01 - 11.1 Posttraumatic Stress Disorder and Acute

11.1 Posttraumatic Stress Disorder and Acute Stress Disorder

Trauma- and Stressor-Related Disorders 11.1 Posttraumatic Stress Disorder and Acute Stress Disorder Both posttraumatic stress disorder (PTSD) and acute stress disorder are marked by increased stress and anxiety following exposure to a traumatic or stressful event. Traumatic or stressful events may include being a witness to or being involved in a violent accident or crime, military combat, or assault, being kidnapped, being involved in a natural disaster, being diagnosed with a life-threatening illness, or experiencing systematic physical or sexual abuse. The person reacts to the experience with fear and helplessness, persistently relives the event, and tries to avoid being reminded of it. The event may be relived in dreams and waking thoughts (flashbacks). The stressors causing both acute stress disorder and PTSD are sufficiently overwhelming to affect almost everyone. They can arise from experiences in war, torture (discussed in detail below), natural catastrophes, assault, rape, and serious accidents, for example, in cars and in burning buildings. Persons reexperience the traumatic event in their dreams and their daily thoughts; they are determined to avoid anything that brings the event to mind and they undergo a numbing of responsiveness along with a state of hyperarousal. Other symptoms are depression, anxiety, and cognitive difficulties such as poor concentration. A link between acute mental syndromes and traumatic events has been recognized for more than 200 years. Observations of trauma-related syndromes were documented following the Civil War, and early psychoanalytic writers, including Sigmund Freud, noted a relation between neurosis and trauma. Considerable interest in posttraumatic mental disorders was stimulated by observations of “battle fatigue,” “shell shock,” and “soldier’s heart” in both World Wars I and II. Moreover, increasing documentation of mental reactions to the Holocaust, to a series of natural disasters, and to assault contributed to the growing recognition of a close relation between trauma and psychopathology. EPIDEMIOLOGY The lifetime incidence of PTSD is estimated to be 9 to 15 percent and the lifetime prevalence of PTSD is estimated to be about 8 percent of the general population, although an additional 5 to 15 percent

may experience subclinical forms of the disorder. The lifetime prevalence rate is 10 percent in women and 4 percent in men. According to the National Vietnam Veterans Readjustment Study (NVVRS), 30 percent of men develop full-blown PTSD after having served in the war and an additional 22.5 percent develop partial PTSD, falling just short of qualifying for the disorder. Among veterans of the Iraq

and Afghanistan wars, 13 percent received the diagnosis of PTSD. Although PTSD can appear at any age, it is most prevalent in young adults, because they tend to be more exposed to precipitating situations. Children can also have the disorder (see Section 31.11b). Men and women differ in the types of traumas to which they are exposed. Historically, men's trauma was usually combat experience, and women's trauma was most commonly assault or rape. The disorder is most likely to occur in those who are single, divorced, widowed, socially withdrawn, or of low socioeconomic level, but anyone can be effected, no one is immune. The most important risk factors, however, for this disorder are the severity, duration, and proximity of a person's exposure to the actual trauma. A familial pattern seems to exist for this disorder, and first-degree biological relatives of persons with a history of depression have an increased risk for developing PTSD following a traumatic event. COMORBIDITY Comorbidity rates are high among patients with PTSD, with about two thirds having at least two other disorders. Common comorbid conditions include depressive disorders, substance-related disorders, anxiety disorders, and bipolar disorders. Comorbid disorders make persons more vulnerable to develop PTSD. ETIOLOGY Stressor By definition, a stressor is the prime causative factor in the development of PTSD. Not everyone experiences the disorder after a traumatic event, however. The stressor alone does not suffice to cause the disorder. Clinicians must also consider individual's preexisting biological and psychosocial factors and events that happened before and after the trauma. For example, a member of a group who lived through a disaster can sometimes better deal with trauma because others have also shared the experience. The stressor's subjective meaning to a person is also important. For example, survivors of a catastrophe may experience guilt feelings (survivor guilt) that can predispose to, or exacerbate, PTSD. Three weeks after a train derailment, a 42-year-old budget analyst presented to the mental health clinic. He noted that he was embarrassed to seek care, as he was previously a firefighter, but he felt he needed "some reassurance that what I'm experiencing is normal." He reported that, since the wreck, he had been feeling nervous and on edge. He experienced some difficulty focusing his attention at work, and he had occasional intrusive recollections of "the way the ground just shook; the tremendous 'bang' and then the screaming when the train rolled over." He noted that he had spoken with five business colleagues who were also on the train, and three acknowledged similar symptoms. However, they said that they were improving. He

was more concerned about the frequency of tearful episodes, sometimes brought on by hearing the name of a severely injured friend, but, at other times, occurring "for no particular reason." In addition, he noted that, when he evacuated the train, rescue workers gave him explicit directions about where to report, and, although he complied, he now felt extremely guilty about not returning to the train to assist in the rescue of others. He reported a modest decrease in appetite and denied weight loss but noted that he had stopped jogging during his lunch break. He had difficulty initiating sleep, so he had begun consuming a "glass or two" of wine before bed to help with this. He did not feel rested on awakening. He denied suicidal ideation or any psychotic symptoms. His sister had taken an antidepressant several years ago, but he did not desire medication. He feared

that side effects could further diminish his ability to function at the workplace and could cause him to gain weight. (Courtesy of D. M. Benedek, M.D., R. J. Ursano, M.D., and H. C. Holloway, M.D.) Risk Factors Even when faced with overwhelming trauma, most persons do not experience PTSD symptoms. The National Comorbidity Study found that 60 percent of males and 50 percent of females had experienced some significant trauma, whereas the reported lifetime prevalence of PTSD, as mentioned earlier, was only about 8 percent. Similarly, events that may appear mundane or less than catastrophic to most persons can produce PTSD in some. Evidence indicates of a dose–response relationship between the degree of trauma and the likelihood of symptoms. Table 11.1-1 summarizes vulnerability factors that appear to play etiological roles in the disorder. Table 11.1-1 Predisposing Vulnerability Factors in Posttraumatic Stress Disorder Psychodynamic Factors The psychoanalytic model of the PTSD hypothesizes that the trauma has reactivated a previously quiescent, yet unresolved psychological conflict. The revival of the childhood trauma results in regression and the use of the defense mechanisms of repression, denial, reaction formation, and undoing. According to Freud, a splitting of

consciousness occurs in patients who reported a history of childhood sexual trauma. A preexisting conflict might be symbolically reawakened by the new traumatic event. The ego relives and thereby tries to master and reduce the anxiety. Psychodynamic themes in PTSD are summarized in Table 11.1-2. Persons who suffer from alexithymia, the inability to identify or verbalize feeling states, are incapable of soothing themselves when under stress. Table 11.1-2 Psychodynamic Themes in Posttraumatic Stress Disorder Cognitive-Behavioral Factors The cognitive model of PTSD posits that affected persons cannot process or rationalize the trauma that precipitated the disorder. They continue to experience the stress and attempt to avoid experiencing it by avoidance techniques. Consistent with their partial ability to cope cognitively with the event, persons experience alternating periods of acknowledging and blocking the event. The attempt of the brain to process the massive amount of information provoked by the trauma is thought to produce these alternating periods. The behavioral model of PTSD emphasizes two phases in its development. First, the trauma (the unconditioned stimulus) that produces a fear response is paired, through classic conditioning, with a conditioned stimulus (physical or mental reminders of the trauma, such as sights, smells, or sounds). Second, through instrumental learning, the conditioned stimuli elicit the fear response independent of the original unconditioned stimulus, and persons develop a pattern of avoiding both the conditioned stimulus and the unconditioned stimulus. Some persons also receive secondary gains from the external world, commonly monetary compensation, increased attention or sympathy, and the satisfaction of dependency needs. These gains reinforce the disorder and its persistence. Biological Factors The biological theories of PTSD have developed both from preclinical studies of animal models of stress and from measures of biological variables in clinical populations with the disorder. Many neurotransmitter systems have been implicated by both sets of data.

Preclinical models of learned helplessness, kindling, and sensitization in animals have led to theories about norepinephrine, dopamine, endogenous opioids, and benzodiazepine receptors and the hypothalamic-pituitary-adrenal (HPA) axis. In clinical populations, data have supported hypotheses that the noradrenergic and endogenous opiate systems, as well as the HPA axis, are hyperactive in at least some patients with PTSD. Other major biological findings are increased activity and responsiveness of the autonomic nervous system, as evidenced by elevated heart rates and blood pressure readings and by abnormal sleep architecture (e.g., sleep fragmentation

and increased sleep latency). Some researchers have suggested a similarity between PTSD and two other psychiatric disorders: major depressive disorder and panic disorder. Noradrenergic System. Soldiers with PTSD-like symptoms exhibit nervousness, increased blood pressure and heart rate, palpitations, sweating, flushing, and tremors— symptoms associated with adrenergic drugs. Studies found increased 24-hour urine epinephrine concentrations in veterans with PTSD and increased urine catecholamine concentrations in sexually abused girls. Further, platelet α2- and lymphocyte βadrenergic receptors are downregulated in PTSD, possibly in response to chronically elevated catecholamine concentrations. About 30 to 40 percent of patients with PTSD report flashbacks after yohimbine (Yocon) administration. Such findings are strong evidence for altered function in the noradrenergic system in PTSD. Opioid System. Abnormality in the opioid system is suggested by low plasma βendorphin concentrations in PTSD. Combat veterans with PTSD demonstrate a naloxone (Narcan)-reversible analgesic response to combat-related stimuli, raising the possibility of opioid system hyperregulation similar to that in the HPA axis. One study showed that nalmefene (Revex), an opioid receptor antagonist, was of use in reducing symptoms of PTSD in combat veterans. Corticotropin-Releasing Factor and the HPA Axis. Several factors point to dysfunction of the HPA axis. Studies have demonstrated low plasma and urinary free cortisol concentrations in PTSD. More glucocorticoid receptors are found on lymphocytes, and challenge with exogenous corticotropin-releasing factor (CRF) yields a blunted corticotropin (ACTH) response. Further, suppression of cortisol by challenge with low-dose dexamethasone (Decadron) is enhanced in PTSD. This indicates hyperregulation of the HPA axis in PTSD. Also, some studies have revealed cortisol hypersuppression in trauma-exposed patients who develop PTSD, compared with patients exposed to trauma who do not develop PTSD, indicating that it might be specifically associated with PTSD and not just trauma. Overall, this hyperregulation of the HPA axis differs from the neuroendocrine activity usually seen during stress and in other disorders such as depression. Recently, the role of the hippocampus in PTSD has received increased attention, although the issue remains controversial. Animal studies have shown that stress is associated with structural changes in the hippocampus, and

studies of combat veterans with PTSD have revealed a lower average volume in the hippocampal region of the brain. Structural changes in the amygdala, an area of the brain associated with fear, have also been demonstrated. DIAGNOSIS The 5th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) criteria for PTSD (Table 11.1-3) specify that the symptoms of intrusion, avoidance, alternations of mood and cognition, and hyperarousal must have lasted more than 1 month. The DSM-5 diagnosis of PTSD allows the physician to specify if the symptoms occur in preschool-aged children or with dissociative (depersonalization/derealization) symptoms. For patients whose symptoms have been present less than 1 month, the appropriate diagnosis may be acute stress disorder (Table 11.1-4). Table 11.1-3 DSM-5 Diagnostic Criteria for Posttraumatic Stress Disorder

Table 11.1-4 DSM-5 Diagnostic Criteria for Acute Stress Disorder

Mrs. M sought treatment for symptoms that she developed in the wake of an assault that had occurred about 6 weeks prior to her psychiatric evaluation. While leaving work late one evening, Mrs. M was attacked in a parking lot next to the hospital in which she worked. She was raped and badly beaten but was able to escape and call for help. On referral, Mrs. M reported frequent intrusive thoughts about the assault, including nightmares about the event and recurrent intrusive

visions of her assailant. She reported that she now took the bus to work to avoid the scene of the attack and that she had to change her work hours so that she did not have to leave the building after dark. In addition, she reported that she had difficulty interacting with men, particularly those who resembled her attacker, and that she consequently avoided

such interactions whenever possible. Mrs. M described increased irritability, difficulty staying asleep at night, poor concentration, and an increased focus on her environment, particularly after dark. (Courtesy of Erin B. McClure-Tone, Ph.D., and Daniel S. Pine, M.D.) CLINICAL FEATURES Individuals with PTSD show symptoms in three domains: intrusion symptoms following the trauma, avoiding stimuli associated with the trauma, and experiencing symptoms of increased automatic arousal, such as an enhanced startle. Flashbacks, in which the individual may act and feel as if the trauma were reoccurring, represent a classic intrusion symptom. Other intrusion symptoms include distressing recollections or dreams and either physiological or psychological stress reactions to exposure to stimuli that are linked to the trauma. An individual must exhibit at least one intrusion symptom to meet the criteria for PTSD. Symptoms of avoidance associated with PTSD include efforts to avoid thoughts or activities related to the trauma, anhedonia, reduced capacity to remember events related to the trauma, blunted affect, feelings of detachment or derealization, and a sense of a foreshortened future. Symptoms of increased arousal include insomnia, irritability, hypervigilance, and exaggerated startle. A 40-year-old man watched the September 11, 2001, terrorist attack on the World Trade Center on television. Immediately thereafter he developed feelings of panic associated with thoughts that he was going to die. The panic disappeared within a few hours; however, for the next few nights he had nightmares with obsessive thoughts about dying. He sought consultation and reported to the psychiatrist that his wife had been killed in a plane crash 20 years earlier. He described having adapted to the loss “normally” and was aware that his current symptoms were probably related to that traumatic event. On further exploration in brief psychotherapy, he realized that his reactions to his wife’s death were muted and that his relationship with her was ambivalent. At the time of her death, he was contemplating divorce and frequently had wished her dead. He had never fully worked through the mourning process for his wife, and his catastrophic reaction to the terrorist attack was related, in part, to those suppressed feelings. He was able to recognize his feelings of guilt related to his wife and his need for punishment manifested by thinking he was going to die. Gulf War Syndrome In the Persian Gulf War against Iraq, which began in 1990 and ended in 1991, approximately 700,000 American soldiers served in the coalition forces. Upon their return, more than 100,000 US veterans reported a vast array of health problems, including irritability, chronic fatigue, shortness of breath, muscle and joint pain,

migraine headaches, digestive disturbances, rash, hair loss, forgetfulness, and difficulty concentrating. Collectively, these symptoms were called the Gulf War syndrome. The US Department of Defense acknowledges that up to 20,000 troops serving in the combat area may have been exposed to chemical weapons, and the best evidence indicates that the condition is a disorder that in some cases may have been precipitated by exposure to an unidentified toxin (Table 11.1-5). One study of loss of memory found structural change in the right parietal lobe and damage to the basal ganglia with associated neurotransmitter dysfunction. A significant number of veterans have developed amyotrophic lateral sclerosis (ALS), thought to be the result of genetic mutations. Table 11.1-5 Syndromes Associated with Toxic Exposurea In a 1997 editorial in the Journal of the American Medical Association, the relationship of the Persian Gulf War syndrome and

stress was stated as follows: Physicians need to acknowledge that many Gulf War veterans are experiencing stress-related disorders and the physical consequences of stress. These conditions should not be hidden or denied, but rather are well-recognized entities that have been studied extensively in survivors of past wars, most notably the Vietnam conflict. As physicians, we should not accept a diagnosis of stress-related disorder in veterans prior to excluding treatable physical factors, but at the same time, we need to recognize the pervasive presence of stress-related illness such as hypertension, fibromyalgia, and chronic fatigue among Persian Gulf War veterans and manage these illnesses appropriately. As a nation, we need to get beyond the fallacious idea that diseases of the mind either are not real or are shameful and to better recognize that the mind and the body are inextricably linked.

In addition, thousands of Gulf War veterans developed PTSD and the differentiation between the two disorders has proved difficult. PTSD is caused by psychological stress, and Gulf War syndrome is presumed to be caused by environmental biological stressors. Signs and symptoms often overlap and both conditions may exist at the same time. 9/11/01 On September 11, 2001, terrorist activity destroyed the World Trade Center in New York City and damaged the Pentagon in Washington (Fig. 11.1-1). It resulted in more than 3,500 deaths and injuries and left many citizens in need of therapeutic intervention. One survey found a prevalence rate of 11.4 percent for PTSD and 9.7 percent for depression in US citizens 1 month after 9/11. It is estimated that more than 25,000 people suffer symptoms of PTSD related to the 9/11 attacks beyond the 1-year mark. FIGURE 11.1-1 The World Trade Center, New York City, prior to 9/11/01. (Courtesy of Kimsamoon, Inc.)

Iraq and Afghanistan In October 2001, the United States, along with Australia, Canada, and the United Kingdom, began the invasion of Afghanistan in the wake of the September 11, 2001, attacks. US forces are scheduled to withdraw by the end of 2014. On March 20, 2003, US forces, along with their allies, invaded Iraq, marking the beginning of the Iraq War, which officially ended on December 15, 2011. Both wars caused an estimated 17 percent of returning soldiers to develop PTSD. The rate of PTSD is higher in women soldiers. Women account for 11 percent of those who served in Iraq and Afghanistan and for 14 percent of patients at Veterans Administration (VA) hospitals and clinics. Women soldiers are more likely to seek help than men soldiers. The rate of suicide for active duty personnel in both of these wars have assumed epidemic proportions, with the likelihood of suicide being double that of the general population. Traumatic brain injury (TBI), the result of direct or indirect trauma to the brain, causes changes in either the gross or microscopic structure of the brain with associated signs and symptoms depending on the location of the lesion. In most cases of TBI there will be signs and symptoms of PTSD as well, complicating the picture. According to the Department of Veterans Affairs, 19 percent of veterans may have TBI. Natural Disasters Tsunami. On December 26, 2004, a massive tsunami struck the shores of Indonesia, Sri Lanka, South India, and Thailand and caused serious damage and deaths as far west as the coast of Africa and South Africa. The tsunami caused nearly 300,000 deaths and left more than 1 million people without homes. Many survivors continue to live in fear and show signs of PTSD; fishermen fear venturing out to sea, children fear playing at beaches they once enjoyed, and many families have trouble sleeping for fear of another tsunami. Hurricane. In August 2005, a category 5 hurricane, Hurricane Katrina, ravaged the Gulf of Mexico, the Bahamas, South Florida, Louisiana, Mississippi, and Alabama. Its high winds and torrential rainfall breached the levee system that protected New Orleans, Louisiana, causing major flooding. More than 1,300 people were killed and tens of thousands were left stranded. In October 2012, Hurricane Sandy landed on

the eastern coast of the United States and in the New York–New Jersey metropolitan area caused almost 150 deaths with an estimated 650,000 homes damaged or destroyed. Over 50,000 persons were believed to have developed full blown PTSD as a result. Earthquake. On January 12, 2010, a 7.0 magnitude earthquake hit Port-au-Prince, the capital of the Republic of Haiti, which had a population of approximately 3 million people. Approximately 316,000 people died, 300,000 were injured, and 1 million were made homeless. The government of Haiti also estimated that 250,000 residences and

30,000 commercial buildings had collapsed or were severely damaged, leaving 10 million cubic meters of rubble. On March 11, 2011, a 9.0 magnitude earthquake hit northeastern Japan, causing a 10-meter tsunami that reached as far as the western coast of the United States, making it the fifth largest earthquake since 1900. Approximately 15,700 people were killed, 4,700 were missing, and 5,700 were injured. It also brought Japan into its second recession in 3 years and triggered the world's biggest nuclear disaster since Chernobyl in 1986. PTSD developed among those who experienced these disasters, the full extent of which remains to be determined. Some estimates range from 50 to 75 percent of survivors experienced some or all of the signs and symptoms of PTSD. Torture The intentional physical and psychological torture of one human by another can have emotionally damaging effects comparable to, and possibly worse than, those seen with combat and other types of trauma. As defined by the United Nations, torture is any deliberate infliction of severe mental pain or suffering, usually through cruel, inhuman, or degrading treatment or punishment. This broad definition includes various forms of interpersonal violence, from chronic domestic abuse to broad-scale genocide. According to Amnesty International, torture is common and widespread in most of the 150 countries worldwide where human rights violations have been documented. Recent figures estimate that between 5 and 35 percent of the world's 14 million refugees have had at least one torture experience, and these numbers do not even account for the consequences of the current political, regional, and religious disputes in various parts of the world where torture is still practiced. DIFFERENTIAL DIAGNOSIS Because patients often exhibit complex reactions to trauma, the clinician must be careful to exclude other syndromes as well when evaluating patients presenting in the wake of trauma. It is particularly important to recognize potentially treatable medical contributors to posttraumatic symptomatology, especially head injury during the trauma. Medical contributors can usually be detected through a careful history and physical examination. Other organic considerations that can both cause and exacerbate the symptoms are epilepsy, alcohol-use disorders, and other substance-related disorders. Acute intoxication or withdrawal from some substances may also present a clinical picture that is difficult to distinguish from the disorder until the effects of the substance have worn off. Symptoms of PTSD can be difficult to distinguish from both panic disorder and generalized anxiety disorder, because all three syndromes are associated with prominent anxiety and autonomic arousal. Keys to correctly diagnosing PTSD involve a careful review of the time course relating the symptoms to a traumatic event. PTSD is also associated with re-experiencing and avoidance of a trauma, features typically not

present in panic or generalized anxiety disorder. Major depression is also a frequent concomitant of PTSD. Although the two syndromes are not usually difficult to distinguish phenomenologically, it is important to note the presence of comorbid depression, because this can influence treatment of PTSD. PTSD must be differentiated from a series of related disorders that can exhibit phenomenological similarities, including borderline personality disorder, dissociative disorders, and

factitious disorders. Borderline personality disorder can be difficult to distinguish from PTSD. The two disorders can coexist or even be causally related. Patients with dissociative disorders do not usually have the degree of avoidance behavior, the autonomic hyperarousal, or the history of trauma that patients with PTSD report. COURSE AND PROGNOSIS PTSD usually develops some time after the trauma. The delay can be as short as 1 week or as long as 30 years. Symptoms can fluctuate over time and may be most intense during periods of stress. Untreated, about 30 percent of patients recover completely, 40 percent continue to have mild symptoms, 20 percent continue to have moderate symptoms, and 10 percent remain unchanged or become worse. After 1 year, about 50 percent of patients will recover. A good prognosis is predicted by rapid onset of the symptoms, short duration of the symptoms (less than 6 months), good premorbid functioning, strong social supports, and the absence of other psychiatric, medical, or substance-related disorders or other risk factors. In general, the very young and the very old have more difficulty with traumatic events than do those in midlife. For example, about 80 percent of young children who sustain a burn injury show symptoms of PTSD 1 or 2 years after the initial injury; only 30 percent of adults who suffer such an injury have symptoms of PTSD after 1 year. Presumably, young children do not yet have adequate coping mechanisms to deal with the physical and emotional insults of the trauma. Likewise, older persons are likely to have more rigid coping mechanisms than younger adults and to be less able to muster a flexible approach to dealing with the effects of trauma. Furthermore, the traumatic effects can be exacerbated by physical disabilities characteristic of late life, particularly disabilities of the nervous system and the cardiovascular system, such as reduced cerebral blood flow, failing vision, palpitations, and arrhythmias. Preexisting psychiatric disability, whether a personality disorder or a more serious condition, also increases the effects of particular stressors. PTSD that is comorbid with other disorders is often more severe and perhaps more chronic and may be difficult to treat. The availability of social supports may also influence the development, severity, and duration of PTSD. In general, patients who have a good network of social support are less likely to have the disorder and to experience it in its severe forms and are more likely to recover faster. TREATMENT When a clinician is faced with a patient who has experienced a significant trauma, the

major approaches are support, encouragement to discuss the event, and education about a variety of coping mechanisms (e.g., relaxation). In encouraging persons to talk about the event it is imperative that the clinician allow the person to proceed at his or her own pace. Some patients will not be willing to talk until well after the event has passed, and those wishes should be respected. To press a person who is reluctant to talk about a trauma into doing so is likely to increase rather than decrease the risk of developing PTSD. The use of sedatives and hypnotics can also be helpful in some cases. When a patient has experienced a traumatic event in the past and has now developed PTSD, the emphasis should be on education about the disorder and its treatment, both pharmacological and psychotherapeutic. The clinician should also work to destigmatize the notion of mental illness and PTSD. Additional support for the patient and the family can be obtained through local and national support groups for patients with PTSD. Pharmacotherapy Selective serotonin reuptake inhibitors (SSRIs), such as sertraline (Zoloft) and paroxetine (Paxil), are considered first-line treatments for PTSD, owing to their efficacy, tolerability, and safety ratings. SSRIs reduce symptoms from all PTSD symptom clusters and are effective in improving symptoms unique to PTSD, not just symptoms similar to those of depression or other anxiety disorders. Buspirone (BuSpar) is serotonergic and may also be of use. The efficacy of imipramine (Tofranil) and amitriptyline (Elavil), two tricyclic drugs, in the treatment of PTSD is supported by a number of

well-controlled clinical trials. Although some trials of the two drugs have had negative findings, most of these trials had serious design flaws, including too short a duration. Dosages of imipramine and amitriptyline should be the same as those used to treat depressive disorders, and an adequate trial should last at least 8 weeks. Patients who respond well should probably continue the pharmacotherapy for at least 1 year before an attempt is made to withdraw the drug. Some studies indicate that pharmacotherapy is more effective in treating the depression, anxiety, and hyperarousal than in treating the avoidance, denial, and emotional numbing. Other drugs that may be useful in the treatment of PTSD include the monoamine oxidase inhibitors (MAOIs) (e.g., phenelzine [Nardil]), trazodone (Desyrel), and the anticonvulsants (e.g., carbamazepine [Tegretol], valproate [Depakene]). Some studies have also revealed improvement in PTSD in patients treated with reversible monoamine oxidase inhibitors (RIMAs). Use of clonidine (Catapres) and propranolol (Inderal), which are antiadrenergic agents, is suggested by the theories about noradrenergic hyperactivity in the disorder. There are almost no positive data concerning the use of antipsychotic drugs in the disorder, so the use of drugs such as haloperidol (Haldol) should be reserved for the short-term control of severe aggression and agitation. Research is ongoing about the use of opioid receptor agonists during traumatic events as a preventative against developing PTSD.

Psychotherapy Psychodynamic psychotherapy may be useful in the treatment of many patients with PTSD. In some cases, reconstruction of the traumatic events with associated abreaction and catharsis may be therapeutic, but psychotherapy must be individualized because reexperiencing the trauma overwhelms some patients. Psychotherapeutic interventions for PTSD include behavior therapy, cognitive therapy, and hypnosis. Many clinicians advocate time-limited psychotherapy for the victims of trauma. Such therapy usually takes a cognitive approach and also provides support and security. The short-term nature of psychotherapy minimizes the risk of dependence and chronicity, but issues of suspicion, paranoia, and trust often adversely affect compliance. Therapists should overcome patients' denial of the traumatic event, encourage them to relax, and remove them from the source of the stress. Patients should be encouraged to sleep, using medication if necessary. Support from persons in their environment (e.g., friends and relatives) should be provided. Patients should be encouraged to review and abreact emotional feelings associated with the traumatic event and to plan for future recovery. Abreaction—experiencing the emotions associated with the event—may be helpful for some patients. The amobarbital (Amytal) interview has been used to facilitate this process. Psychotherapy after a traumatic event should follow a model of crisis intervention with support, education, and the development of coping mechanisms and acceptance of the event. When PTSD has developed, two major psychotherapeutic approaches can be taken. The first is exposure therapy, in which the patient re-experiences the traumatic event through imaging techniques or in vivo exposure. The exposures can be intense, as in implosive therapy, or graded, as in systematic desensitization. The second approach is to teach the patient methods of stress management, including relaxation techniques and cognitive approaches, to coping with stress. Some preliminary data indicate that, although stress management techniques are effective more rapidly than exposure techniques, the results of exposure techniques last longer. Another psychotherapeutic technique that is relatively novel and somewhat controversial is eye movement desensitization and reprocessing (EMDR), in which the patient focuses on the lateral movement of the clinician's finger while maintaining a mental image of the trauma experience. The general belief is that symptoms can be relieved as patients work through the traumatic event while in a state of deep relaxation. Proponents of this treatment state it is as effective, and possibly more effective, than other treatments for PTSD and that it is

preferred by both clinicians and patients who have tried it. In addition to individual therapy techniques, group therapy and family therapy have been reported to be effective in cases of PTSD. The advantages of group therapy include sharing of traumatic experiences and support from other group members. Group therapy has been particularly successful with Vietnam veterans and survivors of catastrophic disasters such as earthquakes. Family therapy often helps sustain a marriage through periods of exacerbated symptoms. Hospitalization may be necessary when symptoms are

particularly severe or when a risk of suicide or other violence exists. UNSPECIFIED TRAUMA- OR STRESSOR-RELATED DISORDER In DSM-5, the category of “unspecified trauma- or stressor-related disorder” is used for patients who develop emotional or behavioral symptoms in response to an identifiable stressor but do not meet the full criteria of any other specified trauma- or stressorrelated disorder (e.g., acute stress disorder, PTSD, or adjustment disorder). The symptoms cannot meet the criteria for another mental, medical disorder and is not an exacerbation of a preexisting mental disorder. The symptoms also cannot be attributed to the direct physiological effects of a substance. See Section 11.2 for a discussion of adjustment disorders. REFERENCES Alexander S, Kuntz S. PTSD-related sleep disturbances: Is there evidence-based treatment? JAAPA. 2012;25:44. Barnes JB, Dickstein BD, Maguen S, Neria Y, Litz BT. The distinctiveness of prolonged grief and posttraumatic stress disorder in adults bereaved by the attacks of September 11th. J Affect Disord. 2012;136:366. Benedek DM, Ursano RJ, Holloway HC. Disaster psychology: Disaster, terrorism, and war. In: Sadock BJ, Sadock VA, Ruiz P, eds. Kaplan & Sadock’s Comprehensive Textbook of Psychiatry. 9th ed. Philadelphia: Lippincott Williams & Wilkins; 2009:2187. Biggs QM, Fullerton CS, Reeves JJ, Grieger TA, Reissman D, Ursano RJ. Acute stress disorder, depression, and tobacco use in disaster workers following 9/11. Am J Orthopsychiatry. 2010;80:586. Bryant RA. Acute stress disorder as a predictor of posttraumatic stress disorder: A systematic review. J Clin Psychiatry. 2011;72:233. Cloitre M, Garvert DW, Brewin CR, Bryant RA, Maercker A. Evidence for proposed ICD-11 PTSD and complex PTSD: A latent profile analysis. Eur J Psychotraumatol. 2013;4. Elklit A, Christiansen DM. Acute stress disorder and posttraumatic stress disorder in rape victims. J Interper Viol. 2010;25(8):1470–1488. Fareed A, Eilender P, Haber M, Bremner J, Whitfield N, Drexler K. Comorbid posttraumatic stress disorder and opiate addiction: A literature review. J Addict Dis. 2013;32(2):168–179. Forneris CA, Gartlehner G, Brownley KA, Gaynes BN, Sonis J, Coker-Schwimmer E, Jonas DE, Greenblatt A, Wilkins TM, Woodell CL, Lohr KN. Interventions to prevent post-traumatic stress disorder: A systematic review. Am J Prev Med. 2013;44(6):635–650. Jamieson JP, Mendes WB, Nock MK. Improving acute stress responses: The power of reappraisal. Curr Dir Psychol Sci. 2013;22(1):51–56. Jovanovic T, Sakoman AJ, Kozari´c-Kovacˇi´c D, Meštrovi´c AH, Duncan EJ, Davis M, Norrholm SD. Acute stress disorder versus chronic posttraumatic stress disorder: Inhibition of fear as a function of time since trauma. Depress Anxiety. 2013;30(3):217–224. Le QA, Doctor JN, Zoellner LA, Feeny NC. Cost-Effectiveness of prolonged exposure therapy versus pharmacotherapy and treatment choice in posttraumatic stress disorder (the optimizing PTSD treatment trial): a doubly randomized preference trial. J Clin Psychiatry. 2014;75(3):222–230. McNally RJ. Posttraumatic stress disorder. In: Sadock BJ, Sadock VA, Ruiz P, eds. Kaplan & Sadock’s Comprehensive Textbook of Psychiatry. 9th ed. Philadelphia: Lippincott Williams & Wilkins; 2009:2650.

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