr. Brady replied that 30% to 60% of patients who are treated for substance use disorders may have lifetime PTSD.[30] For a long time, the mantra in addiction treatment was to treat the addiction first, then any other psychiatric disorder. Several months of abstinence were deemed necessary before comorbid psychiatric disorders could even be assessed properly. But thoughts have changed in the last 5 to 10 years. In individuals with addictions, PTSD can be a driver of substance abuse relapse. When working with substance-using patients, clinicians should be concerned about relapse potential. If people cannot sleep or have intrusive thoughts about the traumatic event, and they want to do something about their pain, what they know how to do best is use drugs or alcohol. Clinicians must be aggressive in the treatment of PTSD symptoms early in the course of substance abuse recovery, although generally embarking on exposure therapy in the first week or two is avoided because that may be stressful for people in early recovery. But teaching coping skills can be started early. A safety manual developed by Najavits and colleagues[31] for treating the addicted individual with PTSD has been useful.

Medication has also been useful in patients with PTSD and comorbid substance disorder, although the risk-benefit ratio must be weighed carefully because these individuals are more likely to abuse anything they are given and more likely to inadvertently or even intentionally overdose. But the SSRIs are a safer group of drugs than the older antidepressants. SSRIs may in fact be helpful in decreasing alcohol consumption.[32]

Dr. Marshall inquired about using stress inoculation training as a substitute for exposure therapy when promoting relapse is a concern. Dr. Brady explained that the techniques used in stress inoculation are similar to those Lisa Najavits integrated into her manual. These are cognitive behavioral

strategies that teach an individual to manage anxiety and affect in ways other than drinking or using drugs.

Managing Trauma Victims With Children

Dr. Pollack suggested that when clinicians work with trauma victims, they ought to suggest ways for the victims to talk about their traumatic experience with their children. Dr. Yehuda agreed that one of the main motivations for trauma survivors to seek treatment is because they recognize that there is an adverse outcome on their children. There should be places for children of trauma survivors to receive treatment. Trauma affects not only the person exposed but all the people that that person interacts with. It changes social interactions and the lives of those who are dependent on that trauma survivor. Asking about a trauma survivor's children should be part of routine clinical care. Dr. Marshall concurred that one of the fundamental clinical issues in PTSD is the sense of safety in the world, and the safety of a child's world is dependent on the existence of the parents and a secure relationship with the parents. Seeing a parent threatened can have profound, disruptive effects on the child's experience of the world and his or her feeling of having a safe place in it.

REFERENCES

1. Foa EB. Psychosocial treatment of posttraumatic stress disorder. J Clin Psychiatry 2000; 61(suppl 5):43–48
2. Baker D, Brady K, Goldstein S, et al. Double-blind flexible dose multicenter study of sertraline and placebo in outpatients with post-traumatic stress disorder [abstract]. Eur Neuropsychopharmacol 1998;8(suppl 2):S261
3. Martenyi F, Brown E, Zhang H, et al. Fluoxetine versus placebo in PTSD. In: New Research Abstracts of the 154th Annual Meeting of the American Psychiatric Association; May 9, 2001; New Orleans, La. Abstract NR687:186
4. Connor KM, Sutherland SM, Tupler LA, et al. Fluoxetine in post-traumatic stress disorder: randomised, double-blind study. Br J Psychiatry 1999;175:17–22
5. van der Kolk BA, Dreyfuss D, Michaels M, et al. Fluoxetine in posttraumatic stress disorder. J Clin Psychiatry 1994;55:517–522
6. Marshall RD, Schneier FR, Fallon BA, et al. An open trial of paroxetine in patients with noncombat-related, chronic posttraumatic stress disorder. J Clin Psychopharmacol 1998; 18:10–18
7. Marshall RD, Beebe KL, Oldham M, et al. Efficacy and safety of paroxetine treatment of chronic PTSD: a fixed dosage placebo-controlled study. Am J Psychiatry 2001. In press
8. Tucker P, Zaninelli R, Yehuda R, et al. Paroxetine in the treatment of chronic posttraumatic stress disorder: results of a placebo-controlled flexible-dosage trial. J Clin Psychiatry 2001; 62:860–868
9. Davidson J, Kudler H, Smith R, et al. Treatment of posttraumatic stress disorder with amitriptyline and placebo. Arch Gen Psychiatry 1990;47:259–266
10. Kosten TR, Frank JB, Dan E, et al. Pharmacotherapy for posttraumatic stress disorder using phenelzine or imipramine. J Nerv Ment Dis 1991;179:366–370
11. Davidson JRT. Pharmacotherapy of posttraumatic stress disorder: treatment options, long-term follow-up, and predictors of outcome. J Clin Psychiatry 2000;61(suppl 5):52–56
12. Pearlstein T. Antidepressant treatment of posttraumatic stress disorder. J Clin Psychiatry 2000;61(suppl 7):40–43
13. Hertzberg MA, Butterfield MI, Feldman ME, et al. A preliminary study of lamotrigine for the treatment of posttraumatic stress disorder. Biol Psychiatry 1999;45:1226–1229
14. Gelpin E, Bonne O, Peri T, et al. Treatment of recent trauma survivors with benzodiazepines: a prospective study. J Clin Psychiatry 1996; 57:390–394
15. Braun P, Greenberg D, Dasberg H, et al. Core symptoms of posttraumatic stress disorder unimproved by alprazolam treatment. J Clin Psychiatry 1990;51:236–238
16. Risse SC, Whitters A, Burke J, et al. Severe withdrawal symptoms after discontinuation of alprazolam in eight patients with combat-induced posttraumatic stress disorder. J Clin Psychiatry 1990;51:206–209
17. Foa EB, Keane TM, Friedman MJ, eds. Effective Treatments for PTSD: Practice Guidelines from the International Society for Traumatic Stress Studies. New York, NY: Guilford Press; 2000
18. Foa EB, Davidson JRT, Frances A, eds. The Expert Consensus Guideline Series: Treatment of Posttraumatic Stress Disorder. J Clin Psychiatry 1999;60(suppl 16):6–76
19. Buckley TC, Blanchard EB, Hickling EJ. A prospective examination of delayed onset PTSD secondary to motor vehicle accidents. J Abnorm Psychol 1996;105:617–625
20. Foa EB, Hearst-Ikeda D, Perry KJ. Evaluation of a brief cognitive-behavioral program for the prevention of chronic PTSD in recent assault victims. J Consult Clin Psychol 1995;63: 948–955
21. Bryant RA, Harvey AG, Dang ST, et al. Treatment of acute stress disorder: a comparison of cognitive-behavioral therapy and supportive counseling. J Consult Clin Psychol 1998;66: 862–866
22. Bryant RA, Sackville T, Dang ST, et al. Treating acute stress disorder: an evaluation of cognitive behavior therapy and supportive counseling techniques. Am J Psychiatry 1999;156:1780–1786
23. Mayou RA, Ehlers A, Hobbs M. Psychological debriefing for road traffic accident victims: three-year follow-up of a randomised controlled trial. Br J Psychiatry 2000;176: 589–593
24. Pitman RK, Altman B, Greenwald E, et al. Psychiatric complications during flooding therapy for posttraumatic stress disorder. J Clin Psychiatry 1991;52:17–20
25. Scott MJ, Stradling SG. Client compliance with exposure treatments for posttraumatic stress disorder. J Trauma Stress 1997;10:523–526
26. McFarlane AC. The treatment of posttraumatic stress disorder. Br J Med Psychol 1989;62:81–90
27. Tarrier N, Pilgrim H, Sommerfield C, et al. A randomized trial of cognitive therapy and imaginal exposure in the treatment of chronic posttraumatic stress disorder. J Consult Clin Psychol 1999;67:13–18
28. Foa EB, Rothbaum BO, Riggs DS, et al. Treatment of posttraumatic stress disorder in rape victims: a comparison between cognitive-behavioral procedures and counseling. J Consult Clin Psychol 1991;59:715–723
29. Foa EB, Dancu CV, Hembree EA, et al. A comparison of exposure therapy, stress inoculation training, and their combination for reducing posttraumatic stress disorder in female assault victims. J Consult Clin Psychol 1999;67:194–200
30. Brady KT, Killeen TK, Brewerton T, et al. Comorbidity of psychiatric disorders and posttraumatic stress disorder. J Clin Psychiatry 2000;61(suppl 7):22–32
31. Najavits LM, Weiss RD, Liese BS. Group cognitive-behavioral therapy for women with PTSD and substance use disorder. J Subst Abuse Treat 1996;13:13–22
32. Brady KT, Sonne SC, Roberts JM. Sertraline treatment of comorbid posttraumatic stress disorder and alcohol dependence. J Clin Psychiatry 1995;56:502–505

Drug names: alprazolam (Xanax and others), amitriptyline (Elavil and others), carbamazepine (Carbatrol, Tegretol), clonazepam (Klonopin and others), fluoxetine (Prozac and others), lamotrigine (Lamictal), paroxetine (Paxil), phenelzine (Nardil), sertraline (Zoloft).

Disclosure of off-label usage:
Dr. Pollack has determined that, to the best of his knowledge, the following drugs have not been approved by the U.S. Food and Drug Administration for the treatment of posttraumatic stress disorder: alprazolam, amitriptyline, carbamazepine, clonazepam, fluoxetine, imipramine, lamotrigine, lithium, paroxetine, phenelzine, and valproate.

To cite a section from this ACADEMIC HIGHLIGHTS, follow the format below:
Brady KT. Presentation and epidemiology of PTSD, pp 907–909. In: Pollack MH, chair. Trauma and Stress: Diagnosis and Treatment [CME ACADEMIC HIGHLIGHTS]. J Clin Psychiatry 2001;62:906–915

Note: For the CME Posttest for this article, see pages 916–917.