Posttraumatic Stress Disorder (DSM-IV-TR #309.81)

Posttraumatic stress disorder (PTSD), formerly known as "traumatic neurosis," may occur in practically anyone who has been exposed to an overwhelmingly traumatic event. Subsequent to the trauma, whether it be a life-threatening accident, torture, a natural disaster, or some other extraordinary calamity, patients re-experience the event over and over again as if unable to lay it to rest. A general withdrawal from present life occurs, and patients tend to be anxious and easily startled. They may have recurrent dreams of the event or experience intrusive recollection of it during the day. In extreme instances patients seem in fact to be actually reliving the event, and they may act accordingly. For example, a combat veteran may dive for cover if a child sets off a firecracker in the park.

Figures for the lifetime prevalence of this disorder vary widely according to the diagnostic criteria used, with estimates ranging from 1 to 9%. It is probably more common in females than males.

ONSET

As trauma may occur at any age, from childhood to senescence, so too can posttraumatic stress disorder. However, given that the most common precipitating traumas, such as combat, occur in early adult years, most cases have an onset in the twenties.

Symptoms may appear either acutely, within days or weeks after the trauma, or in a delayed fashion, after a latency of months or years, and appear to be more likely in situations wherein the patient, during the actual trauma, experienced dissociation, in particular a distorted perception of time. In cases of delayed onset the latency period is generally, but not always, characterized by dysphoria and occasionally by a tendency to avoid situations reminiscent of the trauma. Occasionally the latent period abruptly ends if the patient experiences a new trauma similar to the first one.

CLINICAL FEATURES

In one fashion or another, these patients become numb to the world around them. Events that used to arouse their interest now leave them unaffected and unmoved. They may complain of feeling dead inside or of having no feelings at all; some may appear listless and detached.

The experience of the trauma lives on in these patients. They have intense, vivid memories of it. Nightmares are common, and, unlike most nightmares, they have little of the fantastic in them; rather they tend to stick to the persistently disturbing facts. At times the waking recollection of the trauma may be more vivid and compelling than the patients' actual surroundings, and they may experience a "flashback" wherein they act as if the trauma were actually recurring. In extreme cases illusions or actual visual and auditory hallucinations recreate the trauma.

Situations that remind the patient of the trauma tend to be avoided. Veterans may refuse to see war films, World War II concentration camp survivors may avoid anything German. If unavoidably trapped in the situation, patients become intensely anxious, even to the point of having a panic attack.

These patients tend to be anxious, tense, and easily startled. Though often fatigued, they struggle to remain alert, as if on guard against some fresh onslaught. Most complain of difficulty with concentration. Insomnia is common. The mood is often labile. Irritability is common, and patients may become enraged with little or no provocation.

Major depression is not uncommon.

Many patients use alcohol to excess, and alcohol abuse or alcoholism may occur, with a concurrent florid exacerbation of the symptoms of posttraumatic stress disorder.

The presentation of posttraumatic stress disorder in childhood is somewhat different from that just described for adults. Many children recreate the trauma while playing. A tortured child may again and again torture dolls or stuffed animals. Nightmares may be less connected with the trauma; they may simply be "scary dreams." Some children come to believe that they will die young; they may seem resigned to it and be disinclined to plan or talk about careers, marriage, or family.

COURSE

In about one half of all cases, symptoms remit spontaneously within months, and this appears to be more often the case when the onset is acute.

A chronic course, however, is not uncommon. A delayed onset tends to predict this, as does a persistence of symptoms for more than a half year. When the course is chronic, symptoms may persist in a waxing and waning fashion for years or decades.

COMPLICATIONS

Preoccupied and more involved with the trauma than with their present life, these patients may let marriage and career slip away from them. Their anxiety and irritability often strain whatever relationships remain; violence may incur legal consequences.

ETIOLOGY

By far the best predictor of posttraumatic stress disorder is the type and severity of the trauma itself. Products of human cruelty, such as torture, or incarceration in a death camp, commonly produce this disorder. Events that catch persons by surprise and then leave them with no social support afterward, such as a typhoon that devastates a community, likewise provide fertile ground for the development of posttraumatic stress disorder. Conversely, certain traumatic events, such as car accidents, are less likely to produce this disorder.

The fact that, regardless of the severity of the trauma, not all survivors develop posttraumatic stress disorder indicates that other factors are involved. Twin studies have suggested a genetic susceptibility; however, it is not clear whether the inherited factor is a susceptibility to the development of posttraumatic stress disorder *per se* or rather a tendency to become involved in high-risk activities.

Biochemical and endocrinologic studies of patients have yielded interesting findings. Noradrenergic activity is clearly abnormal. CSF levels of norepinephrine are increased, and there is a generalized increased reactivity of the sympathetic nervous system; furthermore, administration of yohimbine, a noradrenergic agonist, will often provoke symptoms, such as "flashbacks." Serotoninergic activity is also abnormal, as suggested by a decreased number of paroxetine binding sites on platelets and the ability of mCPP, a serotonin mixed agonist/antagonist, to induce flashbacks. The hypothalamo-pitutitary-adrenal cortex axis has been extensively studied, and shows unique abnormalities. CSF levels of CRF are increased, and this is similar to what is found in depression. However, in stark contrast to what is seen in depression, basal cortisol levels are decreased in posttraumatic stress disorder and the low-dose dexamethasone suppression test shows an enhanced suppression of cortisol.

MRI studies, though not entirely in agreement, strongly suggest that the hippocampi of patients with posttraumatic stress disorder are slightly smaller than those of controls. Initially, it was felt that this might be related to the stress of the disorder itself, perhaps being mediated by the high cortisol levels normally seen in times of great stress. The finding, however, of reduced cortisol levels, as noted above, makes this explanation unlikely, and raises the possibility that this subtle hippocampal atrophy might have preceded the stress.

Overall, it appears reasonable to invoke a "stress-diathesis" model for the etiology of posttraumatic stress disorder. However, although the "stress" is clear, the nature of the "diathesis" is not. Conceivably, however, there may be subtle alterations in noradrenergic or serotoninergic activity in those central nervous system structures, such as the amygdala, hippocampus and hypothalamus that mediate anxiety, remembrance and hormonal tone.

DIFFERENTIAL DIAGNOSIS

Diagnostic difficulty may arise if a depressive episode happens to occur after a significant trauma. In such cases patients may be withdrawn, agitated, and tend to ruminate over their misfortune, and thus appear similar to patients with a posttraumatic stress disorder. However, certain qualitative differences exist. The depressed mood contrasts with the detached "numb" experience seen in posttraumatic stress disorder and depressive ruminations are heavy, leaden, and plodding, in sharp contrast to the starkly vivid nature of the intrusive recollections of posttraumatic stress disorder.

Should head injury occur during the trauma, one may see a contribution of a postconcussion syndrome to any difficulty with memory and concentration.

Malingerers or those with factitious illnesses may present a history compatible with posttraumatic stress disorder. Examination of records, particularly military service records, often exposes the lie. In cases where in fact a trauma occurred, a diagnosis of malingering may remain in doubt until symptoms resolve after adjudication of the lawsuit.

Before leaving this discussion of differential diagnosis, some words are in order regarding the putative entity "acute stress disorder." As described in DSM-IV, this diagnosis is given to patients whose illness is essentially clinically identical to posttraumatic stress disorder with the exception of course: when the illness undergoes a spontaneous remission within one month of the trauma, one is asked to make a diagnosis of "acute stress disorder"; however, if the illness persists beyond that time one is supposed to revise the diagnosis to posttraumatic stress disorder. Given that most patients diagnosed with "acute stress disorder" remain ill beyond a month and thus have to have their diagnosis revised, this distinction between "acute stress disorder" and posttraumatic stress disorder may in fact be artificial and unwarranted. It would perhaps be more appropriate to simply say that some cases of posttraumatic stress disorder run a rapid course, resolving within weeks, and let it go at that.

TREATMENT

Both cognitive therapy and behavioral therapy are beneficial. Certain medications are also effective; however, there have not, as yet, been controlled comparisons of medications with either cognitive or behavior therapy. Fluoxetine in doses of 60 mg, paroxetine in doses of 20 to 50 mg, and sertraline in doses of 50 to 200 mg have all been found effective. Both imipramine and phenelzine are also effective; however, their side-effect profiles argue against using them as first line agents. Recent work has also focused on pharmacologic treatment of the troubling nightmares seen in this disorder, and two medications are useful here: cyproheptadine may be given in doses of from 4 to 12 mg hs; prazosin may be started at 1 mg hs and increased in similar increments every few days until patients obtain relief or limiting side-effects occur; most patients respond to a dose of approximately 10 mg. Interestingly, prazosin, in addition to reducing the frequency of nightmares, also led to a diminution of other symptoms.

As noted earlier, alcoholism or alcohol abuse not uncommonly accompanies posttraumatic stress disorder, and when this is the case, it is critical to treat the substance use disorder either first or concurrently with the posttraumatic stress disorder.

BIBLIOGRAPHY

Adler A. Neuropsychiatric complications in victims of Boston's Coconut Grove disaster. *Journal of the American Medical Association* 1943; 123:1098–1101.

Baker DG, West SA, Nicholson WE, et al. Serial CSF corticotropin-releasing hormone levels and adrenocortical activity in combat veterans with posttraumatic stress disorder. *The American Journal of Psychiatry* 1999;156:585–588.

Bryant RA, Harvey AG. Delayed-onset posttraumatic stress disorder: a prospective evaluation. *The Australian and New Zealand Journal of Psychiatry* 2002;36:205–209.

Frank JB, Kosten TR, Giller EL, et al. A randomized clinical

trial of phenelzine and imipramine for posttraumatic stress disorder. *The American Journal of Psychiatry* 1988;145:1289–1291.

Geracioti TD, Baker DG, Ekhator NN, et al. CSF norepinephrine concentrations in posttraumatic stress disorder. *The American Journal of Psychiatry* 2001;158:1227–1230.

Harvey AG, Bryant RA. Two-year prospective evaluation of the relationship between acute stress disorder and posttraumatic stress disorder following mild traumatic brain injury. *The Australian and New Zealand Journal of Psychiatry* 2000;157:626–628.

Horowitz M, Willner N, Kaltreider N, et al. Signs and symptoms of posttraumatic stress disorder. *Archives of General Psychiatry* 1980; 37:85–92.

Jacobs-Rebhun S, Schnurr PP, Friedman MJ, et al. Posttraumatic stress disorder and sleep difficulty. *The American Journal of Psychiatry* 2000;157:1525–1526.

Kanter ED, Wilkinson CW, Radant AD, et al. Glucocorticoid feedback sensitivity and adrenocortical responsiveness in posttraumatic stress disorder. *Biological Psychiatry* 2001;50:238–245.

Kinzie JD, Fredrickson RH, Ben R, et al. Posttraumatic stress disorder among survivors of Cambodian concentration camps. *The American Journal of Psychiatry* 1984;141:645–650.

Lee KA, Vaillant GE, Torrey WC, et al. A 50-year prospective study of the psychological sequelae of World War II combat. *The American Journal of Psychiatry* 1995;152:516–522.

Marks I, Lovell K, Noshirvani H, et al. Treatment of posttraumatic stress disorder by exposure and/or cognitive restructuring: a controlled study. *Archives of General Psychiatry* 1998;55:317–325.

Martenyi F, Brown EB, Zhang H, et al. Fluoxetine versus placebo in posttraumatic stress disorder. *The American Journal of Psychiatry* 2002;63:199–206.

Raskind MA, Peskind ER, Kanter ED, et al. Reduction of nightmares and other PTSD symptoms in combat veterans by prazosin: a placebocontrolled study. *The American Journal of Psychiatry* 2003;160: 371–373.

Southwick SM, Krystal JH, Bremner JD, et al. Noradrenergic and serotoninergic function in posttraumatic stress disorder. *Archives of General Psychiatry* 1997;54:749–758.

Stein MB, Jang KL, Taylor S, et al. Genetic and environmental influences on trauma exposure and posttraumatic stress disorder symptoms: a twin study. *The American Journal of Psychiatry* 2002;259:1675–1681.

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. *The Journal of Clinical Psychiatry* 2001;62: 860–868.

Villarreal G, Hamilton DA, Petropoulos H, et al. Reduced hippocampal volume and total white matter volume in posttraumatic stress disorder. *Biological Psychiatry* 2002;52:119–125.

Zohar J, Amital D, Miodownik C, et al. Double-blind placebo-controlled pilot study of sertraline in military veterans with posttraumatic stress disorder. *Journal of Clinical Psychopharmacology* 2002;22:190–195.