The prevalence of exposure to traumatic events as well as the development of PTSD also varies across racial and ethnic groups, with high rates of exposure to violence among African Americans, American Indians, and Alaska Natives, compared to members of more economically advantaged groups (310, 311). For example, in one study, 82% of American Indians and Alaska Natives had been exposed to one traumatic event, and the prevalence of PTSD was 22% (4). American Indians have a rate of violent victimization that is more than twice the national average (312), whereas rates of PTSD among American Indians and Alaska Natives are about threefold higher than in the general population. An investigation of Northern Plains Indian youths in grades 8 through 11 found that 61% had been exposed to some kind of traumatic event (313). These adolescents were reported to have more trauma-related symptoms but not substantially higher rates of diagnosable PTSD (3%), compared to the general population (313). A study of a Southwestern American Indian community found even higher rates of experience of one or more traumatic events but also noted a higher prevalence of lifetime PTSD in this community, compared with the general U.S. population (314).

Because members of some racial and ethnic groups are more likely to have lower socioeconomic status, live in an inner-city area, or be U.S. combat veterans (315), and because such status is associated with an increased likelihood of experiencing undesirable life events (316), some racial and ethnic groups are more likely to experience ASD and PTSD (4, 314). Among veterans, an increased likelihood of traumatic early experiences (310–312, 317) may contribute to the increased rates of PTSD seen in African Americans, Hispanics, and American Indian/Alaska Natives after combat-related trauma (247, 310).

Differences in the rates of previous exposure to traumas may account, in part, for differences observed in rates of PTSD among U.S. veterans of differing ethnic and racial backgrounds. However, greater war zone exposure to traumatic experiences among African Americans (315) and American Indians (318, 319) is likely to play a large role as well. In terms of racial differences in rates of PTSD among U.S. veterans, the National Vietnam Veterans Readjustment Study found that although 10% of U.S. soldiers in Vietnam were black and 85% were white, more African American (21%) than European American (14%) veterans experienced PTSD (247). In the American Indian Vietnam Veterans Project (319), evaluation of random samples of Vietnam combat veterans from three Northwestern Plains reservations and one Southwest reservation between 1992 and 1995 showed that approximately one-third of the Northern Plains (31%) and Southwestern (27%) American Indian participants had PTSD at the time of the study. Approximately one-half had experienced the disorder in their lifetime (57% and 45%, respectively). This rate was far in excess of rates of current PTSD observed in the European American or African American veterans (247).

Hispanics also have been found to be at higher risk for war-related PTSD than their European American counterparts (247). Because the risk for Hispanics was higher than that for black veterans, minority status must not be the only risk factor (320). Of the Hispanic subgroups, Puerto Rican veterans have been found to have a higher probability of experiencing PTSD than others with similar levels of war zone stressor exposure (321). Because these differences in prevalence were not explained by exposure to stressors or acculturation and were not accompanied by significant reductions in levels of functioning, it has been proposed that differences in symptom reporting may reflect features of expressive style rather than different levels of illness (320).

National variations in rates of PTSD development have been reported across populations exposed to traumatic events. For instance, less than 5% of hospitalized European survivors of unintentional injuries (e.g., motor vehicle crashes, job-related injuries) appear to develop PTSD (322, 323). However, between 10% and 40% of survivors of both intentional (e.g., injuries associated with human malice, such as physical assaults) and unintentional injuries treated within acute care settings in the United States, England, and Australia appear to develop symptoms consistent with the disorder (34, 117, 293, 324–328). The explanations for these different rates include methodological differences, cultural differences, and diagnostic accuracy (329).

The prevalence of PTSD in countries where war and disease are endemic is substantially higher and has been reported to range between 9.4% and 37.0% of the population. For example, Bleich and colleagues (330), in a telephone survey of a representative sample of 512 Israeli adults, found that after 19 months of ongoing terrorist attacks, 16.4% had been directly exposed to a terrorist attack, 37.3% had an exposed family member or friend, and 9.4% of the sample met the symptom criteria for PTSD. Sabin and colleagues (331) found similar rates in a cross-sectional survey of Mayan refugees living in Mexico, of whom 11.8% met the symptom criteria for PTSD, as measured by the Harvard Trauma Questionnaire and Hopkins Symptom Checklist-25, 20 years after fleeing the civil conflict in Guatemala. De Jong et al. (332) used the Composite International Diagnostic Interview to assess for PTSD in community populations of four postconflict low-income countries and found a prevalence rate of PTSD of 37.4% in Algeria, 28.4% in Cambodia, 15.8% in Ethiopia, and 17.8% in Gaza.

Treatment-seeking refugees may have even higher rates of PTSD, ranging from 55% to 90% (333). Studies have revealed alarming rates of PTSD in immigrant communities with a high degree of preimmigration exposure to potentially traumatic experiences (e.g., Asian Americans and Hispanic Americans). For example, in some samples, up to 70% of refugees from Vietnam, Cambodia, and Laos met the diagnostic criteria for PTSD, in contrast to prevalence rates of about

4% for the U.S. population as a whole (334).

Studies of Southeast Asian refugees receiving mental health care have uniformly found high rates of PTSD. One study found that 70% of the subjects met the diagnostic criteria for PTSD, with Mien from the highlands of Laos and Cambodians having the highest rates (333). Another mental health study of Southeast Asian refugees (Hmong, Laotian, Cambodian, and Vietnamese) in Minnesota found that 73% had major depression, 14% had PTSD, and 6% had anxiety or somatoform disorders (335). A random community sample of Cambodian adults revealed that 45% had PTSD, and 81% experienced five or more symptoms of PTSD (336). Similarly, 43% of parents recruited from a community of resettled Cambodian refugees in Massachusetts reported the death of between one and six of their children (337). Child loss was associated with an increased likelihood of health-related concerns, a variety of somatic symptoms, and culture-bound conditions of emotional distress such as deep worrying and sadness not visible to others (337). Finally, Kinzie et al. (338) found that nearly one-half of a sample of Cambodian adolescents who survived Pol Pot's concentration camps as children had PTSD approximately 10 years after this traumatic period. Thus, many Southeast Asian refugees are at risk for PTSD associated with the events they experienced before they immigrated to the United States (311). A large community sample of Southeast Asian refugees in the United States found that preimmigration and refugee camp experiences were significant predictors of psychological distress even 5 or more years after migration (339). In this study, significant subgroup differences were found: Cambodians reported the highest levels of distress, Laotians were next, then Vietnamese. While trauma treatments may be effective for persons from Western cultures, in some Southeast Asian populations, it may be contraindicated to attempt to identify and process traumatic experiences (229).

Central American immigrants to the United States may be at risk for PTSD as a result of their preimmigration exposure to war-related trauma (340), even though they are not recognized as political refugees (311). For example, a study of Los Angeles adults who were examined for symptoms of PTSD and depression found that one-half of the Central American participants reported symptoms that were consistent with a diagnosis of PTSD (341). In comparison with recent Mexican immigrants, a greater proportion of Central American refugees reported symptoms of PTSD (50% versus 25%) (341). In another study, 60% of adult Central American refugee patients received a diagnosis of PTSD (342). Central American immigrant children seeking care at refugee service centers also had high rates of PTSD (33%) (343). In a more recent study of a systematic sample of 638 adult Latino primary care patients living in Los Angeles, Eisenman and colleagues (344) found that 54% of the sample had experienced political

violence before migration, and of these, 18% had symptoms of PTSD. Those who had experienced political violence had a 3.4-fold greater risk of meeting the criteria for a PTSD diagnosis, compared to those who had not experienced political violence.

E. NATURAL HISTORY AND COURSE

Prospective studies suggest that symptomatic distress peaks in the days and weeks after a trauma and then gradually declines over the course of the year after injury (139). In the National Comorbidity Survey, symptoms also decreased most rapidly in the first 12 months after trauma exposure (4). However, approximately one-third of persons who developed PTSD had chronic symptoms that did not remit. Although this issue is not settled (309), rates of recovery from PTSD may vary by gender. Although gender differences in the duration of PTSD are in part explained by gender differences in the type of trauma experienced, Breslau and colleagues (5, 226) found a median time to remission of symptoms of 12 months in men and 48 months in women. However, studies of motor vehicle accident victims have shown initial rates of approximately 35%, decreasing nearly 50% by 12 months postaccident (34, 345).

The responses of traumatized patients fall on a continuum, and the natural course of ASD and PTSD may vary with personality and other individual characteristics. Some individuals are relatively resistant to developing posttraumatic symptoms or report interpersonal growth experiences as a result of their traumatic exposure (229, 346). For other individuals with PTSD, however, long-lasting personality change may occur (252, 347–349). Problems of impaired affect modulation; self-destructive and impulsive behavior; dissociative symptoms; somatic complaints; feelings of ineffectiveness, shame, and despair or hopelessness; feelings of being permanently damaged; a loss of previously supportive beliefs; hostility; social withdrawal; feeling constantly threatened and being in an alert status; and impaired relationships with others all portend personality change from the individual's previous characteristics.

Investigations have also shown symptoms of PTSD to be associated with functional impairment and diminished quality of life (115, 117, 122, 293, 327, 350–353). Across veteran (122), refugee (292), and injured civilian (117, 293, 327) populations, PTSD makes an independent contribution to diminished functioning and quality of life above and beyond the effects of comorbid medical conditions and injury severity. Posttraumatic stress is also coupled with a spectrum of physical health problems and medical disorders (103, 354, 355). These considerations make the treatment of PTSD important not just from the standpoint of individual suffering but also from the perspective of the potential societal costs associated with the disorder (273, 356).

Individuals who have been exposed to trauma may also be vulnerable to subsequent traumas and have an increased likelihood of developing PTSD with repeated traumatic experiences (32, 33, 223). In individuals with a first hospitalization for psychosis, a similar pattern was observed, with exposure to multiple traumatic events being associated with greater rates of PTSD than exposure to a single trauma (48). These findings suggest that in trauma-exposed individuals, interventions should include efforts to decrease the risk for subsequent exposures to traumatic events.

V. REVIEW AND SYNTHESIS OF AVAILABLE EVIDENCE

A. ISSUES IN INTERPRETING THE LITERATURE

The empirical research on the efficacy of treatments for ASD and PTSD is not as extensive at present as that for other disorders such as major depressive disorder, schizophrenia, or bipolar

disorder. Most of the randomized clinical trials of ASD and PTSD treatments have a relatively small number of subjects, and their inclusion in the study is often determined by their having experienced one type of index trauma. In addition, exact replications of methods are the exception rather than the rule. Further study is needed to better establish the generalizability of findings across populations and various traumatic event exposures. For example, many studies are limited to combat veterans with chronic PTSD or to rape victims. Treatment for ASD has only just begun to be examined. Effectiveness studies of ASD and PTSD treatments are also limited.

The rapid recovery rate of patients with ASD and acute PTSD means that outcomes studies need to examine closely the timing of treatment administration and the rates of recovery as well as remission and relapse. Treatment studies that specifically examine critical symptoms (such as sleep disturbance or withdrawal or arousal) are also needed. Gender differences in the rates of PTSD suggest that close attention should be paid to gender differences in treatment outcomes. The widespread nature of traumatic exposures in some subpopulations, including persons living in urban environments in major cities, also means that PTSD may have gone undetected but may have existed long before the index disorder is diagnosed.

The high comorbidity of PTSD with major depression and substance abuse also complicates the interpretation of efficacy studies.

With psychosocial interventions, measuring the efficacy of one treatment may be confounded by the effects of other simultaneous treatments.

B. PSYCHOSOCIAL INTERVENTIONS

1. Individual psychotherapies

In general, psychotherapy, examined across all types of interventions and for different types of victims, is an effective intervention for PTSD. Sherman (203) conducted a meta-analysis of 17 controlled clinical trials of psychotherapy for PTSD that included behavioral, cognitive, and psychodynamic individual and group therapy with veterans, female assault victims, and victims of other traumatic events. Psychotherapy was found to have a significant beneficial effect on PTSD.

Prediction of success in psychotherapy of PTSD, however, is in its infancy. For instance, beliefs about mistrust, helplessness, meaninglessness, and unjustness of the world predict baseline PTSD symptom severity but not treatment outcome (357). Little is known about the relationship of the type of traumatic event to the type or duration of psychotherapy likely to be effective.

a) Psychodynamic psychotherapy

Psychodynamic psychotherapy for either ASD or PTSD has not been well studied by means of randomized, controlled trials. Given the fact that ASD, by definition, is an illness of relatively brief duration, long-term therapy would seem unnecessary. However, ASD may be associated with or may aggravate preexisting psychological problems, and a remote history of repeated trauma (including childhood abuse) predicts the development of PTSD. In the face of an acute trauma, dormant issues may at times become more apparent or more amenable to treatment. Considerable clinical literature and case studies comment on this phenomenon, but the extent to which such intervention might prevent the development of PTSD remains untested. For PTSD, one controlled trial by Brom et al. (201) compared psychodynamic therapy to trauma desensitization, hypnotherapy, and a control condition. All three treatments were significantly effective in reducing intrusive and avoidance symptoms. A meta-analysis of psychotherapies including psychodynamic psychotherapy—also supports this mode of treatment (203). Other less rigorous studies and reviews also suggest the efficacy of psychodynamic therapy in PTSD (21, 208, 358). Again, despite a lack of randomized, controlled trials, clinical consensus reflects the idea that a psychodynamic approach is useful in helping the patient integrate past traumatic experience(s) into a more adaptive or constructive schema of risk, safety, prevention, and protection (359, 360), thereby reducing core symptoms of PTSD.

b) Cognitive and behavior therapies

The cognitive and behavior therapies are applied in the individual, family, or group treatment forms. Although particular behavior therapies have been used as stand-alone treatment, it is more common for behavior therapy to be used in conjunction with other forms of therapy, such as cognitive approaches (e.g., cognitive behavior therapy). These complex treatments may have more than one efficacious component, and in many studies it is somewhat difficult to "dismantle" the specific contributions of the various elements of such combined treatments. Cognitive approaches to the treatment of ASD or PTSD target the distorted threat appraisal process in an effort to desensitize the patient to trauma-related triggers. These approaches often include a component of repeated exposure, either in talking about the trauma or in processing the traumatic experience.

Behavior therapy is derived from psychological models of learning that emphasize the role of environmental cues and consequences in patterning behavior. A behavioral assessment of the PTSD patient would focus on the traumatic event, the reexperiencing symptoms, the maladaptive avoidance and numbing strategies, and the pathological arousal responses that drive the disorder.

Systematic desensitization has been used to reduce anxiety associated with the traumatic stressor. The essential ingredient of systematic desensitization is the gradual and progressive exposure of the patient to feared stimuli while steps are taken to reduce elicited anxiety by displacing it with a sense of relaxation (reciprocal inhibition of the fear response). Improvements in active coping and reductions in traumatic anxiety can occur both inside and outside the sessions through the learning of relaxation techniques such as progressive muscle relaxation, diaphragmatic or meditative breathing, and guided imagery. Progressive muscle relaxation involves alternating the tensing and releasing of muscle groups throughout the body, sometimes proceeding in a head-to-toe direction. Breathing exercises concentrate on exhaling in order to generalize a calming effect, while guided imagery promotes relaxation though visualizing enjoyable places or activities. Biofeedback may be used to augment relaxation by providing the patient with instantaneous feedback on physiological variables, such as blood flow and muscle contraction. These phenomena are not normally sensed, but their continuous presentation permits the patient to exert some degree of voluntary control over variables related to tension and anxiety.

Therapeutic use of prolonged and repeated exposure to traumatic cues, either in a gradual fashion or intensively through flooding or implosion, is based on the principle that traumatic anxiety will decrease in the absence of real danger. Direct therapeutic exposure can be accomplished in vivo (directly) or in imagination. Typically, a course of exposure-based treatment begins with relaxation training and education about the symptoms of PTSD and about the rationale for having participants reexpose themselves to painful experiences. The therapist assesses the patient's ability to tolerate within-session emotion and temporary exacerbations of symptoms before implementing further treatment. If these experiences are acceptable, the patient is then led through a series of sessions in which the traumatic event and its aftermath are imagined and described and patients are asked to focus on the intense negative affects and arousal that are elicited, until they subside. Relaxation exercises and reassurance permit the patient to continue without feeling overwhelmed and abandoning the therapy. Homework assignments allow the patient to practice outside the session. In addition, the treatment may be enhanced if the patient is encouraged to confront specific places or activities in vivo. Success can be measured as complete or partial extinction of PTSD symptoms (173, 174).

Early exposure research was frequently conducted with Vietnam veterans with chronic, combat-related PTSD. Peniston's 1986 randomized, controlled study of biofeedback-assisted systematic desensitization (361) provided preliminary evidence for the potential effectiveness of high-frequency exposure therapy. In one of the early studies, which used flooding, Keane et al. (362) randomly assigned 24 combat veterans with PTSD to 14–16 sessions of flooding (N=11) or to a waiting list (N=13). Assessments at pretreatment, posttreatment, and 6-month follow-up showed improvement in reexperiencing symptoms, startle response, and memory/concentra-

tion. No improvement was seen in numbing or social avoidance. Cooper and Clum (363) studied imaginal flooding as a supplementary treatment to standard VA care. All 26 subjects completed the study, which showed that exposure increased the effectiveness of the usual treatment. Boudewyns et al. (364, 365) compared 58 Vietnam veteran inpatients with severe PTSD randomly assigned to either direct therapeutic exposure (flooding) or standard therapy. At 3-month follow-up, significantly more successes than failures were in the exposure group. In a review of the limited clinical literature on flooding, implosion, and direct therapeutic exposure for PTSD in Vietnam combat veterans, Foy et al. (183) noted the significant reductions in symptoms of intrusion and arousal but did register concern regarding reports of decompensation, distress, depletion of emotional reserves, and symptom exacerbation in some patients.

Richards et al. (366) tested four weekly sessions of imaginal exposure followed by four weekly sessions of real-life exposure (or vice versa) in 14 civilian patients with PTSD. Both groups showed a 65%–80% reduction in symptoms, with only a few differences noted, suggesting the salience of imaginal and in vivo forms of exposure. Rothbaum and Hodges (367) published a single case study of the use of a virtual reality mode of exposure for PTSD in a Vietnam veteran. The patient showed a 34% reduction in clinician-rated PTSD symptoms, which was maintained at 6-month follow-up. An open clinical trial also showed promise (368). Thus, imaginal, virtual, and in vivo exposures may each represent useful methods of delivering exposure therapy to PTSD patients.

Imagery rehearsal is another behavior therapy designed to ameliorate traumatic nightmares by having the patient recall the distressing content of recurring nightmares and repetitively envision (rehearse) a different outcome. Krakow et al. (184, 369) published two reports of a controlled study of imagery rehearsal for chronic nightmares in 168 sexual assault survivors with moderate to severe PTSD. The subjects were randomly assigned to an imagery rehearsal treatment group of to a waiting-list control group. A total of 114 subjects completed follow-up at

Participation in exposure therapy was associated with a decrease in positive (e.g., intrusive and hyperarousal) symptoms of PTSD but not negative (e.g., avoidance/numbing) symptoms. There were no additional therapeutic gains from the family sessions.

Cognitive behavior therapy has often been combined with exposure therapy and shown to be effective. In a randomized, controlled study by Fecteau and Nicki (373) of cognitive behavior therapy (N=10) versus a waiting-list condition (N=10), adults (14 women) with PTSD were treated approximately 18 months after motor vehicle accidents with physical injury. The treatment consisted of four weekly 2-hour sessions of cognitive behavior therapy, including education, relaxation training, imaginal exposure, self-directed in vivo practice, and cognitive restructuring. Five of the 10 cognitive behavior therapy subjects no longer had diagnosable PTSD after treatment, while all 10 of the waiting-list group continued to meet the criteria for PTSD. PTSD symptoms improved significantly, with the Clinician-Administered PTSD Scale (CAPS-2) score decreasing from an average of 70.9 (high/PTSD present) to 37.5 (moderate). In contrast, the subjects' Beck Depression Inventory scores did not show significant improvement, suggesting a specific effect on PTSD rather than merely a nonspecific improvement in comorbid depression. The weak response in depression measures may also have been related to chronic pain and disability status. Follow-up at 3 and 6 months showed persistent improvements in PTSD symptoms.

Cognitive therapy techniques have not always been combined with exposure techniques, allowing for some comparison of these techniques. Foa et al. (374) randomly assigned 55 female rape victims with PTSD to one of four conditions: 17 were assigned to stress inoculation training, 14 to prolonged exposure, 14 to supportive counseling (to control for nonspecific therapy effects), and 10 to a waiting-list control group. PTSD diagnoses were made by an outside clinician who used DSM-III-R criteria. The range of time since the assault varied from 3 months to 12 years, with a mean of 6.2 years (SD=6.7). Treatment consisted of nine biweekly 90minute individual sessions conducted by a female therapist. PTSD symptoms, rape-related distress, general anxiety, and depression were measured pretreatment, posttreatment, and at follow-up (mean=3.5 months posttreatment). Of the 55 patients who started the study, 10 dropped out, with no significant differences in dropout rates across the three treatment groups. However, the 10 noncompleters differed from the completers on three variables: a greater percentage of the noncompleters earned an annual income of less than \$10,000, a greater percentage were blue-collar workers, and they scored higher on the Rape Aftermath Symptom Test. Immediately after treatment, stress inoculation therapy was the most effective treatment in reducing PTSD symptoms, and prolonged exposure was also an effective treatment. The supportive counseling and waiting-list conditions improved arousal symptoms of PTSD but not the intrusion and avoidance symptoms. Three and one-half months after treatment, however, prolonged exposure appeared to be the superior treatment. Thus, although stress inoculation therapy appeared to be the most effective treatment in the short term, prolonged exposure appeared to be the most effective treatment in the long term. Furthermore, the superiority of stress inoculation therapy and prolonged exposure over supportive counseling and waiting-list placement was found only for PTSD symptoms.

Marks et al. (177) showed that cognitive therapy, exposure therapy, and exposure plus cognitive therapy were better than relaxation treatment in 87 subjects randomly assigned to ten 90-minute sessions of the four treatment groups. It is important to note that all three cognitive behavior therapy approaches were markedly better than relaxation at 1, 3, and 6 months but no better than each other in decreasing PTSD symptoms or symptom severity, producing remission of PTSD, or improving functioning at the end of the study. Similarly, Echeburua et al. (375) tested progressive relaxation training versus cognitive restructuring and self-exposure in 20 victims of sexual aggression. Most treated patients improved, but the cognitive restructuring and exposure treatment was more successful on all measures than relaxation alone. In contrast, Silver et al. (376) treated inpatient Vietnam veterans with additional EMDR, biofeedback, or relaxation training and found no statistically significant differences between cognitive restructuring and exposure treatment.

Given the similarities between cognitive behavior therapy and exposure therapy, it is not surprising that comparisons of these two modalities have shown similar treatment outcomes. In a randomized, double-blind trial of cognitive behavior therapy versus exposure therapy for chronic PTSD, Tarrier et al. (178) found that the two treatments were equally effective in reducing symptoms in a diverse group of 72 trauma patients but that neither therapy produced complete symptom remission. It is important to note that nine patients in the exposure condition versus three patients in the cognitive behavior therapy condition showed worsening symptoms. Subsequent studies showed that improvements were maintained at 6 months (186) and 12 months (187), again with no significant differences between therapies.

Similarly, in a small, randomized study of 16 refugee outpatients with PTSD, both cognitive behavior therapy and exposure therapy resulted in large improvement on all measures, and this improvement was maintained at follow-up (377). The two treatments did not differ on any measure, but cognitive behavior therapy required relatively more and longer sessions to obtain

significant results.

Studies of cognitive behavior therapy for PTSD have also examined outcomes for factors other than PTSD symptoms, such as anger. In a randomized trial, Chemtob et al. (378) assigned 15 Vietnam combat veterans with PTSD to routine VA care or to routine VA care plus 12 sessions of cognitive behavior therapy focused on anger. The 1-hour individual cognitive behavior therapy sessions involved self-monitoring of anger, devising an anger hierarchy, relaxation, cognitive restructuring skills training, and skills practice (role playing in anger-provoking situations). The anger therapy subjects had increased capacity to control anger at completion and 18-month follow-up, although there were no differences between groups on measures of psychophysiological reactions to anger provocation at treatment end. This study showed the specific clinical utility of a cognitive behavior treatment for anger as an adjunct to routine care, although no information was given on PTSD symptoms.

A few studies have indicated that a brief cognitive behavior therapy intervention in the acute posttraumatic phase can prevent PTSD while simultaneously treating ASD. Although these studies are few and included only a small number of subjects, the measured outcome of prevention of PTSD makes them very important, and their findings should be replicated. Bryant et al. (135) examined 45 civilian trauma survivors with ASD treated with five sessions of either prolonged exposure, prolonged exposure plus anxiety management, or supportive counseling begun within 2 weeks of the traumatic event. After treatment, the criteria for PTSD were met by significantly fewer of the patients who received prolonged exposure (14%) and prolonged exposure plus anxiety management (20%) than of those who received supportive counseling (56%). The effect of the two active treatments was maintained at 6-month follow-up after the traumatic event. In contrast to previous reports that 80% of patients who initially meet the criteria for ASD will have chronic PTSD 6 months after the trauma, this study found that patients who received supportive counseling had a rate of PTSD of 67%, indicating that supportive counseling may be somewhat helpful in ameliorating symptoms of PTSD. However, substantially fewer individuals met the criteria for PTSD after either prolonged exposure plus anxiety management (23%) or prolonged exposure (15%), suggesting even greater efficacy of these treatments. There were no differences in outcome between the prolonged exposure and prolonged exposure plus anxiety management interventions, indicating that anxiety management did not contribute to treatment efficacy.

Similarly, Foa et al. (379) treated female victims of recent rape or aggravated assault with a brief prevention program consisting of four 2-hour sessions of cognitive behavior therapy and education, compared with a matched assessment control group. Two months after the assault, only 10% of the brief prevention group met the PTSD criteria, in contrast to 70% of the control group. The brief prevention group did significantly better on measures of depression and reexperiencing symptoms than did the control group members, with an effect size for brief prevention of 1.22.

Gillespie et al. (380) found a similar effect size in a case series treated with cognitive therapy. In this study, a consecutive series of 91 patients were treated within 2 weeks after exposure to a car bomb in Omagh, Northern Ireland, with cognitive therapy along the lines advocated by Ehlers and Clark (381). Neither comorbidity nor the presence or absence of a supportive relationship predicted treatment response. Those who were physically injured improved less significantly than witnesses or those who were not injured. Witnesses and emergency personnel did not differ in their degree of improvement. It is interesting to note that there was a nonsignificant trend for patients who were treated later to improve more than those who were treated earlier, which highlights a need to better understand the timing of treatment interventions. Generalization from these findings is limited by the fact that patients received varying numbers of sessions, there was no control group, and the therapy was not manualized.

c) Eye movement desensitization and reprocessing (EMDR)

EMDR is generally seen as a combination of elements of cognitive behavior therapy, exposure therapy (albeit brief and interrupted exposures), and a unique attention to eye movements. Since cognitive behavior therapy and exposure therapy have been shown to have efficacy in treatment of PTSD, a major question about EMDR has been whether the eye movements contribute to therapy outcome. A number of factors have contributed to the difficulty in establishing whether EMDR effects are distinct from those of cognitive behavior therapy and exposure therapy. Studies of EMDR have included a range of trauma types, weighted toward persons with combat exposure but also including adults with histories of childhood sexual abuse, adults with adult sexual assault, adults after a major hurricane, and (for a few studies) adults with mixed civilian traumas. There is great variation in the protocols, from one 90-minute session to 8-10 sessions. The number of subjects in the studies has also varied widely. Several studies compared EMDR to waiting-list, supportive counseling, or active listening control groups. Others compared EMDR to different forms of prolonged exposure, while several employed dismantling designs that compared EMDR with or without eye movement or finger tapping procedures. Outcome variables primarily included self-report PTSD scales (often, the Impact of Event Scale), with a few using more general symptom checklists or depression inventories. No study has included structured or systematic functional outcome measures. Thus, because of the substantial variability in study design and other methodological shortcomings, it is difficult to draw firm conclusions about the independent effective elements of EMDR.

EMDR appears to be effective in ameliorating symptoms of both acute and chronic PTSD. For example, Marcus (382) compared EMDR to standard care for 67 demographically diverse patients at a health maintenance organization who had developed PTSD after assault, rape, incest, accidents, or witnessing of a trauma. Subjects were randomly assigned to a treatment condition, but evaluations were not conducted in a fully blinded fashion, and standard care differed from therapist to therapist. Treatment sessions continued until PTSD symptoms had remitted or until the end of the study, at which point 75% of the EMDR-treated subjects and 50% of subjects who received standard care no longer met the criteria for PTSD. Significant improvements, which were more rapid in the EMDR-treated group, were also noted in PTSD symptoms as measured by the Mississippi PTSD Rating Scale and the Impact of Event Scale as well as in depressive symptoms as measured by the Beck Depression Inventory.

Rothbaum (383) randomly assigned 20 female rape victims either to three weekly 90-minute sessions of EMDR or to a waiting-list control group. The subjects all met the DSM-III-R criteria for PTSD, and most had had symptoms for years. At 4 weeks after the completion of treatment, 90% of the EMDR-treated subjects no longer met the criteria for PTSD. Unblinded symptom ratings for PTSD and depression showed significant improvements, although the duration of these benefits was unclear, since the waiting-list subjects were subsequently treated.

Scheck et al. (384) randomly assigned women (ages 16–25 years) with a self-reported traumatic memory to either EMDR or active listening, which was delivered in two 90-minute sessions

I week apart. Although immediately after the intervention both groups showed improvements on measures of depression and anxiety, including symptoms of PTSD, greater effect sizes were noted for the EMDR group. However, the study was limited by the fact that only one-half of those eligible to participate enrolled in the study, and of those who enrolled, only 70% completed the study. In addition, only 77% of subjects met the criteria for a diagnosis of PTSD at study entry.

Wilson et al. (385) randomly assigned 80 subjects to receive either EMDR or delayed treatment with EMDR. Subjects included equal numbers of men and women who had experienced a variety of traumas that occurred from 3 months to 54 years before treatment. Only one-half of the subjects met the DSM-IV criteria for PTSD, and only one-third of the sample had not received previous therapy for their symptoms. EMDR treatment consisted of three 90-minute sessions, and follow-up assessments were conducted. The subjects who received delayed treatment showed no change in symptoms in the 30 days before EMDR was begun, whereas the subjects who received EMDR showed significant improvements on measures of PTSD symptoms, somatization, interpersonal sensitivity, depression, and anxiety. Similar improvements were seen in the delayed-treatment EMDR group after treatment initiation, with improvements in both groups maintained at 90-day follow-up and again at 15-month follow-up (386).

Ironson et al. (387) compared the efficacy of EMDR and prolonged exposure in 22 civilian patients. Both approaches produced a significant reduction in PTSD and depression symptoms that was maintained at a 3-month follow-up. Successful treatment was faster, better tolerated, and more complete in the EMDR group (387). EMDR also resulted in reduced anxiety on process measures that was disproportionate to overall symptom improvement on outcome measures, with some evidence for sustainable symptom improvement for up to 3 months.

One study with a more extended follow-up period found that treatment gains were lost by 6 months (388). In this EMDR dismantling study, 51 Australian male combat veterans with PTSD were assigned to one of three conditions. Subjects were assigned to groups that received two sessions of EMDR, two sessions of reactive eye dilation desensitization and reprocessing (REDDR), or no intervention. REDDR was the same method as EMDR, except "eye movement" was replaced by "eye dilation," and a black box with a flashing light (opticator) was substituted for the eye movement stimuli. All subjects continued to receive standard care. No statistically significant changes were found from pre- to posttreatment on any of the outcome measures for the three conditions. At 3 months, all three treatment groups had improved somewhat, but there was no statistically significant difference among them. By 6 months, changes from pretreatment were no longer statistically significant for trait anxiety, depression, or PTSD (effect sizes at 6-month follow-up for EMDR plus standard care versus REDDR plus standard care=0.25). However, these findings must be interpreted in light of the brevity of both the EMDR and REDDR conditions. In a 5-year follow-up that compared 13 Vietnam combat veterans who received EMDR to a demographically matched control group of 14 combat veterans with PTSD who did not receive EMDR, both groups showed an overall worsening of PTSD symptoms over the 5-year period and loss of the modest to moderate early benefit of EMDR (389).

In another study, Devilly and Spence (35) compared nine sessions of a cognitive behavior therapy variant with up to eight sessions of EMDR in a total of 23 subjects with mixed trauma histories. The trauma treatment protocol (TTP) used prolonged exposure, in-depth cognitive therapy, and a variant of Foa's stress inoculation training. Compared to EMDR, TTP was more effective from pre- to posttreatment and had a reasonable effect size and high power. TTP's superiority became more pronounced at 3-month follow-up, at which time 83% of the TTP patients no longer met the PTSD criteria, compared to 36% of the EMDR subjects. However, in interpreting these data, it should be noted that the study was not randomized in a conventional manner, as most of the non-EMDR subjects were grouped in an initial block and EMDR was administered in a second block.

Cusack and Spates (390) randomly assigned 38 subjects to three 90-minute sessions of either standard EMDR or eye movement desensitization, which included all components of EMDR ex-

cept the cognitive reprocessing elements. Of the 27 individuals (23 women and four men) who completed the study, two-thirds had met the criteria for a DSM-IV diagnosis of PTSD at study entry and half had experienced either a physical or sexual assault. At posttreatment, both groups showed statistically significant decreases in symptoms as measured by the revised SCL-90, the Impact of Event Scale, the Structured Interview for PTSD, a behavioral assessment of speech anxiety, and a subjective unit-of-discomfort scale. However, both treatment groups showed comparable levels of improvement, suggesting that the imaginal exposure component of EMDR and not the cognitive reprocessing element is important to clinical efficacy.

Meta-analyses of the various controlled trials have generally concluded that EMDR represents an effective treatment. A 1997 review by Foa and Meadows (190) included studies of persons exposed to highly stressful events as well as those who met the criteria for PTSD. Many of the reviewed studies indicated no difference between EMDR and no-treatment or waiting-list control conditions, but one study indicated superiority of EMDR. The authors noted that because of methodological problems, further research to determine effectiveness was needed. Davidson and Parker (194) compared EMDR with no treatment, cognitive behavior therapy, exposure approaches (not involving in vivo exposure), variants of EMDR (e.g., dismantling studies), and "nonspecific" treatments. EMDR was more effective than no treatment and comparable to other active treatments. In this analysis, the dismantling studies appeared to provide comparable effectiveness across variant EMDR protocols. Maxfield and Hyer's meta-analysis (193) compared EMDR to waiting-list conditions, cognitive behavior therapy, and other treatments. EMDR was superior to the waiting-list conditions and either comparable or superior to other treatments (with considerable variability across studies). Although the meta-analysis by Shepherd et al. (191) included traumatized patients who did not all meet the DSM-IV or DSM-III-R criteria for PTSD, the researchers found that EMDR was comparable to a variety of psychotherapies and antidepressant therapy.

In summary, EMDR belongs within a continuum of exposure-related and cognitive behavior treatments. EMDR employs techniques that may give the patient more control over the exposure experience (since EMDR is less reliant on a verbal account) and provides techniques to regulate anxiety in the apprehensive circumstance of exposure treatment. Consequently, it may prove advantageous for patients who cannot tolerate prolonged exposure as well as for patients who have difficulty verbalizing their traumatic experiences. Comparisons of EMDR with other treatments in larger samples are needed to clarify such differences. The dismantling studies, in general, show no incremental effect from the use of eye movement or other proxies during the treatment sessions. Despite the demonstrable efficacy of EMDR, these studies call into question EMDR's theoretical rationale. It would therefore appear that it is the common sharing of trauma exposure techniques and emotional reprocessing that is principally responsible for treatment gains. Thus, EMDR is better than no treatment or supportive counseling and may be as effective as cognitive behavior therapy and other exposure-based techniques. As with the other therapies, the extent to which gains are maintained over the long term requires further evaluation.

2. Group psychotherapy for PTSD

There is a paucity of randomized, controlled treatment outcome studies for group treatment approaches among adults. The studies that have been done have not included groups that receive control or comparison treatments. Drawing conclusions across studies is difficult, since group protocols vary widely and include supportive therapy, psychoeducation, psychodynamic therapy, and various types of cognitive behavior therapy, including anxiety management, stress inoculation, assertiveness training, prolonged exposure, and cognitive restructuring. The patients treated in group psychotherapy studies have predominately been combat veterans and women with histories of childhood sexual abuse. Length of treatment has varied from 10 to 24 sessions that extend over 3 to 6 months. Some treatments have included booster sessions that extend over a year. Most studies have lacked sufficiently structured protocols, specific PTSD diagnostic assessments, and functional outcome measures.

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Of five randomized, controlled trials, one showed modest improvement (combining trauma-focused and present-focused group data) in 64 women who received supportive-expressive group therapy, compared to 61 women in a waiting-list condition, decades after the trauma occurred (391). In another randomized, controlled trial of individuals who experienced childhood trauma and abuse, group therapy as an adjunct to individual therapy produced a decrease in PTSD symptoms (392). Schnurr et al. (393), in a well-designed multisite study of combat veterans with chronic PTSD, used methods that blended efficacy and effectiveness designs and found modest effects of both trauma-focused and present-focused group therapy but no difference between the two treatments (although the dropout rate for the trauma-focused therapy was about twice that for the present-centered treatment). The higher dropout rate highlights a concern that exposure-based therapies—whether group or individual—may prove intolerable for some patients (394, 395). A randomized study of a two-stage group therapy for incarcerated women showed reductions in PTSD, mood, and interpersonal symptoms in subjects who received dialectical behavior therapy skills training and writing assignments, although participants were not all identified as having PTSD before study entry (396). The only randomized, controlled trial that involved more recent trauma investigated group treatment among Serbian concentration camp survivors within 3 months of release from the camps (397). At study entry, 44% of the 120 men in the study met the DSM-III-R criteria for PTSD and were randomly assigned to receive group therapy, group therapy plus medication (anxiolytics and tricyclic antidepressants, but no SSRIs), or medication alone over a 6-month treatment course. The study also followed subjects who refused treatment. Although there were significant differences between treated and untreated groups at 6 months (with a much greater percentage of resolution of PTSD among the treated subjects), a 3-year follow-up among randomly selected subjects revealed the paradoxical finding that the untreated group was improved, relative to the treatment groups, in scores on the Watson Questionnaire for PTSD.

Of the six nonrandomized studies, four related to treatment of women with histories of childhood sexual abuse (180, 398–400), one was a structured inpatient group treatment of Gulf War veterans (401), and one targeted adults after the traumatic loss of an adolescent or young adult child (402). In three of the four group interventions for individuals who had experienced childhood sexual abuse, no measurements of PTSD were used. Group interventions were associated with improvement in various global symptom measures, including measures of self-concept and social adjustment. The one study that examined effects of a psychoeducation group for multiply traumatized women reported mixed and conflicting outcome findings regarding PTSD. Thus, these studies do not provide sufficient strength, in methods or outcomes, to adequately judge the usefulness of group interventions with adults who have been sexually abused in childhood.

The British Gulf War veteran group study, which examined a treatment format that was markedly different from other group interventions, provided an intensive 12-day structured inpatient group therapy, with day-group follow-up sessions for 1 year (401). The intervention included some form of ongoing psychological debriefing. There was a robust decrease in the percentage of patients who met the criteria for PTSD (from 100% to 14.7%) 1 year posttreatment. It is noteworthy that there was no reported use of drugs of abuse or increased alcohol use during the follow-up period. These findings suggest that an intensive, structured 2-week group intervention with extended booster follow-up sessions may provide a useful modality for treatment of combat-related PTSD.

The only group intervention study for traumatic bereavement in adults combined problem solving and emotional support over 12 weeks and found that mothers improved somewhat in PTSD-related reactions, while fathers worsened (402). Those with lower levels of initial PTSD symptoms worsened, while there was mild improvement among those with higher levels of initial PTSD symptoms. This study strongly points to the need for caution in selecting group membership, even among spouses, where there may be varying degrees of exposure and pretreatment levels of PTSD symptoms.

An additional nonrandomized comparison study compared two cognitive behavior approaches—stress inoculation and assertiveness training—to supportive group therapy in a group of 24 rape victims (180). Relative to 13 subjects in a waiting-list control group, all three treatments, each of which included six 2-hour sessions, did equally well in producing moderate improvements in PTSD, depression, anxiety, and self-esteem. In addition, in the active treatment groups, therapeutic benefits were maintained at 3- and 6-month follow-up.

As discussed by Foy et al. (403), the supportive groups tend to place primary emphasis on addressing current life issues, while psychodynamic or cognitive behavior-oriented groups are primarily "trauma-focused," with major work directed at specific traumatic experiences and memories. In group psychotherapy, there is the advantage of being able to provide services to large numbers of individuals in response to a shared traumatic experience or because of shared PTSD symptoms. In regard to trauma-focused group psychotherapy, most of the evidence for efficacy and effectiveness is in the treatment of children and adolescents (304, 404–407). In a study of adults, Schnurr et al. (393) randomly assigned 360 combat veterans into groups of six and compared trauma-focused group therapy in 30 weekly sessions followed by five monthly boosters to a present-centered comparison treatment. Relative to baseline, significant improvements were noted on posttreatment measures of PTSD severity in both groups, but intent-to-treat analysis showed no differences between therapy groups on any outcome measure. These studies together provide evidence that group sessions in conjunction with assigned homework can achieve sufficient prolonged trauma-focused exposure to be a bona fide treatment approach.

The trauma-focused group psychotherapies just described typically share certain principles. The first sessions provide general psychoeducation regarding PTSD, coping skills for trauma reminders and posttraumatic stress reactions, and either anxiety-regulating or emotion-regulating techniques. They also provide group process exercises to improve group cohesion, openness, and tolerance. The trauma exposure sessions utilize different versions of prolonged narrative or imaginal exposure, moving from more general accounts to the most intense traumatic moments. They rely on group members' assisting each other in this difficult task. These sessions are generally followed by problem-solving sessions that address avoidant and aggressive behavior, secondary or current adversities, and developmental hindrances. Group studies would suggest that the group format is especially effective in addressing this latter group of functional impairments.

There are as yet no clear guidelines regarding the contribution of group process to group psychotherapy outcomes in PTSD. Davies et al. (408) provided general guidelines that will need to be specifically adapted for this work. In a study that has important implications for group process, Cloitre and Koenen (398) examined the effects of interpersonal therapy groups for women who had experienced childhood sexual abuse. In mixed groups that included at least one individual with a diagnosis of borderline personality disorder, the group therapy process was no different from a waiting-list control group in symptom diminution but did induce a significant increase in posttreatment anger. In contrast, in groups that did not contain patients with borderline personality disorder, there were significant reductions in anger, depression, and symptoms of PTSD. Thus, the study results raise caution about the diagnostic composition of interpersonal therapy groups.

3. Other early psychosocial intervention strategies

There is substantial evidence that single-session, individual psychological debriefing in the immediate aftermath of a broad range of traumatic exposures (e.g., motor vehicle crashes, combat, physical assaults, burn injury) does not reduce psychological distress or prevent the onset of chronic PTSD (128–130). A series of randomized, controlled trials have assessed the efficacy of debriefing across trauma-exposed populations (213, 217–219, 409). Bisson et al. (217) randomly assigned 43 hospitalized burn survivors to 30–120 minutes of single-session debriefing versus control conditions 2–19 days after traumatic injury. Sixteen percent of the debriefed group versus 9% of the intervention group had PTSD at 13-month follow-up, a difference that

was statistically significant. It is noteworthy that the subjects who were randomly assigned to debriefing had significantly greater injury severity and had more frequent involvement of others in the injury event. Carlier et al. (410) debriefed police officers within 24 hours after exposure to a variety of traumas and found no symptomatic improvement in debriefed subjects, compared with control subjects. Conlon et al. (213) performed a 30-minute debriefing with motor vehicle crash victims and found no PTSD symptom improvement in the intervention group, compared with control subjects who received an advice leaflet and follow-up telephone contact number. Hobbs et al. (218) performed a 1-hour critical incident stress debriefing in randomly selected, symptomatic subjects 24-48 hours after motor vehicle crashes. Patients who received the debriefing demonstrated either similar or worsened symptomatic outcomes, compared to control subjects at 4 months (218) and 36 months (219) posttrauma. Rose et al. (409) delivered a 1-hour critical incident stress debriefing to victims of violent crime within 1 month after the trauma and found no significant differences in PTSD symptoms in intervention patients, relative to control subjects, at the 11-month follow-up assessment.

A handful of randomized and open trials of debriefing suggest limited benefit of group debriefing. In an open trial, Shalev et al. (20) performed group debriefings (emphasizing clarification of individuals' roles, time sequences, and facts surrounding the traumatic event, without exploring emotions) with soldiers 48-72 hours after exposure to combat and found reductions in anxiety, improvement in self-efficacy, and increased homogeneity of the group immediately after the debriefing. Deahl et al. (411) randomly assigned soldiers to a postdeployment debriefing/predeployment stress prevention intervention or to predeployment stress intervention alone. Although PTSD symptoms across the two groups showed no significant differences at 6- and 12-month follow-up, there was evidence of significantly reduced alcohol use in soldiers who received the debriefing. Campfield and Hills (412) randomly assigned robbery victims to immediate (<10 hour) versus delayed (>48 hour) critical incident stress debriefing group conditions. Victims in the immediate debriefing condition demonstrated improved symptom outcomes

2 weeks after the debriefing.

Although the debriefing models that have been investigated generally do not appear to be efficacious, there is only preliminary evidence that other psychosocial interventions with established efficacy for the treatment of PTSD can be effectively delivered as early interventions in complex real-world settings such as postdisaster environments and acute care medical settings. One study suggests that cognitive behavior interventions can be effectively delivered after mass attack, although the number of treatment sessions may need to be extended and high-risk groups of trauma survivors such as the physically injured may be less responsive (380). Preliminary evidence suggests that early psychosocial intervention strategies such as in-person/telephone case management may be effective in both engaging trauma survivors in treatment and reducing acute distress (131–134). Gidron et al. (133) randomly assigned 17 patients who had had motor vehicle crash injuries and elevated heart rates during acute care to receive a telephone-based memory restructuring intervention or a supportive listening control intervention within several days of the accident. Patients who received the active telephone-based intervention demonstrated significantly decreased PTSD symptoms. Zatzick et al. (134) delivered a collaborative care intervention that included posttraumatic concern elicitation and support to 34 randomly selected survivors of intentional and unintentional injuries. At 1-month postinjury, the patients receiving the intervention had significantly diminished PTSD and depressive symptoms, compared with control patients, yet treatment gains were not maintained at the 4month assessment. In a follow-up randomized effectiveness trial with 120 injured trauma survivors, Zatzick et al. (131) extended the stepped care procedure to include case management and evidence-based cognitive behavior therapy and medication treatment targeting PTSD. Compared with control subjects who received usual care, patients who received the combined intervention demonstrated modest and statistically significant prevention of PTSD, which coincided with the initiation of the evidence-based treatments. In a nonrandomized design, Bordow and Porritt (132) delivered a case management intervention to 70 male motor vehicle crash survivors. Intervention patients demonstrated less symptomatic distress than control groups of patients who received no intervention, immediate intervention, or delayed contact 3 months after the injury. More research is needed to determine if these initial engagement interventions will require augmentation with other proven psychotherapeutic and psychopharmacological interventions to prevent development of chronic PTSD.

4. Other psychotherapies

New psychotherapeutic approaches continue to be developed and applied to the treatment of trauma survivors with PTSD. As with previously developed interventions, it is essential that initial small-scale trials be followed by larger-scale randomized, controlled trials to establish efficacy. Recent small-scale trials of Internet-based therapies (413, 414) and Outward Bound group recreational therapies (415, 416) suggest potential beneficial effects on symptoms and functional outcomes.

In a pilot study, Gidron et al. (417) assessed the effects of written emotional disclosure on mental and physical health in Israeli patients with PTSD. One to 3 years after their trauma, subjects were randomly assigned either to the disclosure condition or to a casual writing control condition. Disclosure condition patients were asked to write for 20 minutes for 3 consecutive days about their most traumatic experiences and then, in a brief structured format, to talk about the most severe events about which they had written. Control subjects wrote about their daily agenda without affective content and then discussed one daily activity. The investigators found that a brief return to traumatic narrative may be counterproductive. Disclosure patients reported higher levels of negative affect immediately after writing than did the control patients and also reported larger increases in avoidance symptoms. The proportion of emotional words in the trauma narratives was associated with intrusive and avoidance symptoms of PTSD. The proportion of words on physical health predicted a greater number of health care visits at follow-up.

Monitoring of intrusions has also been suggested as a treatment intervention (418) and was studied in six individuals, all with PTSD. The subjects were given instructions to monitor intrusions—e.g., "try to not think of it," "think your way through," "cope with it"—over a 2-month period; then they were followed up immediately thereafter and again 3 months afterward. Of the six individuals treated with this approach, only one still met the criteria for PTSD at the end of the study, whereas four recovered. Although the small sample size limited the authors' ability to evaluate differences statistically, this innovative treatment of specific symptoms highlights future directions for possible public health interventions that may limit the need for specialty care.

With regard to novel techniques, a key question is whether they contain active components of efficacy-proven PTSD interventions. For instance, a review of case studies of Native American healing rituals that have been applied to the treatment of trauma survivors, such as sweat lodge and shamanic healing ceremonies, suggests that these interventions may contain an imaginal exposure component (419, 420). These "culturally sensitive" interventions may therefore combine "active" PTSD intervention components with socially accepted service delivery modalities that enhance adherence and reduce dropout.

C. PHARMACOTHERAPIES

1. Antidepressants

a) SSRIs

SSRIs are the most extensively studied medications in PTSD treatment research. Eight randomized, controlled trials have investigated SSRIs. These trials were often large, industry-sponsored clinical studies with hundreds of subjects. The general finding is that SSRIs are significantly

more effective than placebo. In a 12-week randomized, controlled trial of sertraline, Davidson et al. (421) randomly assigned 208 civilian men and women to receive either medication or placebo. Subjects treated with the SSRI were more likely to show a significant clinical response consisting of at least a 30% reduction in PTSD symptoms and were also more likely to experience a global improvement in symptoms (improvement was found in 60% of sertraline-treated subjects, compared to 38% of the placebo group subjects). In a similarly designed study, in which a separate sample of 187 civilian subjects were randomly assigned to receive sertraline or placebo, responder rates were 55% in the SSRI group, in contrast to 35% for the placebo subjects (144). Two randomized clinical trials with the SSRI paroxetine have also had favorable results. In one, 551 civilian men and women were randomly assigned to receive 20 mg/day of paroxetine (N=183), 40 mg/day of paroxetine (N=182), or placebo (N=186). Subjects in the two medication groups did not differ from one another but demonstrated significant improvement on all three PTSD symptom clusters, global improvement, and improvement in social and occupational functioning (141). In the second large paroxetine study, 307 civilian subjects were randomly assigned to receive medication or placebo with similar positive results; the medication group showed significantly greater improvement with regard to all three clusters of PTSD symptoms, global improvement, and improvement of functional capacity (e.g., in work, social interactions, and family life) (145). Unlike the sertraline results, which were positive for women but not for men (possibly because so few men participated in these trials), paroxetine was equally effective for men and women. As a result of these large-scale multisite randomized, clinical trials, SSRIs are currently considered first-line pharmacotherapeutic treatment for PTSD, and both sertraline and paroxetine have received the approval of the U.S. Food and Drug Administration as indicated treatments for PTSD. A randomized clinical trial with fluoxetine has also had favorable results. In this study, in which 301 mostly white, male non-American veterans of United Nations peacekeeping deployments were randomly assigned to receive medication or placebo, the SSRI subjects exhibited significantly greater improvement in PTSD symptom severity and global functioning than did the placebo group (146). Open-label trials with two other SSRIs—citalopram (278) and fluvoxamine (150)—were also promising.

A few long-term continuation and discontinuation studies with sertraline are also noteworthy. Fifty-five percent of patients who failed to respond positively to sertraline after 12 weeks of treatment did exhibit a favorable response when treatment was extended for an additional 24 weeks (279). Discontinuation of sertraline treatment in patients who had previously responded favorably was six times more likely to lead to clinical relapse than was continuation of sertraline treatment (123).

In addition to finding reduction of PTSD symptoms, studies with sertraline and fluoxetine have suggested that SSRI treatment also promotes improvement in functional status and quality of life and that discontinuation of medication is associated with decreased quality of life and functional measures in addition to symptom relapse (148, 280).

To summarize, in short- and intermediate-term trials, SSRIs have proven efficacy for PTSD symptoms and related functional problems. Patients who respond favorably will generally need to continue taking medication in order to maintain clinical gains.

b) Other second-generation antidepressants

Despite high utilization of second-generation antidepressants to treat depression and other anxiety disorders, no randomized, controlled trials of these medications have been carried out in patients with PTSD. The most extensively tested medication, nefazodone, might be expected to have a favorable effect on PTSD symptoms since, like the SSRIs, it promotes serotonergic activity. Indeed, several open-label trials with nefazodone suggest that this medication may have efficacy for treatment of all three PTSD symptom clusters, especially for patients with treatment-resistant symptoms (422-426). Nefazodone is also an attractive possibility because it is often better tolerated than SSRIs, although caution must be taken given its association with irreversible and life-threatening hepatic failure.

Positive reports from small open-label trials with trazodone (427), bupropion (428), venlafaxine (429), and mirtazapine (430) do not provide sufficient evidence to justify endorsing any of these medications for PTSD patients at this time; one double-blind, placebo-controlled pilot study with mirtazapine also suggests efficacy (431). Trazodone may have a unique niche in treatment because its serotonergic action is synergistic with SSRIs while its sedative properties are often an effective antidote to SSRI-induced insomnia, and sleep disturbance is often central to the clinical picture in ASD and PTSD. Efficacy of such agents in ASD or in acutely traumatized individuals who do not meet the full diagnostic criteria for PTSD warrants further investigation.

c) Tricyclic antidepressants

In three randomized, controlled trials conducted with tricyclic antidepressants, all subjects were Vietnam veterans seeking PTSD treatment in VA hospital settings. In a study in which 60 veterans in a VA setting were randomly assigned to receive the tricyclic antidepressant imipramine (N=23, mean dose=225 mg/day), the MAOI phenelzine (N=19, mean dose=68 mg/ day), or placebo (N=18), imipramine produced significantly more improvement than placebo but not as much as phenelzine (151) (see further details in the next section, Section V.C.1.d, "MAOIs"). In an 8-week trial in which 40 veterans in a VA setting were randomly assigned to receive either the tricyclic antidepressant amitriptyline (N=22, mean dose=169 mg/day) or placebo (N=18), the response rate was 47% for the patients who received amitriptyline, compared to 19% for placebo subjects; this difference was statistically significant (152). Taken together, both studies indicated that tricyclic antidepressant treatment produced global improvement and reduction of reexperiencing symptoms. It should be noted, however, that in the third published randomized, controlled trial, which included only 18 veterans randomly assigned to receive the tricyclic antidepressant desipramine (mean dose=165 mg/day) or placebo for 4 weeks, no response by either group was found (153). A quantitative analysis of all trials (randomized, controlled trials and open-label trials) with these medications indicated that tricyclic antidepressants in general produce global improvement and reductions in reexperiencing symptoms (432). Thus, although clinical management with tricyclic antidepressants may be more complicated than that with newer agents, the tricyclic antidepressants are effective medications that still have a potential role in PTSD treatment.

Robert et al. (433) compared imipramine with chloral hydrate as treatment in a randomized clinical trial. Twenty-five children, ages 2–19 years, with symptoms of ASD and hospitalized on a burn unit for severe injury (with a mean total burn surface area of 45%), received either imipramine (1 mg/kg, with a maximum dose of 100 mg/day) or chloral hydrate (25 mg/kg, with a maximum dose of 500 mg/day). After 7 days of treatment, ASD symptoms remitted in 83% of the patients treated with imipramine, compared with 38% of those treated with chloral hydrate. Stated differently, 10 of the 12 children who received imipramine were considered to have a positive treatment response. Unfortunately, there was no long-term follow-up, so it is unclear whether this early tricyclic antidepressant treatment prevented later development of PTSD. This study stands as the best demonstration that acute pharmacotherapy can be an effective treatment for acutely traumatized subjects.

d) MAOIs

Two randomized, controlled trials have been carried out with the MAOI phenelzine. In the 8-week study with American Vietnam veterans in a VA setting mentioned in the previous section, 60 subjects were randomly assigned to receive the MAOI phenelzine (N=19), the tricyclic anti-depressant imipramine (N=23), or placebo (N=18) (151). In assessments with the Impact of Event Scale, both medication groups did significantly better than the placebo group, with 44% improvement among the phenelzine subjects, compared with 25% improvement among the imipramine subjects. The difference between the MAOI and tricyclic antidepressant groups was statistically significant (151). A single report of a successful open trial of the reversible

monoamine oxidase type A inhibitor moclobemide (434) also supported the use of MAOIs as a class in treatment of PTSD. Moclobemide, which is not presently available in the United States, was tested in a 12-week open trial with 20 subjects and yielded promising results (434). At the end of the trial, 11 subjects no longer met the PTSD diagnostic criteria, and there was a significant reduction in PTSD symptom severity and significant improvement in global function. In addition to studies with phenelzine, two randomized, controlled trials that used brofaromine, a unique MAOI/SSRI medication that is not available commercially, showed some improvement in PTSD symptoms (155, 435). Finally, there are two reports of meta-analyses that synthesized results from a number of published reports (432, 436). Although there have been some negative reports, MAOIs have generally been shown to produce global clinical improvement and reductions in PTSD symptom severity, with specific effectiveness for reexperiencing symptoms. In the only head-to-head comparison of an MAOI (phenelzine) and a tricyclic antidepressant (imipramine), as noted earlier, the MAOI was more effective, although the tricyclic antidepressant was still more effective than placebo (151). Clinicians' reluctance to prescribe MAOIs generally relates to concerns about the capacity of patients to adhere to tyramine-free diets or to abstain from alcohol, certain drugs of abuse, and contraindicated prescription medications (e.g., SSRIs, CNS stimulants, decongestants, and meperidine). However, it must be emphasized that MAOIs are clinically effective and that many patients can adhere to such constraints. Finally, reversible monoamine oxidase type A inhibitors are much easier to manage clinically because patients need not observe such dietary or pharmacological restrictions.

2. Benzodiazepines

Benzodiazepines cannot be recommended as monotherapy for PTSD patients, despite their proven efficacy in generalized anxiety disorder. Despite widespread use in treatment of PTSD, their utility in PTSD has not been adequately evaluated. In the only pertinent randomized, controlled trial, alprazolam was tested with 10 civilians and veterans who received treatment for 5 weeks (437). The benzodiazepine was ineffective against PTSD reexperiencing and avoidant/numbing symptoms, although it did improve sleep and general anxiety. Rebound anxiety related to alprazolam treatment was also observed during this trial. In addition, a postdiscontinuation benzodiazepine withdrawal syndrome has been described that was characterized by a profound exacerbation of PTSD symptoms (158). Although a limited open-label case series also suggested improvement in insomnia and core PTSD symptoms in acutely traumatized individuals (438), positive long-term outcome data have not been reported, and a controlled study did not show advantage over placebo (156). Indeed, early administration of benzodiazepines was associated with a higher incidence of PTSD at 1- and 6-month follow-up in one study (157).

Miscellaneous medications

A variety of classes of psychopharmacological agents have been tested for the treatment of PTSD. Initial open and randomized trials of carbamazepine (160), valproic acid (161, 162), and lamotrigine (163) suggested that these agents may be efficacious in targeting discrete PTSD symptom clusters. Two small open-label trials showed promising results with the serotonergic anxiolytic buspirone (439, 440), but the data are insufficient to recommend it for use at this time. Two studies (169, 441) suggested that prazosin may be effective in treating nightmares and other PTSD symptoms in male combat veterans.

Olanzapine, a second-generation antipsychotic agent, when prescribed to augment ongoing sertraline treatment, was shown to produce improvement in PTSD, depressive, and sleep-related symptoms in Vietnam veterans (166). Open-label studies of adjunctive olanzapine and quetiapine have demonstrated symptom reduction in veterans with PTSD (165, 167). However, olanzapine alone did not show an effect in a small randomized, double-blind, placebocontrolled trial in female veterans (164). A small controlled study of risperidone in chronic combat-related PTSD was similarly disappointing for core PTSD symptoms, although reexperiencing and global psychotic symptoms were reduced (168).

Early case reports suggested that cyproheptadine, a serotonin antagonist, might ameliorate PTSD flashbacks and traumatic nightmares, but a randomized, controlled trial by Jacobs-Rebhun et al. (442) and a large open-label trial by Clark et al. (443) disconfirmed these findings. In the randomized trial by Jacobs-Rebhun et al., 69 veteran subjects in a VA setting were randomly assigned to receive either cyproheptadine or placebo. After 2 weeks of treatment, the cyproheptadine subjects exhibited a (nonsignificant) worsening of PTSD symptom severity, sleep quality, and traumatic nightmare severity (442). The large open-label trial of cyproheptadine by Clark et al. (443) also failed to produce positive results. Therefore, cyproheptadine cannot be recommended for PTSD treatment.

Inositol is a second messenger with limited evidence supporting efficacy in treating depression and panic disorder. However, in a small randomized, crossover study, with 13 subjects randomly assigned to receive medication or placebo for a 4-week trial, inositol was ineffective in alleviating PTSD symptom severity (444).

A number of agents have been pilot tested in the secondary prevention of PTSD. There is preliminary evidence from two studies that steroid administration during inpatient medical/surgical hospitalization may diminish PTSD symptom development in patients with critical medical illness (445, 446). One observational study among youths hospitalized after burn injury suggested that patients who received the highest doses of opiate analgesics exhibited the lowest PTSD symptom severity after discharge from the hospital (447). As mentioned previously, another randomized investigation on a pediatric burn unit suggested that imipramine is efficacious in ameliorating ASD (433). A single investigation pilot tested the use of propranolol among injured patients seen in an emergency department after a motor vehicle accident and had interesting findings; although no significant improvement in PTSD was detected and high dropout rates were observed in the intervention group, subjects who received propranolol had a significant reduction in physiological reactivity that persisted for 3 months after acute treatment (137). In addition, a recent controlled but nonblind, nonrandomized study reported that acute administration of propranolol posttrauma reduced subsequent PTSD symptoms (172). These findings will also be important to pursue further in larger randomized trials.

PART C

FUTURE RESEARCH NEEDS

Research over the past decade has led to considerable advances in our understanding of the epidemiology of the acute and long-term neurobiological and psychological changes that occur after highly stressful experiences. Research has also identified a variety of treatment approaches for pathological responses to traumatic events, including ASD and PTSD. Although much has been accomplished, future study is required to expand current understanding and inform future assessment, prevention, and treatment strategies. The following future research needs are not presented in any effort to prioritize, nor are they intended to be exhaustive. They serve to illustrate the fact that our understanding of the range of human response to traumatic stress is in its infancy and only beginning to evolve.

• Early interventions/posttrauma treatment. In early intervention (in the hours or days after a traumatic event), the aim is to reduce immediate distress, but ideally it might also be to prevent the development of ASD or PTSD. However, relatively little is known about prevention. Small, controlled studies of psychotherapy suggest efficacy (135, 136, 448),

as do the studies of early case management interventions (131–134). In addition, a few small controlled studies suggest that early pharmacological interventions may reduce development of posttraumatic symptoms (137, 172, 433). However, larger controlled trials and long-term follow-up studies are needed to fully address the efficacy and effectiveness of psychotherapeutic, psychopharmacological, psychoeducational, and supportive interventions in reducing initial distress and later development of ASD or PTSD, as well as in improving social and occupational functioning.

- Identification of risk factors for development of ASD or PTSD. Given the wide variability of human response to traumatic events, future intervention strategies would be aided by a greater understanding of the extent to which ASD or other diagnoses or factors are associated with subsequent development of PTSD. Elucidation of markers or risk factors (e.g., biological or genetic markers, psychological traits, other life experiences, or ethnocultural variables) that specifically relate to the development or severity of ASD or PTSD after initial or subsequent exposures to potentially traumatic events would be valuable (179, 449, 450). Neurobiological markers are being identified, for example, that are associated with reduced susceptibility to developing disorders after exposure (or exposures) to potentially traumatic events (451). Further study of markers for both vulnerability and resilience may help explain variability in the development of ASD or PTSD within populations exposed to similar traumatic events and may contribute to a better understanding of the natural history of these conditions. Better identification of at-risk populations within groups similarly exposed may also guide future preventive and acute intervention strategies. In addition to the independent effects of specific markers or risk factors, interactions among identified biological, psychological, and social factors may further alter the likelihood of developing ASD or PTSD and also merit additional study.
 - Subthreshold and complex PTSD. Persons may develop significant symptoms in one or more of the three ASD or PTSD symptom clusters but not meet the full diagnostic criteria for ASD or PTSD (452–454). These individuals may be significantly impaired (452, 455), raising questions about the appropriateness of current threshold criteria for PTSD. Similar questions may be raised about the current DSM-IV-TR criterion that to be considered traumatic, a person's response to an event must include "intense fear, helplessness, or horror," since this criterion excludes many persons who report feeling numb or who demonstrate dissociative responses (19). Further study is needed to determine whether such individuals, who might otherwise qualify for these diagnoses, would benefit from treatment.

Randomized, controlled trials of therapy and medications have focused on reducing readily identifiable core symptoms that are outlined in the current diagnostic criteria for PTSD; these symptoms lend themselves to quantification with available severity scales. Clinicians recognize that PTSD and ASD are associated with changes in belief systems, view of self, and ability to trust others, as well as related changes in social, occupational, and interpersonal functioning that may affect patients' lives to a far greater extent than more readily quantifiable core clinical features. The extent to which these issues rather than the more easily recognized or reliably reported reexperiencing phenomena or hyperarousal represent the more disabling aspects of the illnesses also bears further investigation. Another question for further study is whether these often-observed changes represent symptoms that should be included in refined diagnostic criteria for PTSD or should signify a separate diagnostic entity (e.g., occurring perhaps as a consequence of earlier or repeated exposure to trauma). More difficult to assess is the extent to which deterioration in spheres of functioning is mitigated by currently available treatments and which approaches may be most effective for addressing the illnesses' effects on functioning.

Whether or not traumatic grief and complicated bereavement should be recognized as separate diagnostic entities, response to loss is often a focus for persons seeking treatment (303, 456). Since traumatic loss is common, further study of potential treatments for prolonged or disabling grief is warranted.

Medication treatments and psychotherapies. For the most part, studies of psychotherapy
and medication treatment for ASD and PTSD have been small and of relatively brief
duration. While larger, well-controlled studies of SSRIs have been conducted, similar
studies are lacking and are needed for virtually all other available medication treatments.
Newer medications such as tiagabine (457) have been pilot tested but will also require
larger-scale controlled studies to establish efficacy.

Benzodiazepines are a widely used and effective treatment for other psychiatric disorders, including anxiety disorders. Although they may improve sleep in ASD or PTSD, some evidence suggests that benzodiazepines also may increase the likelihood of developing PTSD (157, 438). Given the widespread use and prescription of these medications in emergency settings, well-controlled studies are needed in patients with ASD and PTSD.

Studies of pharmacological treatments are also needed to provide evidence on stepwise or algorithmic approaches to treatment choice and to define the role of adjunctive medications in patients with partial responses to first-line agents. Pharmacokinetic or pharmacodynamic properties of medications within subclasses have yet to be studied with regard to their effect on efficacy in treatment of PTSD, nor have the effects of ethnic or cultural considerations on treatment response been clearly delineated.

At the neurobiological level, the mechanisms by which specific medications alter putative disease processes remain unclear. Studies of the neurobiological effects of specific interventions may provide clues to the pathophysiology of these disorders and suggest other avenues of treatment.

Cognitive and behavioral therapies—particularly as early interventions—have demonstrated efficacy largely in victims of sexual assault, interpersonal violence, and industrial or vehicular accidents. Replication of these studies in combat veterans or other victims of mass violence is also important. Preliminary findings with innovative psychotherapies (368, 413, 415–417) require further study in larger controlled trials. Manualizing both emerging and traditional psychotherapies is one approach that may promote more rigorous study. Given the widespread use of psychodynamic psychotherapy, it is particularly important to encourage controlled studies to examine the techniques used and their efficacy.

In the clinical setting, psychotherapeutic approaches are most often used in combination with one another. Regardless of theoretical orientation, clinicians use elements of psychodynamic therapy, supportive therapy, cognitive behavior therapy, or other approaches incorporating various degrees of imaginal or in vivo exposure. Identification of the effective subcomponents of various cognitive and behavior therapies and EMDR in the research setting has not been accomplished, and even less is known about effective subcomponents of these therapies in typical clinical populations. Investigations of combinations of various psychotherapies are few (177, 397, 458). Effectiveness trials that assess whether efficacious psychotherapeutic and psychopharmacological interventions can be adapted beneficially to typical clinical settings are similarly necessary (25).

• Treatment of specific symptoms or clinical concerns. Given mixed results with benzodiazepines and the prominence of sleep disturbance in traumatized individuals (459– 461), it is critical to identify medications or therapies that can target nightmares and insomnia without increasing the patient's likelihood of developing other symptoms (426, 462). Further study may also help to identify particular interventions that reduce other specific symptoms in patients with ASD or PTSD, such as self-injurious, deliberately selfharmful, or suicidal behaviors (277). The role of active involvement of family members and community supports in enhancing adherence—as has been applied to other severe mental disorders—requires further exploration (84). There are few studies of the potential of family or couples therapy for reducing symptoms or dysfunction in PTSD (372). The effect of other treatments on reducing functional impairment is another broad area that requires further investigation. • Generalization of research trials to clinical populations. As for most disorders, the generalizability of medication trials and therapy studies for the treatment of ASD and PTSD is frequently limited by high levels of subject exclusion because of comorbidity, high subject dropout rates, and relatively short durations of follow-up periods (277). Consequently, the robust treatment responses observed in research settings may not always be seen in typical patients treated in clinical practice. Longer-term follow-up studies must also be conducted to determine whether initial gains made in therapy or with medication are long-lasting and whether maintenance treatment is necessary. More studies are needed to clarify potential adverse effects of treatment and patient factors that reduce adherence to specific regimens (463). Effectiveness trials are also necessary to assess whether efficacious therapeutic and/or psychopharmacological interventions for ASD or PTSD can produce meaningful and lasting changes in patients who typically present in community settings. The importance of PTSD as a comorbid disorder in serious and persistent mental disorders such as schizophrenia or bipolar disorder highlights a particular need for study of PTSD treatment in these patient groups.

The fact that stressful life events may cause emotional and behavioral effects has long been recognized. Psychiatrists concerned themselves with the consequences of traumatic experience decades before the diagnoses of ASD and PTSD were specifically identified. Clinical experience, descriptive literature, and case study guided treatment of persons suffering from the effects of traumatic exposure long before randomized, controlled trials were conceptualized or became a standard for evaluating new evidence. Disregarding clinical experience accumulated before these advances in research design would be as imprudent as believing that research conducted under current standards has adequately demonstrated the full range of effective treatment. Standards for gathering and evaluating new evidence are evolving and should inform the development of future guidelines for assessing and treating mental disorders that arise in the aftermath of exposure to traumatic events.

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American Association of Community Psychiatrists
American Group Psychotherapy Association
American Music Therapy Association
American Nurses Association
International Federation of Psychoanalytic Societies
National Association of Social Workers
National Center for Post-Traumatic Stress Disorder

REFERENCES

The following coding system is used to indicate the nature of the supporting evidence in the references:

- [A] Randomized double-blind clinical trial. A study of an intervention in which subjects are prospectively followed over time; there are treatment and control groups; subjects are randomly assigned to the two groups; both the subjects and the investigators are blind to the assignments.
- [A-] Randomized clinical trial. Same as above but not double-blind.
- [B] Clinical trial. A prospective study in which an intervention is made and the results of that intervention are tracked longitudinally; study does not meet standards for a randomized clinical trial.
- [C] Cohort or longitudinal study. A study in which subjects are prospectively followed over time without any specific intervention.
- [D] Control study. A study in which a group of patients and a group of control subjects are identified in the present and information about them is pursued retrospectively or backward in time.
- [E] Review with secondary data analysis. A structured analytic review of existing data, e.g., a meta-analysis or a decision analysis.
- [F] Review. A qualitative review and discussion of previously published literature without a quantitative synthesis of the data.
- [G] Other. Textbooks, expert opinion, case reports, and other reports not included above.

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- Veterans Health Administration: Management of Post-Traumatic Stress. Office of Quality and Performance publication 10Q-CPG/PTSD-04. Washington, DC, VA/DoD Clinical Practice Guideline Working Group, Veterans Health Administration, Department of Veterans Affairs and Health Affairs, Department of Defense, 2003. http://www.oqp.med.va.gov/cpg/ PTSD/PTSD_Base.htm [G]
- Foa EB, Keane TM, Friedman MJ (eds): Effective Treatments for PTSD: Practice Guidelines
 From the International Society for Traumatic Stress Studies. New York, Guilford, 2000 [G]
- American Academy of Child and Adolescent Psychiatry: Summary of the Practice Parameters for the Assessment and Treatment of Children and Adolescents With Posttraumatic Stress Disorder. J Am Acad Child Adolesc Psychiatry 1998; 37:997–1001 [G]
- Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB: Posttraumatic stress disorder in the National Comorbidity Survey. Arch Gen Psychiatry 1995; 52:1048–1060 [G]
- Breslau N, Kessler RC, Chilcoat HD, Schultz LR, Davis GC, Andreski P: Trauma and posttraumatic stress disorder in the community: the 1996 Detroit Area Survey of Trauma. Arch Gen Psychiatry 1998; 55:626–632 [G]
- Harvey AG, Bryant RA: The relationship between acute stress disorder and posttraumatic stress disorder: a 2-year prospective evaluation. J Consult Clin Psychol 1999; 67:985–988 [C]
- 7. Marshall RD, Spitzer R, Liebowitz MR: Review and critique of the new DSM-IV diagnosis of acute stress disorder. Am J Psychiatry 1999; 156:1677–1685 [F]
- 8. Staab JP, Grieger TA, Fullerton CS, Ursano RJ: Acute stress disorder, subsequent posttraumatic stress disorder and depression after a series of typhoons. Anxiety 1996; 2:219–225 [G]
- 9. Bryant RA, Harvey AG, Guthrie RM, Moulds ML: A prospective study of psychophysiological arousal, acute stress disorder, and posttraumatic stress disorder. J Abnorm Psychol 2000; 109:341–344 [C]
- Stein MB, McQuaid JR, Pedrelli P, Lenox R, McCahill ME: Posttraumatic stress disorder in the primary care medical setting. Gen Hosp Psychiatry 2000; 22:261–269 [G]
- 11. Silver RC, Holman EA, McIntosh DN, Poulin M, Gil-Rivas V: Nationwide longitudinal study of psychological responses to September 11. JAMA 2002; 288:1235–1244 [G]
- 12. North CS, Nixon SJ, Shariat S, Mallonee S, McMillen JC, Spitznagel EL, Smith EM: Psychiatric disorders among survivors of the Oklahoma City bombing. JAMA 1999; 282:755–762 [G]
- 13. Weine SM, Becker DF, McGlashan TH, Laub D, Lazrove S, Vojvoda D, Hyman L: Psychiatric consequences of "ethnic cleansing": clinical assessments and trauma testimonies of newly resettled Bosnian refugees. Am J Psychiatry 1995; 152:536–542 [G]
- Bromet E, Sonnega A, Kessler RC: Risk factors for DSM-III-R posttraumatic stress disorder: findings from the National Comorbidity Survey. Am J Epidemiol 1998; 147:353–361 [G]
- Breslau N, Davis GC, Andreski P, Peterson EL, Schultz LR: Sex differences in posttraumatic stress disorder. Arch Gen Psychiatry 1997; 54:1044–1048 [G]
- 16. Meltzer-Brody S, Churchill E, Davidson JR: Derivation of the SPAN, a brief diagnostic screening test for post-traumatic stress disorder. Psychiatry Res 1999; 88:63–70 [G]
- 17. Breslau N, Peterson EL, Kessler RC, Schultz LR: Short screening scale for DSM-IV posttraumatic stress disorder. Am J Psychiatry 1999; 156:908–911 [G]
- 18. Brewin CR, Rose S, Andrews B, Green J, Tata P, McEvedy C, Turner S, Foa EB: Brief screening instrument for post-traumatic stress disorder. Br J Psychiatry 2002; 181:158–162 [G]
- 19. Shalev AY: Acute stress reactions in adults. Biol Psychiatry 2002; 51:532–543 [F]
- Shalev AY, Peri T, Rogel-Fuchs Y, Ursano RJ, Marlowe D: Historical group debriefing after combat exposure. Mil Med 1998; 163:494–498 [B]
- Marmar CR, Weiss DS, Pynoos RS: Dynamic psychotherapy of post-traumatic stress disorder, in Neurobiological and Clinical Consequences of Stress: From Normal Adaptation to Post-Traumatic Stress Disorder. Edited by Friedman MJ, Charney DS, Deutch AY. Philadelphia, Lippincott-Raven, 1995, pp 495–506 [F]

- 22. Marmar CR, Weiss DS, Schlenger WE, Fairbank JA, Jordan BK, Kulka RA, Hough RL: Peritraumatic dissociation and posttraumatic stress in male Vietnam theater veterans. Am J Psychiatry 1994; 151:902–907 [G]
- 23. Weathers FW, Litz BT, Huska J, Keane TM: PTSD Checklist (PCL) for DSM-IV. Boston, National Center for PTSD, Behavioral Science Division, 1994 [G]
- Horowitz M, Wilner N, Alvarez W: Impact of Event Scale: a measure of subjective stress. Psychosom Med 1979; 41:209–218 [G]
- 25. Horowitz MJ: Treatment of Stress Response Syndromes. Arlington, VA, American Psychiatric Publishing, 2003 [G]
- Davidson JR, Book SW, Colket JT, Tupler LA, Roth S, David D, Hertzberg M, Mellman T, Beckham JC, Smith RD, Davison RM, Katz R, Feldman ME: Assessment of a new selfrating scale for post-traumatic stress disorder. Psychol Med 1997; 27:153–160 [G]
- 27. Weathers FW, Keane TM, Davidson JR: Clinician-Administered PTSD Scale: a review of the first ten years of research. Depress Anxiety 2001; 13:132–156 [E]
- 28. Davidson JR, Malik MA, Travers J: Structured Interview for PTSD (SIP): psychometric validation for DSM-IV criteria. Depress Anxiety 1997; 5:127–129 [G]
- 29. American Psychiatric Association: Practice Guideline for Psychiatric Evaluation of Adults, 2nd ed. Am J Psychiatry (in press) [G]
- 30. Connor KM, Butterfield MI: Posttraumatic stress disorder. Focus 2003; 1:247-262 [F]
- 31. Scurfield RM, Blank AS: A guide to obtaining a military history from Viet Nam veterans, in The Trauma of War: Stress and Recovery in Viet Nam Veterans. Edited by Sonnenberg SM, Blank AS Jr, Talbott JA. Washington, DC, American Psychiatric Press, 1985, pp 263–291 [G]
- 32. Ballenger JC, Davidson JR, Lecrubier Y, Nutt DJ, Foa EB, Kessler RC, McFarlane AC, Shalev AY: Consensus statement on posttraumatic stress disorder from the International Consensus Group on Depression and Anxiety. J Clin Psychiatry 2000; 61(suppl 5):60–66 [G]
- 33. Breslau N, Chilcoat HD, Kessler RC, Davis GC: Previous exposure to trauma and PTSD effects of subsequent trauma: results from the Detroit Area Survey of Trauma. Am J Psychiatry 1999; 156:902–907 [G]
- 34. Ursano RJ, Fullerton CS, Epstein RS, Crowley B, Kao TC, Vance K, Craig KJ, Dougall AL, Baum A: Acute and chronic posttraumatic stress disorder in motor vehicle accident victims. Am J Psychiatry 1999; 156:589–595 [C]
- 35. Devilly GJ, Spence SH: The relative efficacy and treatment distress of EMDR and a cognitive-behavior trauma treatment protocol in the amelioration of posttraumatic stress disorder. J Anxiety Disord 1999; 13:131–157 [A–]
- 36. Strasburger LH, Gutheil TG, Brodsky A: On wearing two hats: role conflict in serving as both psychotherapist and expert witness. Am J Psychiatry 1997; 154:448–456 [G]
- 37. Gutheil TG, Hilliard JT: The treating psychiatrist thrust into the role of expert witness. Psychiatr Serv 2001; 52:1526–1527 [G]
- 38. Vallabhajosula B, van Gorp WG: Post-Daubert admissibility of scientific evidence on malingering of cognitive deficits. J Am Acad Psychiatry Law 2001; 29:207–215 [F]
- 39. Beckham JC, Lytle BL, Vrana SR, Hertzberg MA, Feldman ME, Shipley RH: Smoking withdrawal symptoms in response to a trauma-related stressor among Vietnam combat veterans with posttraumatic stress disorder. Addict Behav 1996; 21:93–101 [B]
- Beckham JC: Smoking and anxiety in combat veterans with chronic posttraumatic stress disorder: a review. J Psychoactive Drugs 1999; 31:103–110 [F]
- Silverman JJ, Peed SF, Goldberg S, Hamer RM, Stockman SJ: Surgical staff recognition of psychopathology in trauma patients. J Trauma 1985; 25:544–546 [C]
- 42. Whetsell LA, Patterson CM, Young DH, Schiller WR: Preinjury psychopathology in trauma patients. J Trauma 1989; 29:1158–1161 [C]
- 43. Cerda G, Zatzick D, Wise M, Greenhalgh D: Computerized registry recording of psychiatric disorders of pediatric patients with burns. J Burn Care Rehabil 2000; 21:368–370 [C]

- 44. Zatzick DF, Kang SM, Kim SY, Leigh P, Kravitz R, Drake C, Sue S, Wisner D: Patients with recognized psychiatric disorders in trauma surgery: incidence, inpatient length of stay, and cost. J Trauma 2000; 49:487–495 [C]
- 45. Coverdale JH, Turbott SH: Sexual and physical abuse of chronically ill psychiatric outpatients compared with a matched sample of medical outpatients. J Nerv Ment Dis 2000; 188:440–445 [D]
- 46. Goodman LA, Rosenberg SD, Mueser KT, Drake RE: Physical and sexual assault history in women with serious mental illness: prevalence, correlates, treatment, and future research directions. Schizophr Bull 1997; 23:685–696 [F]
- 47. Mueser KT, Goodman LB, Trumbetta SL, Rosenberg SD, Osher C, Vidaver R, Auciello P, Foy DW: Trauma and posttraumatic stress disorder in severe mental illness. J Consult Clin Psychol 1998; 66:493–499 [G]
- 48. Neria Y, Bromet EJ, Sievers S, Lavelle J, Fochtmann LJ: Trauma exposure and posttraumatic stress disorder in psychosis: findings from a first-admission cohort. J Consult Clin Psychol 2002; 70:246–251 [G]
- 49. Switzer GE, Dew MA, Thompson K, Goycoolea JM, Derricott T, Mullins SD: Posttraumatic stress disorder and service utilization among urban mental health center clients. J Trauma Stress 1999; 12:25–39 [G]
- 50. Rose SM, Peabody CG, Stratigeas B: Undetected abuse among intensive case management clients. Hosp Community Psychiatry 1991; 42:499–503 [D]
- 51. Mueser KT, Salyers MP, Rosenberg SD, Goodman LA, Essock SM, Osher FC, Swartz MS, Butterfield MI, 5 Site Health and Risk Study Research Committee: Interpersonal trauma and posttraumatic stress disorder in patients with severe mental illness: demographic, clinical, and health correlates. Schizophr Bull 2004; 30:45–57 [D]
- Famularo R, Kinscherff R, Fenton T: Posttraumatic stress disorder among children clinically diagnosed as borderline personality disorder. J Nerv Ment Dis 1991; 179:428–431 [G]
- 53. Goldman SJ, D'Angelo EJ, DeMaso DR, Mezzacappa E: Physical and sexual abuse histories among children with borderline personality disorder. Am J Psychiatry 1992; 149:1723–1726 [D]
- 54. Yen S, Shea MT, Battle CL, Johnson DM, Zlotnick C, Dolan-Sewell R, Skodol AE, Grilo CM, Gunderson JG, Sanislow CA, Zanarini MC, Bender DS, Rettew JB, McGlashan TH: Traumatic exposure and posttraumatic stress disorder in borderline, schizotypal, avoidant, and obsessive-compulsive personality disorders: findings from the collaborative longitudinal personality disorders study. J Nerv Ment Dis 2002; 190:510–518 [G]
- 55. Bonin MF, Norton GR, Asmundson GJ, Dicurzio S, Pidlubney S: Drinking away the hurt: the nature and prevalence of PTSD in substance abuse patients attending a community-based treatment program. J Behav Ther Exp Psychiatry 2000; 31:55–66 [G]
- 56. Epstein JN, Saunders BE, Kilpatrick DG, Resnick HS: PTSD as a mediator between childhood rape and alcohol use in adult women. Child Abuse Negl 1998; 22:223–234 [G]
- 57. Duncan RD, Saunders BE, Kilpatrick DG, Hanson RF, Resnick HS: Childhood physical assault as a risk factor for PTSD, depression, and substance abuse: findings from a national survey. Am J Orthopsychiatry 1996; 66:437–448 [D]
- 58. Brady KT, Wohlreich M: Victimization, PTSD, and substance abuse. Women's Psychiatric Health 1994; 3:12–14 [D]
- 59. deGroot JM, Kennedy S, Rodin G, McVey G: Correlates of sexual abuse in women with anorexia nervosa and bulimia nervosa. Can J Psychiatry 1992; 37:516–518 [G]
- 60. McFarlane AC, McFarlane CM, Gilchrist PN: Posttraumatic bulimia and anorexia nervosa. Int J Eat Disord 1988; 7:705–708 [G]
- 61. Herzog DB, Staley JE, Carmody S, Robbins WM, van der Kolk BA: Childhood sexual abuse in anorexia nervosa and bulimia nervosa: a pilot study. J Am Acad Child Adolesc Psychiatry 1993; 32:962–966 [G]

- 62. Zisook S, Chentsova-Dutton Y, Shuchter SR: PTSD following bereavement. Ann Clin Psychiatry 1998; 10:157–163 [B]
- 63. Dobie DJ, Kivlahan DR, Maynard C, Bush KR, Davis TM, Bradley KA: Posttraumatic stress disorder in female veterans: association with self-reported health problems and functional impairment. Arch Intern Med 2004; 164:394–400 [G]
- 64. American Psychiatric Association: Practice Guideline for the Assessment and Treatment of Patients With Suicidal Behaviors. Am J Psychiatry 2003; 160(Nov suppl) [G]
- 65. Morgan HG, Burns-Cox CJ, Pocock H, Pottle S: Deliberate self-harm: clinical and socioeconomic characteristics of 368 patients. Br J Psychiatry 1975; 127:564–574 [G]
- Pattison EM, Kahan J: The deliberate self-harm syndrome. Am J Psychiatry 1983; 140:867– 872 [G]
- 67. Anderson PL, Tiro JA, Price AW, Bender MA, Kaslow NJ: Additive impact of childhood emotional, physical, and sexual abuse on suicide attempts among low-income African American women. Suicide Life Threat Behav 2002; 32:131–138 [G]
- 68. Windle M, Windle RC, Scheidt DM, Miller GB: Physical and sexual abuse and associated mental disorders among alcoholic inpatients. Am J Psychiatry 1995; 152:1322–1328 [D]
- 69. Friedman MJ: Post Traumatic Stress Disorder: The Latest Assessment and Treatment Strategies. Kansas City, Mo, Compact Clinicals, 2000 [G]
- 70. Wilson JP, Friedman MJ, Lindy JD (eds): Treating Psychological Trauma and PTSD. New York, Guilford, 2001 [G]
- 71. Williams MB, Sommer JF Jr: Handbook of Post-Traumatic Therapy. Westport, Conn, Greenwood Press, 1994 [G]
- 72. Rosenheck R, Fontana A: Impact of efforts to reduce inpatient costs on clinical effectiveness: treatment of posttraumatic stress disorder in the Department of Veterans Affairs. Med Care 2001; 39:168–180 [D]
- 73. Roy-Byrne P, Berliner L, Russo J, Zatzick D, Pitman RK: Treatment preferences and determinants in victims of sexual and physical assault. J Nerv Ment Dis 2003; 191:161–165 [G]
- 74. Weisaeth L: The stressors and the post-traumatic stress syndrome after an industrial disaster. Acta Psychiatr Scand Suppl 1989; 355:25–37 [D]
- 75. Wilson JP, Lindy JD (eds): Countertransference in the Treatment of PTSD. New York, Guilford, 1994 [G]
- 76. Kimerling R, Calhoun KS: Somatic symptoms, social support, and treatment seeking among sexual assault victims. J Consult Clin Psychol 1994; 62:333–340 [C]
- 77. McFarlane AC, Atchison M, Rafalowicz E, Papay P: Physical symptoms in post-traumatic stress disorder. J Psychosom Res 1994; 38:715–726 [C]
- 78. Andreski P, Chilcoat H, Breslau N: Post-traumatic stress disorder and somatization symptoms: a prospective study. Psychiatry Res 1998; 79:131–138 [C]
- 79. Engel CC Jr, Liu X, McCarthy BD, Miller RF, Ursano R: Relationship of physical symptoms to posttraumatic stress disorder among veterans seeking care for Gulf War-related health concerns. Psychosom Med 2000; 62:739–745 [D]
- 80. Ghahramanlou M, Boradbeck C: Predictors of secondary trauma in sexual assault counselors. Int J Emerg Ment Health 2000; 4:229–240 [D]
- 81. Figley C (ed): Compassion Fatigue: Coping With Secondary Traumatic Stress Disorders in Those Who Treat the Traumatized. New York, Brunner-Routledge, 1995 [G]
- 82. Lin EH, Von Korff M, Katon W, Bush T, Simon GE, Walker E, Robinson P: The role of the primary care physician in patients' adherence to antidepressant therapy. Med Care 1995; 33:67–74 [B]
- 83. Zygmunt A, Olfson M, Boyer CA, Mechanic D: Interventions to improve medication adherence in schizophrenia. Am J Psychiatry 2002; 159:1653–1664 [E]

- 84. Phillips SD, Burns BJ, Edgar ER, Mueser KT, Linkins KW, Rosenheck RA, Drake RE, McDonel Herr EC: Moving assertive community treatment into standard practice. Psychiatr Serv 2001; 52:771–779 [F]
- 85. Marshall M, Lockwood A: Assertive community treatment for people with severe mental disorders. Cochrane Database Syst Rev 2000; (2):CD001089 [E]
- 86. American Psychiatric Association: Practice Guideline for the Treatment of Patients With Schizophrenia, Second Edition. Am J Psychiatry 2004; 161(Feb suppl) [G]
- 87. Galea S, Resnick H, Ahern J, Gold J, Bucuvalas M, Kilpatrick D, Stuber J, Vlahov D: Posttraumatic stress disorder in Manhattan, New York City, after the September 11th terrorist attacks. J Urban Health 2002; 79:340–353 [D]
- 88. Glantz MD, Sloboda Z: Analysis and reconceptualization of resilience, in Resilience and Development: Positive Life Adaptations. Edited by Glantz MD, Johnson JL. New York, Kluwer Academic/Plenum, 1999, pp 109–126 [G]
- 89. Kaplan HB: Toward an understanding of resilience: a critical review of definitions and models, in Resilience and Development: Positive Life Adaptations. Edited by Glantz MD, Johnson JL. New York, Kluwer Academic/Plenum, 1999, pp 17–84 [F]
- 90. Barnes DH, Bell CC: Paradoxes of black suicide. Preventing Suicide: The National Journal 2003; 2:2–4 [F]
- 91. Beck AT, Weissman A, Kovacs M: Alcoholism, hopelessness and suicidal behavior. J Stud Alcohol 1976; 37:66–77 [D]
- 92. Linehan MM, Goodstein JL, Nielsen SL, Chiles JA: Reasons for staying alive when you are thinking of killing yourself: the reasons for living inventory. J Consult Clin Psychol 1983; 51:276–286 [F]
- 93. Range LM, Penton SR: Hope, hopelessness, and suicidality in college students. Psychol Rep 1994; 75:456–458 [G]
- 94. Scheier M, Carver C: Effects of optimism on psychological and physical well-being: theoretical overview and empirical update. Cognitive Therapy and Research 1992; 16:201–228 [F]
- 95. Taylor SE, Kemeny ME, Reed GM, Bower JE, Gruenewald TL: Psychological resources, positive illusions, and health. Am Psychol 2000; 55:99–109 [G]
- 96. Werner EE: Vulnerable but invincible: high risk children from birth to adulthood. Eur Child Adolesc Psychiatry 1996; 5(suppl 1):47–51 [G]
- 97. Wyman PA, Cowen EL, Work WC, Kerley JH: The role of children's future expectations in self-esteem functioning and adjustment to life stress: a prospective study of urban at-risk children. Dev Psychopathol 1993; 5:649–661 [C]
- 98. Antoni MH, Lehman JM, Kilbourn KM, Boyers AE, Culver JL, Alferi SM, Yount SE, McGregor BA, Arena PL, Harris SD, Price AA, Carver CS: Cognitive-behavioral stress management intervention decreases the prevalence of depression and enhances benefit finding among women under treatment for early-stage breast cancer. Health Psychol 2001; 20:20–32 [D]
- Brissette I, Scheier MF, Carver CS: The role of optimism in social network development, coping, and psychological adjustment during a life transition. J Pers Soc Psychol 2002; 82:102–111 [C]
- 100. Jaycox LH, Reivich KJ, Gillham J, Seligman ME: Prevention of depressive symptoms in school children. Behav Res Ther 1994; 32:801–816 [B]
- 101. Gillham JE, Reivich KJ, Jaycox LH, Seligman MEP: Prevention of depressive symptoms in school children: two-year follow-up. Psychol Sci 1995; 6:343–351 [A–]
- Chemtob CM, Nakashima JP, Hamada RS: Psychosocial intervention for postdisaster trauma symptoms in elementary school children: a controlled community field study. Arch Pediatr Adolesc Med 2002; 156:211–216 [A]

- 103. Friedman M, Schnurr PP: The relationship between trauma, post-traumatic stress disorder and physical health, in Neurobiological and Clinical Consequences of Stress: From Normal Adaptation to Post-Traumatic Stress Disorder. Edited by Friedman M, Charney DS, Deutch AY. Philadelphia, Lippincott-Raven, 1995, pp 507–524 [G]
- Breslau N, Davis GC, Peterson EL, Schultz L: Psychiatric sequelae of posttraumatic stress disorder in women. Arch Gen Psychiatry 1997; 54:81–87 [D]
- McCarroll JE, Ursano RJ, Fullerton CS, Liu X, Lundy A: Somatic symptoms in Gulf War mortuary workers. Psychosom Med 2002; 64:29–33 [G]
- Schnurr PP, Friedman MJ, Sengupta A, Jankowski MK, Holmes T: PTSD and utilization of medical treatment services among male Vietnam veterans. J Nerv Ment Dis 2000; 188:496–504 [D]
- 107. Benedikt RA, Kolb LC: Preliminary findings on chronic pain and posttraumatic stress disorder. Am J Psychiatry 1986; 143:908–910 [G]
- 108. Geisser ME, Roth RS, Bachman JE, Eckert TA: The relationship between symptoms of post-traumatic stress disorder and pain, affective disturbance and disability among patients with accident and non-accident related pain. Pain 1996; 66:207–214 [D]
- Pecukonis EV: Childhood sex abuse in women with chronic intractable back pain. Soc Work Health Care 1996; 23:1–16 [C]
- 110. Badura AS, Reiter RC, Altmaier EM, Rhomberg A, Elas D: Dissociation, somatization, substance abuse, and coping in women with chronic pelvic pain. Obstet Gynecol 1997; 90:405–410 [G]
- 111. Drossman DA: Sexual and physical abuse and gastrointestinal illness. Scand J Gastroenterol Suppl 1995; 208:90–96 [G]
- 112. Plichta SB, Abraham C: Violence and gynecologic health in women <50 years old. Am J Obstet Gynecol 1996; 174:903–907 [G]
- 113. Walling MK, O'Hara MW, Reiter RC, Milburn AK, Lilly G, Vincent SD: Abuse history and chronic pain in women, II: a multivariate analysis of abuse and psychological morbidity. Obstet Gynecol 1994; 84:200–206 [D]
- 114. Walker EA, Gelfand AN, Gelfand MD, Green C, Katon WJ: Chronic pelvic pain and gynecological symptoms in women with irritable bowel syndrome. J Psychosom Obstet Gynaecol 1996; 17:39–46 [G]
- 115. Amaya-Jackson L, Davidson JR, Hughes DC, Swartz M, Reynolds V, George LK, Blazer DG: Functional impairment and utilization of services associated with posttraumatic stress in the community. J Trauma Stress 1999; 12:709–724 [C]
- 116. Booth BM, Blow FC, Cook CA: Functional impairment and co-occurring psychiatric disorders in medically hospitalized men. Arch Intern Med 1998; 158:1551–1559 [D]
- 117. Holbrook TL, Anderson JP, Sieber WJ, Browner D, Hoyt DB: Outcome after major trauma: 12-month and 18-month follow-up results from the Trauma Recovery Project. J Trauma 1999; 46:765–771 [C]
- 118. Holbrook TL, Hoyt DB, Stein MB, Sieber WJ: Perceived threat to life predicts posttraumatic stress disorder after major trauma: risk factors and functional outcome. J Trauma 2001; 51:287–292 [G]
- 119. Mollica RF, Donelan K, Tor S, Lavelle J, Elias C, Frankel M, Blendon RJ: The effect of trauma and confinement on functional health and mental health status of Cambodians living in Thailand-Cambodia border camps. JAMA 1993; 270:581–586 [G]
- 120. Momartin S, Silove D, Manicavasagar V, Steel Z: Dimensions of trauma associated with posttraumatic stress disorder (PTSD) caseness, severity and functional impairment: a study of Bosnian refugees resettled in Australia. Soc Sci Med 2003; 57:775–781 [G]
- 121. Schnurr PP, Ford JD, Friedman MJ, Green BL, Dain BJ, Sengupta A: Predictors and outcomes of posttraumatic stress disorder in World War II veterans exposed to mustard gas. J Consult Clin Psychol 2000; 68:258–268 [G]

- 122. Zatzick DF, Marmar CR, Weiss DS, Browner WS, Metzler TJ, Golding JM, Stewart A, Schlenger WE, Wells KB: Posttraumatic stress disorder and functioning and quality of life outcomes in a nationally representative sample of male Vietnam veterans. Am J Psychiatry 1997; 154:1690–1695 [C]
- 123. Davidson J, Pearlstein T, Londborg P, Brady KT, Rothbaum B, Bell J, Maddock R, Hegel MT, Farfel G: Efficacy of sertraline in preventing relapse of posttraumatic stress disorder: results of a 28-week double-blind, placebo-controlled study. Am J Psychiatry 2001; 158:1974–1981 [A]
- 124. Kudler HS, Blank AS, Krupnick JL: The psychodynamic treatment of PTSD, in Effective Treatments for PTSD: Practice Guidelines From the International Society for Traumatic Stress Studies. Edited by Foa EB, Keane TM, Friedman MJ. New York, Guilford, 2000, pp 176–198 [F]
- 125. Harvey AG, Bryant RA, Tarrier N: Cognitive behaviour therapy for posttraumatic stress disorder. Clin Psychol Rev 2003; 23:501–522 [F]
- 126. Plichta S: Violence and abuse, in Women's Health: The Commonwealth Fund Survey. Edited by Falik M, Collins KS. Baltimore, Johns Hopkins University Press, 1996, pp 237–270 [D]
- 127. Holloway HC, Ursano RJ: The Vietnam veteran: memory, social context, and metaphor. Psychiatry 1984; 47:103–108 [G]
- 128. Wessely S, Bisson J, Rose S: A systematic review of brief psychological interventions ("debriefing") for the treatment of immediate trauma related symptoms and the prevention of posttraumatic stress disorder, in The Cochrane Library 1998, issue 3. Oxford, UK, Update Software (http://www.update-software.com/cochrane) [E]
- 129. van Emmerik AA, Kamphuis JH, Hulsbosch AM, Emmelkamp PM: Single session debriefing after psychological trauma: a meta-analysis. Lancet 2002; 360:766–771 [E]
- 130. Litz BT, Gray M: Early intervention for trauma in adults, in Early Intervention for Trauma and Traumatic Loss. Edited by Litz BT. New York, Guilford, 2004, pp 87–111 [G]
- 131. Zatzick D, Roy-Byrne P, Russo J, Rivara F, Droesh R, Wagner A, Dunn C, Jurkovich G, Uehara E, Katon W: A randomized effectiveness trial of stepped collaborative care for acutely injured trauma survivors. Arch Gen Psychiatry 2004; 61:498–506 [A–]
- 132. Bordow S, Porritt D: An experimental evaluation of crisis intervention. Soc Sci Med 1979; 13A:251–256 [B]
- 133. Gidron Y, Gal R, Freedman S, Twiser I, Lauden A, Snir Y, Benjamin J: Translating research findings to PTSD prevention: results of a randomized-controlled pilot study. J Trauma Stress 2001; 14:773–780 [A–]
- 134. Zatzick DF, Roy-Byrne P, Russo JE, Rivara FP, Koike A, Jurkovich GJ, Katon W: Collaborative interventions for physically injured trauma survivors: a pilot randomized effectiveness trial. Gen Hosp Psychiatry 2001; 23:114–123 [A–]
- 135. Bryant RA, Sackville T, Dang ST, Moulds M, Guthrie R: Treating acute stress disorder: an evaluation of cognitive behavior therapy and supportive counseling techniques. Am J Psychiatry 1999; 156:1780–1786 [A]
- 136. Bryant RA, Harvey AG, Dang ST, Sackville T, Basten C: Treatment of acute stress disorder: a comparison of cognitive-behavioral therapy and supportive counseling. J Consult Clin Psychol 1998; 66:862–866 [B]
- 137. Pitman RK, Sanders KM, Zusman RM, Healy AR, Cheema F, Lasko NB, Cahill L, Orr SP: Pilot study of secondary prevention of posttraumatic stress disorder with propranolol. Biol Psychiatry 2002; 51:189–192 [A]
- 138. Rehm LP: How can we better disentangle placebo and drug effects? Commentary on The emperor's new drugs: an analysis of antidepressant medication data submitted to the US Food and Drug Administration. Prevention & Treatment 2002, volume 5. Washington, DC, American Psychological Association (http://journals.apa.org/prevention/volume5/pre0050031c.html) [G]

- 139. Rothbaum B, Foa E: Subtypes of posttraumatic stress disorder and duration of symptoms, in Posttraumatic Stress Disorder: DSM-IV and Beyond. Edited by Davidson J, Foa E. Washington, DC, American Psychiatric Press, 1994, pp 23–35 [F]
- 140. Blanchard EB, Hickling EJ: Acute stress disorder among MVA survivors, in After the Crash: Assessment and Treatment of Motor Vehicle Accident Survivors. Washington, DC, American Psychological Association, 1997, pp 187–196 [G]
- 141. Marshall RD, Beebe KL, Oldham M, Zaninelli R: Efficacy and safety of paroxetine treatment for chronic PTSD: a fixed-dose, placebo-controlled study. Am J Psychiatry 2001; 158:1982–1988 [A]
- 142. Connor KM, Sutherland SM, Tupler LA, Malik ML, Davidson JR: Fluoxetine in post-traumatic stress disorder: randomised, double-blind study. Br J Psychiatry 1999; 175:17–22 [A]
- 143. van der Kolk BA, Dreyfuss D, Michaels M, Shera D, Berkowitz R, Fisler R, Saxe G: Fluoxetine in posttraumatic stress disorder. J Clin Psychiatry 1994; 55:517–522 [A]
- 144. Brady K, Pearlstein T, Asnis GM, Baker D, Rothbaum B, Sikes CR, Farfel GM: Efficacy and safety of sertraline treatment of posttraumatic stress disorder: a randomized controlled trial. JAMA 2000; 283:1837–1844 [A]
- Tucker P, Zaninelli R, Yehuda R, Ruggiero L, Dillingham K, Pitts CD: Paroxetine in the treatment of chronic posttraumatic stress disorder: results of a placebo-controlled, flexibledosage trial. J Clin Psychiatry 2001; 62:860–868 [A]
- Martenyi F, Brown EB, Zhang H, Prakash A, Koke SC: Fluoxetine versus placebo in posttraumatic stress disorder. J Clin Psychiatry 2002; 63:199–206 [A]
- Neylan TC, Metzler TJ, Schoenfeld FB, Weiss DS, Lenoci M, Best SR, Lipsey TL, Marmar CR: Fluvoxamine and sleep disturbances in posttraumatic stress disorder. J Trauma Stress 2001; 14:461–467 [B]
- 148. Rapaport MH, Endicott J, Clary CM: Posttraumatic stress disorder and quality of life: results across 64 weeks of sertraline treatment. J Clin Psychiatry 2002; 63:59–65 [A]
- Davidson JR, Landerman LR, Farfel GM, Clary CM: Characterizing the effects of sertraline in post-traumatic stress disorder. Psychol Med 2002; 32:661–670 [A]
- Marmar CR, Schoenfeld F, Weiss DS, Metzler T, Zatzick D, Wu R, Smiga S, Tecott L, Neylan T: Open trial of fluvoxamine treatment for combat-related posttraumatic stress disorder. J Clin Psychiatry 1996; 57(suppl 8):66–70 [B]
- 151. Kosten TR, Frank JB, Dan E, McDougle CJ, Giller EL Jr: Pharmacotherapy for posttraumatic stress disorder using phenelzine or imipramine. J Nerv Ment Dis 1991; 179:366–370 [G]
- 152. Davidson J, Kudler H, Smith R, Mahorney SL, Lipper S, Hammett E, Saunders WB, Cavenar JO Jr: Treatment of posttraumatic stress disorder with amitriptyline and placebo. Arch Gen Psychiatry 1990; 47:259–266 [A]
- 153. Reist C, Kauffmann CD, Haier RJ, Sangdahl C, DeMet EM, Chicz-DeMet A, Nelson JN: A controlled trial of desipramine in 18 men with posttraumatic stress disorder. Am J Psychiatry 1989; 146:513–516 [A]
- 154. Katz RJ, Lott MH, Arbus P, Crocq L, Herlobsen P, Lingjaerde O, Lopez G, Loughrey GC, MacFarlane DJ, McIvor R, Mehlum L, Nugent D, Turner SW, Waisaeth L, Yule W: Pharmacotherapy of post-traumatic stress disorder with a novel psychotropic. Anxiety 1994–1995; 1:169–174 [G]
- 155. Baker DG, Diamond BI, Gillette GM, Hamner MB, Katzelnick D, Keller TW, Mellman TA, Pontius EB, Rosenthal M, Tucker P, van der Kolk BA, Katz RJ: A double-blind, randomized, placebo-controlled, multi-center study of brofaromine in the treatment of post-traumatic stress disorder. Psychopharmacology (Berl) 1995; 122:386–389 [A]
- 156. Mellman TA, Bustamante V, David D, Fins AI: Hypnotic medication in the aftermath of trauma. J Clin Psychiatry 2002; 63:1183–1184 [G]
- 157. Gelpin E, Bonne O, Peri T, Brandes D, Shalev AY: Treatment of recent trauma survivors with benzodiazepines: a prospective study. J Clin Psychiatry 1996; 57:390–394 [B]

- 158. Risse SC, Whitters A, Burke J, Chen S, Scurfield RM, Raskind MA: Severe withdrawal symptoms after discontinuation of alprazolam in eight patients with combat-induced posttraumatic stress disorder. J Clin Psychiatry 1990; 51:206–209 [B]
- 159. Kosten TR, Fontana A, Sernyak MJ, Rosenheck R: Benzodiazepine use in posttraumatic stress disorder among veterans with substance abuse. J Nerv Ment Dis 2000; 188:454–459 [C]
- Lipper S, Davidson JR, Grady TA, Edinger JD, Hammett EB, Mahorney SL, Cavenar JO
 Jr: Preliminary study of carbamazepine in post-traumatic stress disorder. Psychosomatics 1986; 27:849–854 [B]
- 161. Clark RD, Canive JM, Calais LA, Qualls CR, Tuason VB: Divalproex in posttraumatic stress disorder: an open-label clinical trial. J Trauma Stress 1999; 12:395–401 [B]
- 162. Fesler FA: Valproate in combat-related posttraumatic stress disorder. J Clin Psychiatry 1991; 52:361–364 [B]
- 163. Hertzberg MA, Butterfield MI, Feldman ME, Beckham JC, Sutherland SM, Connor KM, Davidson JR: A preliminary study of lamotrigine for the treatment of posttraumatic stress disorder. Biol Psychiatry 1999; 45:1226–1229 [A]
- 164. Butterfield MI, Becker ME, Connor KM, Sutherland S, Churchill LE, Davidson JR: Olanzapine in the treatment of post-traumatic stress disorder: a pilot study. Int Clin Psychopharmacol 2001; 16:197–203 [A]
- Petty F, Brannan S, Casada J, Davis LL, Gajewski V, Kramer GL, Stone RC, Teten AL, Worchel J, Young KA: Olanzapine treatment for post-traumatic stress disorder: an openlabel study. Int Clin Psychopharmacol 2001; 16:331–337 [B]
- Stein MB, Kline NA, Matloff JL: Adjunctive olanzapine for SSRI-resistant combat-related PTSD: a double-blind, placebo-controlled study. Am J Psychiatry 2002; 159:1777–1779 [A]
- Hamner MB, Deitsch SE, Brodrick PS, Ulmer HG, Lorberbaum JP: Quetiapine treatment in patients with posttraumatic stress disorder: an open trial of adjunctive therapy. J Clin Psychopharmacol 2003; 23:15–20 [B]
- 168. Hamner MB, Faldowski RA, Ulmer HG, Frueh BC, Huber MG, Arana GW: Adjunctive risperidone treatment in post-traumatic stress disorder: a preliminary controlled trial of effects on comorbid psychotic symptoms. Int Clin Psychopharmacol 2003; 18:1–8 [A]
- 169. Raskind MA, Peskind ER, Kanter ED, Petrie EC, Radant A, Thompson CE, Dobie DJ, Hoff D, Rein RJ, Straits-Troster K, Thomas RG, McFall MM: Reduction of nightmares and other PTSD symptoms in combat veterans by prazosin: a placebo-controlled study. Am J Psychiatry 2003; 160:371–373 [A]
- 170. Kinzie JD, Leung P: Clonidine in Cambodian patients with posttraumatic stress disorder. J Nerv Ment Dis 1989; 177:546–550 [B]
- 171. Faustman WO, White PA: Diagnostic and psychopharmacological treatment characteristics of 536 inpatients with posttraumatic stress disorder. J Nerv Ment Dis 1989; 177:154–159 [D]
- 172. Vaiva G, Ducrocq F, Jezequel K, Averland B, Lestavel P, Brunet A, Marmar CR: Immediate treatment with propranolol decreases posttraumatic stress disorder two months after trauma. Biol Psychiatry 2003; 54:947–949 [B]
- 173. Foa EB, Rothbaum BO: Treating the Trauma of Rape: Cognitive-Behavioral Therapy for PTSD. New York, Guilford, 1997 [G]
- 174. Keane TM, Gerarali RJ, Quinn SJ, Litz BT: Behavioral treatment of posttraumatic stress disorder, in Handbook of Clinical Behavior Therapy, 2nd ed. Edited by Turner SM, Calhoun KS, Adams HE. New York, Wiley, 1992, pp 87–97 [G]
- 175. Bryant RA, Moulds ML, Nixon RV: Cognitive behaviour therapy of acute stress disorder: a four-year follow-up. Behav Res Ther 2003; 41:489–494 [A]
- 176. Solomon SD, Johnson DM: Psychosocial treatment of posttraumatic stress disorder: a practice-friendly review of outcome research. J Clin Psychol 2002; 58:947–959 [F]

- 177. Marks I, Lovell K, Noshirvani H, Livanou M, Thrasher S: Treatment of posttraumatic stress disorder by exposure and/or cognitive restructuring: a controlled study. Arch Gen Psychiatry 1998; 55:317–325 [A]
- 178. Tarrier N, Pilgrim H, Sommerfield C, Faragher B, Reynolds M, Graham E, Barrowclough C: 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 [A]
- 179. Hembree EA, Foa EB: Posttraumatic stress disorder: psychological factors and psychosocial interventions. J Clin Psychiatry 2000; 61(suppl 7):33–39 [F]
- Resick PA, Jordan CG, Girelli SA, Hutter CK, Marhoefer-Dvorak S: A comparative victim study of behavioral group therapy for sexual assault victims. Behav Ther 1988; 19:385–401 [B]
- 181. Resick PA, Nishith P, Weaver TL, Astin MC, Feuer CA: A comparison of cognitive-processing therapy with prolonged exposure and a waiting condition for the treatment of chronic posttraumatic stress disorder in female rape victims. J Consult Clin Psychol 2002; 70:867–879 [A–]
- 182. Foa EB: Trauma and women: course, predictors, and treatment. J Clin Psychiatry 1997; 58(suppl 9):25–28 [E]
- 183. Foy DW, Ruzek JI, Glynn SM, Riney SA, Gusman FD: Trauma focus group therapy for combat-related PTSD. Psychotherapy in Practice 1996; 3(4):59–73 [G]
- 184. Krakow B, Hollifield M, Johnston L, Koss M, Schrader R, Warner TD, Tandberg D, Lauriello J, McBride L, Cutchen L, Cheng D, Emmons S, Germain A, Melendrez D, Sandoval D, Prince H: Imagery rehearsal therapy for chronic nightmares in sexual assault survivors with posttraumatic stress disorder: a randomized controlled trial. JAMA 2001; 286:537–545 [A-]
- 185. Forbes D, Phelps A, McHugh T: Treatment of combat-related nightmares using imagery rehearsal: a pilot study. J Trauma Stress 2001; 14:433–442 [B]
- Tarrier N, Sommerfield C, Pilgrim H, Faragher B: Factors associated with outcome of cognitive-behavioural treatment of chronic post-traumatic stress disorder. Behav Res Ther 2000; 38:191–202 [A]
- 187. Tarrier N, Sommerfield C, Pilgrim H, Humphreys L: Cognitive therapy or imaginal exposure in the treatment of post-traumatic stress disorder: twelve-month follow-up. Br J Psychiatry 1999; 175:571–575 [A]
- 188. Resick PA, Schnicke MK: Cognitive processing therapy for sexual assault victims. J Consult Clin Psychol 1992; 60:748–756 [A–]
- PTSD Treatment Guidelines Task Force: Guidelines for treatment of PTSD: eye movement desensitization and reprocessing. J Trauma Stress 2000; 13:539–588 [E]
- 190. Foa EB, Meadows EA: Psychosocial treatments for posttraumatic stress disorder: a critical review. Annu Rev Psychol 1997; 48:449–480 [F]
- 191. Shepherd J, Stein K, Milne R: Eye movement desensitization and reprocessing in the treatment of post-traumatic stress disorder: a review of an emerging therapy. Psychol Med 2000; 30:863–871 [F]
- 192. Van Etten M, Taylor S: Comparative efficacy of treatments for post-traumatic stress disorder: a meta-analysis. Clinical Psychology and Psychotherapy 1998; 5:126–144 [E]
- 193. Maxfield L, Hyer L: The relationship between efficacy and methodology in studies investigating EMDR treatment of PTSD. J Clin Psychol 2002; 58:23–41 [E]
- 194. Davidson PR, Parker KC: Eye movement desensitization and reprocessing (EMDR): a meta-analysis. J Consult Clin Psychol 2001; 69:305–316 [E]
- 195. Hembree EA, Foa EB, Dorfan NM, Street GP, Kowalski J, Tu X: Do patients drop out prematurely from exposure therapy for PTSD? J Trauma Stress 2003; 16:555–562 [F]
- 196. Herbert JD, Lilienfeld SO, Lohr JM, Montgomery RW, O'Donohue WT, Rosen GM, Tolin DF: Science and pseudoscience in the development of eye movement desensitization and reprocessing: implications for clinical psychology. Clin Psychol Rev 2000; 20:945–971 [F]

- 197. Lipke H: Comment on Hembree and Foa (2003) and EMDR. J Trauma Stress 2003; 16:573–574 [G]
- 198. Kardiner A: The Traumatic Neurosis of War. New York, Hoeber, 1941 [G]
- 199. Grinker RR Sr, Spiegel JP: Men Under Stress. Philadelphia, Blakiston, 1945 [G]
- 200. Grinker RR Sr, Spiegel JP: A psychodynamic view of character pathology in Vietnam combat veterans. Bull Menninger Clin 1983; 47:472–474 [G]
- Brom D, Kleber RJ, Defares PB: Brief psychotherapy for posttraumatic stress disorders.
 J Consult Clin Psychol 1989; 57:607–612 [B]
- 202. Regarding Hypnosis: Position Statement [APA Document Reference No. 610001]. Washington, DC, American Psychiatric Association, Feb 1961. http://www.psych.org/edu/other_res/lib_archives/archives/610001.pdf [G]
- 203. Sherman JJ: Effects of psychotherapeutic treatments for PTSD: a meta-analysis of controlled clinical trials. J Trauma Stress 1998; 11:413–435 [E]
- 204. Freud A: Comments on trauma, in Psychic Trauma. Edited by Furst SS. New York, Basic Books, 1967, pp 235–245 [G]
- Weiss J, Sampson H, Mount Zion Psychotherapy Research Group: The Psychoanalytic Process: Theory, Clinical Observations, and Empirical Research. New York, Guilford, 1986 [E]
- 206. Modlin HC: The trauma in "traumatic neurosis." Bull Menninger Clin 1960; 24:49-56 [F]
- 207. Shaw JA: Unmasking the illusion of safety: psychic trauma in war. Bull Menninger Clin 1987; 51:49–63 [F]
- 208. Lindy JD, Green BL, Grace M, Titchener J: Psychotherapy with survivors of the Beverly Hills Supper Club fire. Am J Psychother 1983; 37:593–610 [D]
- 209. Marmar CR: Brief dynamic psychotherapy of post-traumatic stress disorder. Psychiatr Ann 1991; 21:405–414 [F]
- 210. Gabbard GO: Psychodynamic Psychiatry in Clinical Practice, 3rd ed. Washington, DC, American Psychiatric Press, 2000 [G]
- 211. Ursano RJ, Silberman EK: Psychoanalysis, psychoanalytic psychotherapy, and supportive psychotherapy, in The American Psychiatric Publishing Textbook of Clinical Psychiatry, 4th ed. Edited by Hales RE, Yudofsky SC. Arlington, VA, American Psychiatric Publishing, 2003, pp 1177–1203 [F]
- 212. Chertoff J: Psychodynamic assessment and treatment of traumatized patients. J Psychother Pract Res 1997; 7:35–46 [F]
- 213. Conlon L, Fahy TJ, Conroy R: PTSD in ambulant RTA victims: a randomized controlled trial of debriefing. J Psychosom Res 1999; 46:37–44 [A]
- 214. Lee C, Slade P, Lygo V: The influence of psychological debriefing on emotional adaptation in women following early miscarriage: a preliminary study. Br J Med Psychol 1996; 69(pt 1): 47–58 [B]
- 215. Rose S, Brewin CR, Andrews B, Kirk M: A randomized controlled trial of individual psychological debriefing for victims of violent crime. Psychol Med 1999; 29:793–799 [A–]
- 216. Raphael B: Early interventions and the debriefing debate, in Terrorism and Disaster: Individual and Community Mental Health Interventions. Edited by Ursano RJ, Fullerton CS, Norwood AE. Cambridge, UK, Cambridge University Press, 2003, pp 146–161 [F]
- 217. Bisson JI, Jenkins PL, Alexander J, Bannister C: Randomised controlled trial of psychological debriefing for victims of acute burn trauma. Br J Psychiatry 1997; 171:78–81 [A–]
- 218. Hobbs M, Mayou R, Harrison B, Worlock P: A randomised controlled trial of psychological debriefing for victims of road traffic accidents. BMJ 1996; 313:1438–1439 [A–]
- 219. 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 [C]
- 220. Litz BT, Gray MJ, Bryant R, Adler AB: Early intervention for trauma: current status and future directions. Clinical Psychology: Science and Practice 2002; 9:112–134 [F]

- 221. Suzanna R, Jonathan B, Simon W: Psychological debriefing for preventing post traumatic stress disorder (PTSD). Cochrane Database Syst Rev 2001; (3):CD000560 [E]
- 222. Brewin CR, Andrews B, Valentine JD: Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. J Consult Clin Psychol 2000; 68:748–766 [E]
- 223. Breslau N: The epidemiology of posttraumatic stress disorder: what is the extent of the problem? J Clin Psychiatry 2001; 62(suppl 17):16–22 [F]
- 224. Russell DE: The incidence and prevalence of intrafamilial and extrafamilial sexual abuse of female children. Child Abuse Negl 1983; 7:133–146 [G]
- 225. Finkelhor D, Hotaling G, Lewis IA, Smith C: Sexual abuse in a national survey of adult men and women: prevalence, characteristics, and risk factors. Child Abuse Negl 1990; 14:19–28 [D]
- Breslau N: Gender differences in trauma and posttraumatic stress disorder. J Gend Specif Med 2002; 5:34–40 [G]
- 227. de Vries MW: Trauma in cultural perspective, in Traumatic Stress: The Effects of Overwhelming Experience on Mind, Body, and Society. Edited by van der Kolk BA, McFarlane AC, Weisaeth L. New York, Guilford, 1996, pp 398–413 [F]
- 228. Lazarus RS, Folkman S: Stress, Appraisal, and Coping. New York, Springer, 1984 [G]
- 229. Kluft RP, Bloom SL, Kinzie JD: Treating traumatized patients and victims of violence. New Dir Ment Health Serv 2000; 86:79–102 [F]
- 230. Rosenheck R, Fontana A: Utilization of mental health services by minority veterans of the Vietnam era. J Nerv Ment Dis 1994; 182:685–691 [E]
- 231. Lin KM, Smith MW, Ortiz V: Culture and psychopharmacology. Psychiatr Clin North Am 2001; 24:523–538 [F]
- 232. Cascorbi I: Pharmacogenetics of cytochrome p4502D6: genetic background and clinical implication. Eur J Clin Invest 2003; 33(suppl 2):17–22 [G]
- 233. Poolsup N, Li Wan Po A, Knight TL: Pharmacogenetics and psychopharmacotherapy. J Clin Pharm Ther 2000; 25:197–220 [F]
- 234. Bertilsson L, Dahl ML, Tybring G: Pharmacogenetics of antidepressants: clinical aspects. Acta Psychiatr Scand Suppl 1997; 391:14–21 [F]
- 235. Gelernter J, Kranzler H, Cubells JF: Serotonin transporter protein (SLC6A4) allele and haplotype frequencies and linkage disequilibria in African- and European-American and Japanese populations and in alcohol-dependent subjects. Hum Genet 1997; 101:243–246 [G]
- 236. Williams RB, Marchuk DA, Gadde KM, Barefoot JC, Grichnik K, Helms MJ, Kuhn CM, Lewis JG, Schanberg SM, Stafford-Smith M, Suarez EC, Clary GL, Svenson IK, Siegler IC: Serotonin-related gene polymorphisms and central nervous system serotonin function. Neuropsychopharmacology 2003; 28:533–541 [G]
- 237. Salmon P, Calderbank S: The relationship of childhood physical and sexual abuse to adult illness behavior. J Psychosom Res 1996; 40:329–336 [G]
- 238. Fukudo S, Nomura T, Muranaka M, Taguchi F: Brain-gut response to stress and cholinergic stimulation in irritable bowel syndrome: a preliminary study. J Clin Gastroenterol 1993; 17:133–141 [A–]
- 239. Irwin C, Falsetti SA, Lydiard RB, Ballenger JC, Brock CD, Brener W: Comorbidity of posttraumatic stress disorder and irritable bowel syndrome. J Clin Psychiatry 1996; 57:576–578 [G]
- 240. Leserman J, Drossman DA, Li Z, Toomey TC, Nachman G, Glogau L: Sexual and physical abuse history in gastroenterology practice: how types of abuse impact health status. Psychosom Med 1996; 58:4–15 [F]
- 241. Walker EA, Katon WJ, Hansom J, Harrop-Griffiths J, Holm L, Jones ML, Hickok L, Jemelka RP: Medical and psychiatric symptoms in women with childhood sexual abuse. Psychosom Med 1992; 54:658–664 [E]

- 242. Davidson JR, Hughes D, Blazer DG, George LK: Post-traumatic stress disorder in the community: an epidemiological study. Psychol Med 1991; 21:713–721 [G]
- 243. Amir M, Kaplan Z, Neumann L, Sharabani R, Shani N, Buskila D: Posttraumatic stress disorder, tenderness and fibromyalgia. J Psychosom Res 1997; 42:607–613 [D]
- 244. Walker EA, Gelfand A, Katon WJ, Koss MP, Von Korff M, Bernstein D, Russo J: Adult health status of women with histories of childhood abuse and neglect. Am J Med 1999; 107:332–339 [D]
- Fierman EJ, Hunt MF, Pratt LA, Warshaw MG, Yonkers KA, Peterson LG, Epstein-Kaye TM, Norton HS: Trauma and posttraumatic stress disorder in subjects with anxiety disorders. Am J Psychiatry 1993; 150:1872–1874 [D]
- Green BL, Lindy JD, Grace MC, Leonard AC: Chronic posttraumatic stress disorder and diagnostic comorbidity in a disaster sample. J Nerv Ment Dis 1992; 180:760–766 [C]
- 247. Kulka RA, Schlenger WE, Fairbank JA, Hough RL, Jordan BK, Marmar CR, Weiss DS: Trauma and the Vietnam War Generation: Report of Findings From the National Vietnam Veterans Readjustment Study. New York, Brunner/Mazel, 1990 [G]
- 248. Boudewyns PA, Albrecht JW, Talbert FS, Hyer LA: Comorbidity and treatment outcome of inpatients with chronic combat-related PTSD. Hosp Community Psychiatry 1991; 42:847–849 [C]
- Davidson JET, Fairbank JA: The epidemiology of posttraumatic stress disorder, in Posttraumatic Stress Disorder: DSM-IV and Beyond. Edited by Davidson JET, Foa EB. Washington, DC, American Psychiatric Press, 1993, pp 147–169 [F]
- 250. Keane TM, Wolfe J: Comorbidity in post-traumatic stress disorder: an analysis of community and clinical studies. J Appl Soc Psychol 1990; 20:1776–1788 [F]
- 251. Jordan BK, Schlenger WE, Hough R, Kulka RA, Weiss D, Fairbank JA, Marmar CR: Lifetime and current prevalence of specific psychiatric disorders among Vietnam veterans and controls. Arch Gen Psychiatry 1991; 48:207–215 [D]
- Southwick SM, Yehuda R, Giller EL Jr: Personality disorders in treatment-seeking combat veterans with posttraumatic stress disorder. Am J Psychiatry 1993; 150:1020–1023 [G]
- 253. Solomon SD, Davidson JR: Trauma: prevalence, impairment, service use, and cost. J Clin Psychiatry 1997; 58(suppl 9):5–11 [F]
- 254. Breslau N, Davis GC, Andreski P, Peterson E: Traumatic events and posttraumatic stress disorder in an urban population of young adults. Arch Gen Psychiatry 1991; 48:216–222 [G]
- 255. Smith DW, Christiansen EH, Vincent R, Hann NE: Population effects of the bombing of Oklahoma City. J Okla State Med Assoc 1999; 92:193–198 [G]
- 256. Vlahov D, Galea S, Resnick H, Ahern J, Boscarino JA, Bucuvalas M, Gold J, Kilpatrick D: Increased use of cigarettes, alcohol, and marijuana among Manhattan, New York, residents after the September 11th terrorist attacks. Am J Epidemiol 2002; 155:988–996 [C]
- 257. Galea S, Ahern J, Resnick H, Kilpatrick D, Bucuvalas M, Gold J, Vlahov D: Psychological sequelae of the September 11 terrorist attacks in New York City. N Engl J Med 2002; 346:982–987 [C]
- 258. Goenjian A: A mental health relief programme in Armenia after the 1988 earthquake: implementation and clinical observations. Br J Psychiatry 1993; 163:230–239 [C]
- 259. Curtis T, Miller BC, Berry EH: Changes in reports and incidence of child abuse following natural disasters. Child Abuse Negl 2000; 24:1151–1162 [C]
- 260. Reijneveld SA, Crone MR, Verhulst FC, Verloove-Vanhorick SP: The effect of a severe disaster on the mental health of adolescents: a controlled study. Lancet 2003; 362:691–696 [D]
- 261. Kagan J: A conceptual analysis of the affects. J Am Psychoanal Assoc 1991; 39(suppl):109–130 [G]
- 262. Ornitz EM, Pynoos RS: Startle modulation in children with posttraumatic stress disorder. Am J Psychiatry 1989; 146:866–870 [C]

- van der Kolk BA, Fisler RE: Childhood abuse and neglect and loss of self-regulation. Bull Menninger Clin 1994; 58:145–168 [G]
- 264. Briere J, Runtz M: Symptomatology associated with childhood sexual victimization in a nonclinical adult sample. Child Abuse Negl 1988; 12:51–59 [G]
- Hall RC, Tice L, Beresford TP, Wooley B, Hall AK: Sexual abuse in patients with anorexia nervosa and bulimia. Psychosomatics 1989; 30:73–79 [D]
- 266. Palmer RL, Oppenheimer R, Dignon A, Chaloner DA, Howells K: Childhood sexual experiences with adults reported by women with eating disorders: an extended series. Br J Psychiatry 1990; 156:699–703 [G]
- van der Kolk BA, Perry JC, Herman JL: Childhood origins of self-destructive behavior. Am J Psychiatry 1991; 148:1665–1671 [C]
- 268. Browne A, Finkelhor D: Impact of child sexual abuse: a review of the research. Psychol Bull 1986; 99:66–77 [F]
- 269. Amir M, Kaplan Z, Efroni R, Kotler M: Suicide risk and coping styles in posttraumatic stress disorder patients. Psychother Psychosom 1999; 68:76–81 [B]
- 270. Grieger TA, Fullerton CS, Ursano RJ: Posttraumatic stress disorder, alcohol use, and perceived safety after the terrorist attack on the Pentagon. Psychiatr Serv 2003; 54:1380–1382 [G]
- Lewis DO: Neuropsychiatric and experiential correlates of violent juvenile delinquency. Neuropsychol Rev 1990; 1:125–136 [F]
- 272. Lewis DO: From abuse to violence: psychophysiological consequences of maltreatment. J Am Acad Child Adolesc Psychiatry 1992; 31:383–391 [F]
- 273. Kessler RC: Posttraumatic stress disorder: the burden to the individual and to society. J Clin Psychiatry 2000; 61(suppl 5):4–12 [E]
- Molnar BE, Berkman LF, Buka SL: Psychopathology, childhood sexual abuse and other childhood adversities: relative links to subsequent suicidal behaviour in the US. Psychol Med 2001; 31:965–977 [F]
- 275. Kessler RC, Borges G, Walters EE: Prevalence of and risk factors for lifetime suicide attempts in the National Comorbidity Survey. Arch Gen Psychiatry 1999; 56:617–626 [C]
- 276. Allgulander C: Psychiatric aspects of suicidal behavior: anxiety disorders, in The International Handbook of Suicide and Attempted Suicide. Edited by Hawton K, van Herringen K. Chichester, UK, John Wiley & Sons, 2000, pp 179–192 [F]
- Institute of Medicine: Reducing Suicide: A National Imperative. Washington, DC, National Academies Press, 2002 [E]
- Seedat S, Lockhat R, Kaminer D, Zungu-Dirwayi N, Stein DJ: An open trial of citalopram in adolescents with post-traumatic stress disorder. Int Clin Psychopharmacol 2001; 16:21–25 [B]
- Londborg PD, Hegel MT, Goldstein S, Goldstein D, Himmelhoch JM, Maddock R, Patterson WM, Rausch J, Farfel GM: Sertraline treatment of posttraumatic stress disorder: results of 24 weeks of open-label continuation treatment. J Clin Psychiatry 2001; 62:325–331 [B]
- 280. Malik ML, Connor KM, Sutherland SM, Smith RD, Davison RM, Davidson JR: Quality of life and posttraumatic stress disorder: a pilot study assessing changes in SF-36 scores before and after treatment in a placebo-controlled trial of fluoxetine. J Trauma Stress 1999; 12:387–393 [A]
- 281. Cowdry RW, Gardner DL: Pharmacotherapy of borderline personality disorder: alprazolam, carbamazepine, trifluoperazine, and tranylcypromine. Arch Gen Psychiatry 1988; 45:111–119 [A]
- 282. Wickham EA, Reed JV: Lithium for the control of aggressive and self-mutilating behavior. Int Clin Psychopharmacol 1987; 2:181–190 [F]
- 283. Herman BH, Hammock MK, Arthur-Smith A, Egan J, Chatoor I, Werner A, Zelnik N: Naltrexone decreases self-injurious behavior. Ann Neurol 1987; 22:550–552 [B]

- 284. Linehan MM: Behavioral treatments of suicidal behaviors: definitional obfuscation and treatment outcomes. Ann N Y Acad Sci 1997; 836:302–328 [G]
- 285. Mellman L: Consequences of violence against women, in Women's Health: A Lifelong Guide. New York, Scientific American Digital, 1998 [G]
- 286. Ursano RJ, Fullerton CS, Epstein RS, Crowley B, Vance K, Kao TC, Baum A: Peritraumatic dissociation and posttraumatic stress disorder following motor vehicle accidents. Am J Psychiatry 1999; 156:1808–1810 [C]
- 287. Epstein RS, Fullerton CS, Ursano RJ: Posttraumatic stress disorder following an air disaster: a prospective study. Am J Psychiatry 1998; 155:934–938 [C]
- 288. Zatzick D: Posttraumatic stress, functional impairment, and service utilization after injury: a public health approach. Semin Clin Neuropsychiatry 2003; 8:149–157 [F]
- 289. Rynearson EK, Favell J: Manual for Criminal Death Support Groups. Seattle, Virginia Mason Medical Center, 2000 [F]
- 290. Prigerson HG, Shear MK, Jacobs SC, Reynolds CF 3rd, Maciejewski PK, Davidson JR, Rosenheck R, Pilkonis PA, Wortman CB, Williams JB, Widiger TA, Frank E, Kupfer DJ, Zisook S: Consensus criteria for traumatic grief: a preliminary empirical test. Br J Psychiatry 1999; 174:67–73 [G]
- 291. Shear MK, Smith-Caroff K: Traumatic loss and the syndrome of complicated grief. PTSD Research Quarterly 2002; 13:1–8 [G]
- 292. Mollica RF, Sarajlic N, Chernoff M, Lavelle J, Vukovic IS, Massagli MP: Longitudinal study of psychiatric symptoms, disability, mortality, and emigration among Bosnian refugees. JAMA 2001; 286:546–554 [C]
- 293. Zatzick DF, Jurkovich GJ, Gentilello L, Wisner D, Rivara FP: Posttraumatic stress, problem drinking, and functional outcomes after injury. Arch Surg 2002; 137:200–205 [C]
- 294. Soderstrom CA, Smith GS, Dischinger PC, McDuff DR, Hebel JR, Gorelick DA, Kerns TJ, Ho SM, Read KM: Psychoactive substance use disorders among seriously injured trauma center patients. JAMA 1997; 277:1769–1774 [F]
- 295. Dunn C, Zatzick D, Russo J, Rivara F, Roy-Byrne P, Ries R, Wisner D, Gentilello L: Hazardous drinking by trauma patients during the year after injury. J Trauma 2003; 54:707–712 [C]
- 296. Rivara FP, Jurkovich GJ, Gurney JG, Seguin D, Fligner CL, Ries R, Raisys VA, Copass M: The magnitude of acute and chronic alcohol abuse in trauma patients. Arch Surg 1993; 128:907–912 [C]
- 297. Brown PJ, Wolfe J: Substance abuse and post-traumatic stress disorder comorbidity. Drug Alcohol Depend 1994; 35:51–59 [F]
- 298. McFarlane AC: Epidemiological evidence about the relationship between PTSD and alcohol abuse: the nature of the association. Addict Behav 1998; 23:813–825 [E]
- 299. Jacobsen LK, Southwick SM, Kosten TR: Substance use disorders in patients with posttraumatic stress disorder: a review of the literature. Am J Psychiatry 2001; 158:1184–1190 [F]
- 300. Engel CC Jr, Ursano R, Magruder C, Tartaglione R, Jing Z, Labbate LA, Debakey S: Psychological conditions diagnosed among veterans seeking Department of Defense care for Gulf War-related health concerns. J Occup Environ Med 1999; 41:384–392 [G]
- 301. Zatzick DF, Russo JE, Katon W: Somatic, posttraumatic stress, and depressive symptoms among injured patients treated in trauma surgery. Psychosomatics 2003; 44:479–484 [C]
- 302. Schnurr PP, Green BL: Trauma and Health: Physical Health Consequences of Exposure to Extreme Stress. Washington, DC, American Psychological Association, 2004 [G]
- 303. Horowitz MJ, Siegel B, Holen A, Bonanno GA, Milbrath C, Stinson CH: Diagnostic criteria for complicated grief disorder. Am J Psychiatry 1997; 154:904–910 [C]
- 304. Layne CM, Pynoos RS, Saltzman WR, Arslanagic B, Black M, Savjak N, Popovic T, Durakovic E, Music M, Campara N, Djapo N, Houston R: Trauma/grief-focused group

- psychotherapy: school-based postwar intervention with traumatized Bosnian adolescents. Group Dynamics 2001; 5:277–290 [B]
- 305. Giaconia RM, Reinherz HZ, Silverman AB, Pakiz B, Frost AK, Cohen E: Traumas and posttraumatic stress disorder in a community population of older adolescents. J Am Acad Child Adolesc Psychiatry 1995; 34:1369–1380 [G]
- 306. Resnick HS, Kilpatrick DG, Dansky BS, Saunders BE, Best CL: Prevalence of civilian trauma and posttraumatic stress disorder in a representative national sample of women. J Consult Clin Psychol 1993; 61:984–991 [G]
- 307. Norris FH: Epidemiology of trauma: frequency and impact of different potentially traumatic events on different demographic groups. J Consult Clin Psychol 1992; 60:409–418 [G]
- 308. Helzer JE, Robins LN, McEvoy L: Post-traumatic stress disorder in the general population: findings of the Epidemiologic Catchment Area survey. N Engl J Med 1987; 317:1630–1634 [G]
- Freedman SA, Gluck N, Tuval-Mashiach R, Brandes D, Peri T, Shalev AY: Gender differences in responses to traumatic events: a prospective study. J Trauma Stress 2002; 15:407–413 [C]
- 310. Jenkins EJ, Bell CC: Exposure and response to community violence among children and adolescents, in Children in a Violent Society. Edited by Osofsky J. New York, Guilford, 1997, pp 9–31 [G]
- 311. US Department of Health and Human Services: Mental Health: Culture, Race, Ethnicity— A Supplement to Mental Health: Report of the Surgeon General. Rockville, Md, US Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Center for Mental Health Services, 2001 [G]
- 312. Greenfeld LA, Smith SK: American Indians and Crime. Washington, DC, US Department of Justice, Office of Justice Programs, Bureau of Justice Statistics, 1999 [G]
- Jones MC, Dauphinais P, Sack WH, Somervell PD: Trauma-related symptomatology among American Indian adolescents. J Trauma Stress 1997; 10:163–173 [G]
- 314. Robin RW, Chester B, Rasmussen JK, Jaranson JM, Goldman D: Prevalence and characteristics of trauma and posttraumatic stress disorder in a southwestern American Indian community. Am J Psychiatry 1997; 154:1582–1588 [G]
- 315. Fairbank JA, Friedman MJ, Southwick SM: Veterans of armed conflicts, in The Mental Health Consequences of Torture. Edited by Gerrity ET, Keane TM, Tuma F. New York, Kluwer Academic/Plenum, 2001, pp 121–131 [G]
- 316. McLeod JD, Kessler RC: Socioeconomic status differences in vulnerability to undesirable life events. J Health Soc Behav 1990; 31:162–172 [C]
- 317. Bremner JD, Southwick SM, Johnson DR, Yehuda R, Charney DS: Childhood physical abuse and combat-related posttraumatic stress disorder in Vietnam veterans. Am J Psychiatry 1993; 150:235–239 [D]
- 318. Matsunaga Vietnam Veterans Project. White River Junction, Vt, National Center for Post-Traumatic Stress Disorder and the National Center for American Indian and Alaska Native Mental Health Research, 1996 [C]
- 319. Beals J, Manson SM, Shore JH, Friedman M, Ashcraft M, Fairbank JA, Schlenger WE: The prevalence of posttraumatic stress disorder among American Indian Vietnam veterans: disparities and context. J Trauma Stress 2002; 15:89–97 [F]
- 320. Ruef AM, Litz BT, Schlenger WE: Hispanic ethnicity and risk for combat-related posttraumatic stress disorder. Cultur Divers Ethnic Minor Psychol 2000; 6:235–251 [F]
- 321. Ortega AN, Rosenheck R: Posttraumatic stress disorder among Hispanic Vietnam veterans. Am J Psychiatry 2000; 157:615–619 [E]
- 322. Schnyder U, Moergeli H, Klaghofer R, Buddeberg C: Incidence and prediction of post-traumatic stress disorder symptoms in severely injured accident victims. Am J Psychiatry 2001; 158:594–599 [C]

- 323. Malt U: The long-term psychiatric consequences of accidental injury: a longitudinal study of 107 adults. Br J Psychiatry 1988; 153:810–818 [C]
- 324. Harvey AG, Bryant RA: The relationship between acute stress disorder and posttraumatic stress disorder: a prospective evaluation of motor vehicle accident survivors. J Consult Clin Psychol 1998; 66:507–512 [C]
- 325. Shalev AY, Peri T, Canetti L, Schreiber S: Predictors of PTSD in injured trauma survivors: a prospective study. Am J Psychiatry 1996; 153:219–225 [C]
- 326. Mayou R, Bryant B, Duthie R: Psychiatric consequences of road traffic accidents. BMJ 1993; 307:647–651 [C]
- 327. Michaels AJ, Michaels CE, Moon CH, Smith JS, Zimmerman MA, Taheri PA, Peterson C: Posttraumatic stress disorder after injury: impact on general health outcome and early risk assessment. J Trauma 1999; 47:460–466 [C]
- 328. Mellman TA, David D, Bustamante V, Fins AI, Esposito K: Predictors of post-traumatic stress disorder following severe injury. Depress Anxiety 2001; 14:226–231 [C]
- 329. O'Donnell ML, Creamer M, Bryant RA, Schnyder U, Shalev A: Posttraumatic disorders following injury: empirical and methodological review. Clin Psychol Rev 2003; 23:587–603 [E]
- 330. Bleich A, Gelkopf M, Solomon Z: Exposure to terrorism, stress-related mental health symptoms, and coping behaviors among a nationally representative sample in Israel. JAMA 2003; 290:612–620 [G]
- 331. Sabin M, Lopes Cardozo B, Nackerud L, Kaiser R, Varese L: Factors associated with poor mental health among Guatemalan refugees living in Mexico 20 years after civil conflict. JAMA 2003; 290:635–642 [G]
- 332. de Jong JT, Komproe IH, Van Ommeren M, El Masri M, Araya M, Khaled N, van De Put W, Somasundaram D: Lifetime events and posttraumatic stress disorder in 4 postconflict settings. JAMA 2001; 286:555–562 [C]
- 333. Kinzie JD, Boehnlein JK, Leung PK, Moore LJ, Riley C, Smith D: The prevalence of posttraumatic stress disorder and its clinical significance among Southeast Asian refugees. Am J Psychiatry 1990; 147:913–917 [G]
- 334. US Department of Health and Human Services: Mental Health: A Report of the Surgeon General. Rockville, Md, US Department of Health and Human Services, 1999 [C]
- 335. Kroll J, Habenicht M, Mackenzie T, Yang M, Chan S, Vang T, Nguyen T, Ly M, Phommasouvanh B, Nguyen H, Vang Y, Souvannasoth L, Cabugao R: Depression and posttraumatic stress disorder in Southeast Asian refugees. Am J Psychiatry 1989; 146:1592–1597 [C]
- 336. Blair RG: Risk factors associated with PTSD and major depression among Cambodian refugees in Utah. Health Soc Work 2000; 25:23–30 [C]
- 337. Caspi Y, Poole C, Mollica RF, Frankel M: Relationship of child loss to psychiatric and functional impairment in resettled Cambodian refugees. J Nerv Ment Dis 1998; 186:484–491 [G]
- 338. Kinzie JD, Sack W, Angell R, Clarke G, Ben R: A three-year follow-up of Cambodian young people traumatized as children. J Am Acad Child Adolesc Psychiatry 1989; 28:501–504 [C]
- 339. Chung RC, Kagawa-Singer M: Predictors of psychological distress among Southeast Asian refugees. Soc Sci Med 1993; 36:631–639 [G]
- 340. Farias P: Central and South American refugees: some mental health challenges, in Amidst Peril and Pain: The Mental Health and Well Being of the World's Refugees. Edited by Marsella AJ, Bornemann T, Ekblad S, Orley J. Washington, DC, American Psychological Association, 1994, pp 101–113 [G]
- 341. Cervantes RC, Salgado de Snyder VN, Padilla AM: Posttraumatic stress in immigrants from Central America and Mexico. Hosp Community Psychiatry 1989; 40:615–619 [D]
- 342. Michultka D, Blanchard EB, Kalous T: Responses to civilian war experiences: predictors of psychological functioning and coping. J Trauma Stress 1998; 11:571–577 [G]

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- 343. Arroyo W, Eth S: Children traumatized by Central American warfare, in Posttraumatic Stress Disorder in Children. Edited by Eth S, Pynoos RS. Washington, DC, American Psychiatric Press, 1984, pp 103–117 [G]
- 344. Eisenman DP, Gelberg L, Liu H, Shapiro MF: Mental health and health-related quality of life among adult Latino primary care patients living in the United States with previous exposure to political violence. JAMA 2003; 290:627–634 [G]
- 345. Blanchard EB, Hickling EJ, Barton KA, Taylor AE, Loos WR, Jones-Alexander J: One-year prospective follow-up of motor vehicle accident victims. Behav Res Ther 1996; 34:775–786 [C]
- 346. Tedeschi RG, Calhoun LG: The Posttraumatic Growth Inventory: measuring the positive legacy of trauma. J Trauma Stress 1996; 9:455–471 [G]
- 347. Herman JL, Perry JC, van der Kolk BA: Childhood trauma in borderline personality disorder. Am J Psychiatry 1989; 146:490–495 [D]
- 348. Perry JC, Herman HL, van der Kolk BA, Holk LA: Psychotherapy and psychological trauma in borderline personality disorder. Psychiatr Ann 1990; 20:33–43 [G]
- 349. Pollack MH, Otto MW, Rosenbaum JF, Sachs GS: Personality disorders in patients with panic disorder: association with childhood anxiety disorders, early trauma, comorbidity, and chronicity. Compr Psychiatry 1992; 33:78–83 [C]
- 350. Zatzick DF, Weiss DS, Marmar CR, Metzler TJ, Wells K, Golding JM, Stewart A, Schlenger WE, Browner WS: Post-traumatic stress disorder and functioning and quality of life outcomes in female Vietnam veterans. Mil Med 1997; 162:661–665 [C]
- Mendlowicz MV, Stein MB: Quality of life in individuals with anxiety disorders. Am J Psychiatry 2000; 157:669–682 [F]
- 352. Bryant RA, Marosszeky JE, Crooks J, Baguley IJ, Gurka JA: Posttraumatic stress disorder and psychosocial functioning after severe traumatic brain injury. J Nerv Ment Dis 2001; 189:109–113 [C]
- 353. Zayfert C, Dums AR, Ferguson RJ, Hegel MT: Health functioning impairments associated with posttraumatic stress disorder, anxiety disorders, and depression. J Nerv Ment Dis 2002; 190:233–240 [C]
- 354. Beckham JC, Moore SD, Feldman ME, Hertzberg MA, Kirby AC, Fairbank JA: Health status, somatization, and severity of posttraumatic stress disorder in Vietnam combat veterans with posttraumatic stress disorder. Am J Psychiatry 1998; 155:1565–1569 [C]
- 355. Boscarino JA: Diseases among men 20 years after exposure to severe stress: implications for clinical research and medical care. Psychosom Med 1997; 59:605–614 [C]
- 356. Greenberg PE, Sisitsky T, Kessler RC, Finkelstein SN, Berndt ER, Davidson JR, Ballenger JC, Fyer AJ: The economic burden of anxiety disorders in the 1990s. J Clin Psychiatry 1999; 60:427–435 [E]
- 357. Livanou M, Basoglu M, Marks IM, De SP, Noshirvani H, Lovell K, Thrasher S: Beliefs, sense of control and treatment outcome in post-traumatic stress disorder. Psychol Med 2002; 32:157–165 [A]
- 358. Krupnick JL: Brief psychodynamic treatment of PTSD. J Clin Psychol 2002; 58:919–932 [G]
- Plakun EM, Shapiro ER: Psychodynamic psychotherapy for PTSD. J Clin Psychiatry 2000;
 61:787–788 [F]
- 360. Lansky MR: The transformation of affect in posttraumatic nightmares. Bull Menninger Clin 1991; 55:470–490 [G]
- Peniston EG: EMG biofeedback-assisted desensitization treatment for Vietnam combat veterans' post-traumatic stress disorder. Clinical Biofeedback and Health 1986; 9:35–41 [A–]
- 362. Keane TM, Fairbank JA, Caddell JM, Zimering RT: Implosive (flooding) therapy reduces symptoms of PTSD in Vietnam combat veterans. Behav Ther 1989; 20:245–260 [A–]
- 363. Cooper NA, Clum GA: Imaginal flooding as a supplementary treatment for PTSD in combat veterans: a controlled study. Behav Ther 1989; 20:381–391 [B]

- 364. Boudewyns PA, Hyer L: Physiological response to combat memories and preliminary treatment outcome in Vietnam veteran PTSD patients treated with direct therapeutic exposure. Behav Ther 1990; 21:63–87 [A–]
- 365. Boudewyns PA, Hyer L, Woods MG, Harrison WR, McCranie E: PTSD among Vietnam veterans: an early look at treatment outcome using direct therapeutic exposure. J Trauma Stress 1990; 3:359–368 [B]
- 366. Richards DA, Lovell K, Marks IM: Post-traumatic stress disorder: evaluation of a behavioral treatment program. J Trauma Stress 1994; 7:669–680 [A–]
- 367. Rothbaum BO, Hodges LF: The use of virtual reality exposure in the treatment of anxiety disorders. Behav Modif 1999; 23:507–525 [G]
- 368. Rothbaum BO, Hodges LF, Ready D, Graap K, Alarcon RD: Virtual reality exposure therapy for Vietnam veterans with posttraumatic stress disorder. J Clin Psychiatry 2001; 62:617–622 [B]
- 369. Krakow B, Hollifield M, Schrader R, Koss M, Tandberg D, Lauriello J, McBride L, Warner TD, Cheng D, Edmond T, Kellner R: A controlled study of imagery rehearsal for chronic nightmares in sexual assault survivors with PTSD: a preliminary report. J Trauma Stress 2000; 13:589–609 [A–]
- 370. Forbes D, Phelps AJ, McHugh AF, Debenham P, Hopwood M, Creamer M: Imagery rehearsal in the treatment of posttraumatic nightmares in Australian veterans with chronic combat-related PTSD: 12-month follow-up data. J Trauma Stress 2003; 16:509–513 [B]
- 371. Falsetti SA, Resnick HS, Davis HJ, Gallagher NG: Treatment of posttraumatic stress disorder with comorbid panic attacks: combining cognitive processing therapy with panic control treatment techniques. Group Dynamics 2001; 5:252–260 [A]
- 372. Glynn SM, Eth S, Randolph ET, Foy DW, Urbaitis M, Boxer L, Paz GG, Leong GB, Firman G, Salk JD, Katzman JW, Crothers J: A test of behavioral family therapy to augment exposure for combat-related posttraumatic stress disorder. J Consult Clin Psychol 1999; 67:243–251 [A–]
- 373. Fecteau G, Nicki R: Cognitive behavioural treatment of posttraumatic stress disorder after motor vehicle accident. Behavioural and Cognitive Psychotherapy 1999; 27:201–214 [E]
- 374. Foa EB, Rothbaum BO, Riggs DS, Murdock TB: Treatment of posttraumatic stress disorder in rape victims: a comparison between cognitive-behavioral procedures and counseling. J Consult Clin Psychol 1991; 59:715–723 [A]
- 375. Echeburua E, de Corral P, Zubizarreta I, Sarasua B: Psychological treatment of chronic posttraumatic stress disorder in victims of sexual aggression. Behav Modif 1997; 21:433–456 [A–]
- 376. Silver SM, Brooks A, Obenchain J: Treatment of Vietnam War veterans with PTSD: a comparison of eye movement desensitization and reprocessing, biofeedback, and relaxation training. J Trauma Stress 1995; 8:337–342 [B]
- 377. Paunovic N, Ost LG: Cognitive-behavior therapy vs exposure therapy in the treatment of PTSD in refugees. Behav Res Ther 2001; 39:1183–1197 [A–]
- 378. Chemtob CM, Novaco RW, Hamada RS, Gross DM: Cognitive-behavioral treatment for severe anger in posttraumatic stress disorder. J Consult Clin Psychol 1997; 65:184–189 [A–]
- 379. 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 [B]
- 380. Gillespie K, Duffy M, Hackmann A, Clark DM: Community based cognitive therapy in the treatment of posttraumatic stress disorder following the Omagh bomb. Behav Res Ther 2002; 40:345–357 [B]
- 381. Ehlers A, Clark D: Early psychological interventions for adult survivors of trauma: a review. Biol Psychiatry 2003; 53:817–826 [F]

- 382. Marcus SV: Controlled study of treatment of PTSD using EMDR in an HMO setting. Psychotherapy 1997; 34:307–315 [A–]
- 383. Rothbaum BO: A controlled study of eye movement desensitization and reprocessing in the treatment of posttraumatic stress disordered sexual assault victims. Bull Menninger Clin 1997; 61:317–334 [A–]
- 384. Scheck MM, Schaeffer JA, Gillette C: Brief psychological intervention with traumatized young women: the efficacy of eye movement desensitization and reprocessing. J Trauma Stress 1998; 11:25–44 [A–]
- 385. Wilson SA, Becker LA, Tinker RH: Eye movement desensitization and reprocessing (EMDR) treatment for psychologically traumatized individuals. J Consult Clin Psychol 1995; 63:928–937 [A–]
- 386. Wilson SA, Becker LA, Tinker RH: Fifteen-month follow-up of eye movement desensitization and reprocessing (EMDR) treatment for posttraumatic stress disorder and psychological trauma. J Consult Clin Psychol 1997; 65:1047–1056 [C]
- 387. Ironson G, Freund B, Strauss JL, Williams J: Comparison of two treatments for traumatic stress: a community-based study of EMDR and prolonged exposure. J Clin Psychol 2002; 58:113–128 [A–]
- 388. Devilly GJ, Spence SH, Rapee RM: Statistical and reliable change with eye movement desensitization and reprocessing: treating trauma within a veteran population. Behav Ther 1998; 29:435–455 [A]
- 389. Macklin ML, Metzger LJ, Lasko NB, Berry NJ, Orr SP, Pitman RK: Five-year follow-up study of eye movement desensitization and reprocessing therapy for combat-related post-traumatic stress disorder. Compr Psychiatry 2000; 41:24–27 [B]
- 390. Cusack K, Spates CR: The cognitive dismantling of eye movement desensitization and reprocessing (EMDR) treatment of posttraumatic stress disorder (PTSD). J Anxiety Disord 1999; 13:87–99 [A–]
- 391. Classen C, Butler LD, Koopman C, Miller E, DiMiceli S, Giese-Davis J, Fobair P, Carlson RW, Kraemer HC, Spiegel D: Supportive-expressive group therapy and distress in patients with metastatic breast cancer: a randomized clinical intervention trial. Arch Gen Psychiatry 2001; 58:494–501 [F]
- 392. Zlotnick C, Shea TM, Rosen K, Simpson E, Mulrenin K, Begin A, Pearlstein T: An affect-management group for women with posttraumatic stress disorder and histories of childhood sexual abuse. J Trauma Stress 1997; 10:425–436 [A]
- 393. Schnurr PP, Friedman MJ, Foy DW, Shea MT, Hsieh FY, Lavori PW, Glynn SM, Wattenberg M, Bernardy NC: Randomized trial of trauma-focused group therapy for posttraumatic stress disorder: results from a Department of Veterans Affairs cooperative study. Arch Gen Psychiatry 2003; 60:481–489 [A–]
- 394. Zoellner LA, Feeny NC, Cochran B, Pruitt L: Treatment choice for PTSD. Behav Res Ther 2003; 41:879–886 [C]
- Foa EB, Zoellner LA, Feeny NC, Hembree EA, Alvarez-Conrad J: Does imaginal exposure exacerbate PTSD symptoms? J Consult Clin Psychol 2002; 70:1022–1028 [D]
- 396. Bradley RG, Follingstad DR: Group therapy for incarcerated women who experienced interpersonal violence: a pilot study. J Trauma Stress 2003; 16:337–340 [A–]
- 397. Drozdek B: Follow-up study of concentration camp survivors from Bosnia-Herzegovina: three years later. J Nerv Ment Dis 1997; 185:690–694 [G]
- 398. Cloitre M, Koenen KC: The impact of borderline personality disorder on process group outcome among women with posttraumatic stress disorder related to childhood abuse. Int J Group Psychother 2001; 51:379–398 [A–]
- 399. Vaa G, Egner R, Sexton H: Sexually abused women after multimodal group therapy: a long-term follow-up study. Nord J Psychiatry 2002; 56:215–221 [B]

- 400. Vandeusen KM, Carr JL: Recovery from sexual assault: an innovative two-stage group therapy model. Int J Group Psychother 2003; 53:201–223 [G]
- 401. Busuttil W, Turnbull GJ, Neal LA, Rollins J, West AG, Blanch N, Herepath R: Incorporating psychological debriefing techniques within a brief group psychotherapy programme for the treatment of post-traumatic stress disorder. Br J Psychiatry 1995; 167:495–502 [B]
- 402. Murphy SA: A bereavement intervention for parents following the sudden, violent deaths of their 12–28-year-old children: description and applications to clinical practice. Can J Nurs Res 1997; 29:51–72 [B]
- 403. Foy DW, Glynn SM, Schnurr PP, Jankowski MK, Wattenberg MS, Weiss DS, Marmar CR, Gusman GD: Group psychotherapy, in Effective Treatments for PTSD: Practical Guidelines From the International Society for Traumatic Stress Studies. Edited by Foa EB, Keane TM, Friedman MJ. New York, Guilford, 2000, pp 155–175 [F]
- 404. Cohen JA, Mannarino AP: A treatment outcome study for sexually abused preschool children: initial findings. J Am Acad Child Adolesc Psychiatry 1996; 35:42–50 [A]
- 405. Cohen JA, Mannarino AP: Factors that mediate treatment outcome of sexually abused preschool children: six- and 12-month follow-up. J Am Acad Child Adolesc Psychiatry 1998: 37:44–51 [B]
- 406. March JS, Amaya-Jackson L, Murray MC, Schulte A: Cognitive-behavioral psychotherapy for children and adolescents with posttraumatic stress disorder after a single-incident stressor. J Am Acad Child Adolesc Psychiatry 1998; 37:585–593 [B]
- 407. Saltzman WR, Pynoos RS, Layne CM, Steinberg A, Aisenberg E: School-based trauma/grief group psychotherapy program for youth exposed to community violence. Group Dynamics 2001; 5:291–303 [B]
- 408. Davies DR, Burlingame GM, Layne CM: Integrating small group process principles into trauma-focused group psychotherapy: what should a group trauma therapist know? in Group Approaches for Psychological Effects of Terrorist Disasters. Edited by Schein LA, Burlingame GM, Spitz HI, Muskin PR. New York, Haworth (in press) [G]
- 409. Rose S, Brewin CR, Andrews B, Kirk M: A randomized controlled trial of individual psychological debriefing for victims of violent crime. Psychol Med 1999; 29:793–799 [A–]
- 410. Carlier IV, Voerman AE, Gersons BP: The influence of occupational debriefing on post-traumatic stress symptomatology in traumatized police officers. Br J Med Psychol 2000; 73(pt 1):87–98 [A]
- 411. Deahl M, Srinivasan M, Jones N, Thomas J, Neblett C, Jolly A: Preventing psychological trauma in soldiers: the role of operational stress training and psychological debriefing. Br J Med Psychol 2000; 73(pt 1):77–85 [A]
- 412. Campfield KM, Hills AM: Effect of timing of critical incident stress debriefing (CISD) on posttraumatic symptoms. J Trauma Stress 2001; 14:327–340 [B]
- 413. Lange A, van de Ven JP, Schrieken B, Emmelkamp PM: Interapy, treatment of posttraumatic stress through the Internet: a controlled trial. J Behav Ther Exp Psychiatry 2001; 32:73–90 [A]
- 414. Lange A, Rietdijk D, Hudcovicova M, van de Ven JP, Schrieken B, Emmelkamp PM: Interapy: a controlled randomized trial of the standardized treatment of posttraumatic stress through the Internet. J Consult Clin Psychol 2003; 71:901–909 [A]
- 415. Ragsdale KG, Cox RD, Finn P, Eisler RM: Effectiveness of short-term specialized inpatient treatment for war-related posttraumatic stress disorder: a role for adventure-based counseling and psychodrama. J Trauma Stress 1996; 9:269–283 [B]
- 416. Hyer L, Boyd S, Scurfield R, Smith D, Burke J: Effects of Outward Bound experience as an adjunct to inpatient PTSD treatment of war veterans. J Clin Psychol 1996; 52:263–278 [B]
- 417. Gidron Y, Peri T, Connolly JF, Shalev AY: Written disclosure in posttraumatic stress disorder: is it beneficial for the patient? J Nerv Ment Dis 1996; 184:505–507 [A]
- 418. Reynolds M, Tarrier N: Monitoring of intrusions in post-traumatic stress order: a report of single case studies. Br J Med Psychol 1996; 69(pt 4):371–379 [G]

- 419. Silver SM, Wilson JP: Native American healing and purification rituals for war stress, in Human Adaptation to Extreme Stress: From the Holocaust to Vietnam. Edited by Wilson JP, Kahana B. New York, Plenum, 1989, pp 337–355 [G]
- 420. Zatzick DF, Johnson FA: Alternative psychotherapeutic practice among middle class Americans, I: case studies and follow-up. Cult Med Psychiatry 1997; 21:53–88 [G]
- 421. Davidson JR, Rothbaum BO, van der Kolk BA, Sikes CR, Farfel GM: Multicenter, doubleblind comparison of sertraline and placebo in the treatment of posttraumatic stress disorder. Arch Gen Psychiatry 2001; 58:485–492 [A]
- 422. Davis LL, Nugent AL, Murray J, Kramer GL, Petty F: Nefazodone treatment for chronic posttraumatic stress disorder: an open trial. J Clin Psychopharmacol 2000; 20:159–164 [B]
- 423. Hidalgo R, Hertzberg MA, Mellman T, Petty F, Tucker P, Weisler R, Zisook S, Chen S, Churchill E, Davidson J: Nefazodone in post-traumatic stress disorder: results from six open-label trials. Int Clin Psychopharmacol 1999; 14:61–68 [E]
- 424. Zisook S, Chentsova-Dutton YE, Smith-Vaniz A, Kline NA, Ellenor GL, Kodsi AB, Gillin JC: Nefazodone in patients with treatment-refractory posttraumatic stress disorder. J Clin Psychiatry 2000; 61:203–208 [B]
- 425. Neylan TC, Lenoci M, Maglione ML, Rosenlicht NZ, Leykin Y, Metzler TJ, Schoenfeld FB, Marmar CR: The effect of nefazodone on subjective and objective sleep quality in posttraumatic stress disorder. J Clin Psychiatry 2003; 64:445–450 [B]
- 426. Mellman TA, David D, Barza L: Nefazodone treatment and dream reports in chronic PTSD. Depress Anxiety 1999; 9:146–148 [B]
- 427. Hertzberg MA, Feldman ME, Beckham JC, Davidson JR: Trial of trazodone for posttraumatic stress disorder using a multiple baseline group design. J Clin Psychopharmacol 1996; 16:294–298 [B]
- 428. Canive JM, Clark RD, Calais LA, Qualls C, Tuason VB: Bupropion treatment in veterans with posttraumatic stress disorder: an open study. J Clin Psychopharmacol 1998; 18:379–383 [B]
- 429. Smajkic A, Weine S, Djuric-Bijedic Z, Boskailo E, Lewis J, Pavkovic I: Sertraline, paroxetine, and venlafaxine in refugee posttraumatic stress disorder with depression symptoms. J Trauma Stress 2001; 14:445–452 [B]
- 430. Connor KM, Davidson JR, Weisler RH, Ahearn E: A pilot study of mirtazapine in post-traumatic stress disorder. Int Clin Psychopharmacol 1999; 14:29–31 [B]
- 431. Davidson JR, Weisler RH, Butterfield MI, Casat CD, Connor KM, Barnett S, van Meter S: Mirtazapine vs placebo in posttraumatic stress disorder: a pilot trial. Biol Psychiatry 2003; 53:188–191 [A]
- 432. Southwick SM, Yehuda R, Giller EL Jr, Charney DS: Use of tricyclics and monoamine oxidase inhibitors in the treatment of PTSD: a quantitative review, in Catecholamine Function in Post-Traumatic Stress Disorder: Emerging Concepts. Edited by Murburg MM. Washington, DC, American Psychiatric Press, 1994, pp 293–305 [E]
- 433. Robert R, Blakeney PE, Villarreal C, Rosenberg L, Meyer WJ 3rd: Imipramine treatment in pediatric burn patients with symptoms of acute stress disorder: a pilot study. J Am Acad Child Adolesc Psychiatry 1999; 38:873–882 [A]
- 434. Neal LA, Shapland W, Fox C: An open trial of moclobemide in the treatment of post-traumatic stress disorder. Int Clin Psychopharmacol 1997; 12:231–237 [B]
- 435. Katz RJ, Lott MH, Arbus P, Crocq L, Herlobsen P, Lingjaerde O, Lopez G, Loughrey GC, MacFarlane DJ, McIvor R, Mehlum L, Nugent D, Turner SW, Weisaeth L, Yule W: Pharmacotherapy of post-traumatic stress disorder with a novel psychotropic. Anxiety 1994–1995; 1:169–174 [A]
- 436. Connor KM, Hidalgo RB, Crockett B, Malik M, Katz RJ, Davidson JR: Predictors of treatment response in patients with posttraumatic stress disorder. Prog Neuropsychopharmacol Biol Psychiatry 2001; 25:337–345 [E]

- 437. Braun P, Greenberg D, Dasberg H, Lerer B: Core symptoms of posttraumatic stress disorder unimproved by alprazolam treatment. J Clin Psychiatry 1990; 51:236–238 [A]
- 438. Mellman TA, Byers PM, Augenstein JS: Pilot evaluation of hypnotic medication during acute traumatic stress response. J Trauma Stress 1998; 11:563–569 [B]
- 439. Duffy JD, Malloy PF: Efficacy of buspirone in the treatment of posttraumatic stress disorder: an open trial. Ann Clin Psychiatry 1994; 6:33–37 [B]
- 440. Wells BG, Chu CC, Johnson R, Nasdahl C, Ayubi MA, Sewell E, Statham P: Buspirone in the treatment of posttraumatic stress disorder. Pharmacotherapy 1991; 11:340–343 [B]
- 441. Raskind MA, Thompson C, Petrie EC, Dobie DJ, Rein RJ, Hoff DJ, McFall ME, Peskind ER: Prazosin reduces nightmares in combat veterans with posttraumatic stress disorder. J Clin Psychiatry 2002; 63:565–568 [B]
- 442. Jacobs-Rebhun S, Schnurr PP, Friedman MJ, Peck R, Brophy M, Fuller D: Posttraumatic stress disorder and sleep difficulty. Am J Psychiatry 2000; 157:1525–1526 [A]
- 443. Clark RD, Canive JM, Calais LA, Qualls C, Brugger RD, Vosburgh TB: Cyproheptadine treatment of nightmares associated with posttraumatic stress disorder. J Clin Psychopharmacol 1999; 19:486–487 [B]
- 444. Kaplan Z, Amir M, Swartz M, Levine J: Inositol treatment of post-traumatic stress disorder.

 Anxiety 1996; 2:51–52 [A]
- 445. Schelling G: Effects of stress hormones on traumatic memory formation and the development of posttraumatic stress disorder in critically ill patients. Neurobiol Learn Mem 2002; 78:596–609 [E]
- 446. Schelling G, Briegel J, Roozendaal B, Stoll C, Rothenhausler HB, Kapfhammer HP: The effect of stress doses of hydrocortisone during septic shock on posttraumatic stress disorder in survivors. Biol Psychiatry 2001; 50:978–985 [B]
- 447. Saxe G, Stoddard F, Courtney D, Cunningham K, Chawla N, Sheridan R, King D, King L: Relationship between acute morphine and the course of PTSD in children with burns. J Am Acad Child Adolesc Psychiatry 2001; 40:915–921 [D]
- 448. Ehlers A, Clark DM, Hackmann A, McManus F, Fennell M, Herbert C, Mayou R: A randomized controlled trial of cognitive therapy, a self-help booklet, and repeated assessments as early interventions for posttraumatic stress disorder. Arch Gen Psychiatry 2003; 60:1024–1032 [A–]
- 449. Heim C, Newport DJ, Heit S, Graham YP, Wilcox M, Bonsall R, Miller AH, Nemeroff CB: Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. JAMA 2000; 284:592–597 [G]
- 450. Heim C, Nemeroff CB: The role of childhood trauma in the neurobiology of mood and anxiety disorders: preclinical and clinical studies. Biol Psychiatry 2001; 49:1023–1039 [F]
- 451. Charney DS: Psychobiological mechanisms of resilience and vulnerability: implications for successful adaptation to extreme stress. Am J Psychiatry 2004; 161:195–216 [G]
- 452. Zlotnick C, Franklin CL, Zimmerman M: Does "subthreshold" posttraumatic stress disorder have any clinical relevance? Compr Psychiatry 2002; 43:413–419 [D]
- 453. Weiss DS, Marmar CR, Schlenger WE, Fairbank JA, Jordan BK, Hough RL, Kulka RA: The prevalence of lifetime and partial post-traumatic stress disorder in Vietnam theater veterans. J Trauma Stress 1992; 5:365–376 [D]
- 454. Schutzwohl M, Maercker A: Effects of varying diagnostic criteria for posttraumatic stress disorder are endorsing the concept of partial PTSD. J Trauma Stress 1999; 12:155–165 [G]
- 455. Stein MB, Walker JR, Hazen AL, Forde DR: Full and partial posttraumatic stress disorder: findings from a community survey. Am J Psychiatry 1997; 154:1114–1119 [G]
- 456. Shear MK, Zuckoff A, Frank E: The syndrome of traumatic grief. CNS Spectrums 2001; 6:339–346 [G]
- 457. Taylor FB: Tiagabine for posttraumatic stress disorder: a case series of 7 women. J Clin Psychiatry 2003; 64:1421–1425 [G]

- 458. Foa EB, Rothbaum BO, Furr JM: Augmenting exposure therapy with other CBT procedures. Psychiatr Ann 2003; 33:47–53 [F]
- 459. Ross RJ, Ball WA, Dinges DF, Kribbs NB, Morrison AR, Silver SM, Mulvaney FD: Rapid eye movement sleep disturbance in posttraumatic stress disorder. Biol Psychiatry 1994; 35:195–202 [D]
- 460. Ross RJ, Ball WA, Sullivan KA, Caroff SN: Sleep disturbance as the hallmark of posttraumatic stress disorder. Am J Psychiatry 1989; 146:697–707 [G]
- 461. Pitman RK, Orr SP, Shalev AY, Metzger LJ, Mellman TA: Psychophysiological alterations in post-traumatic stress disorder. Semin Clin Neuropsychiatry 1999; 4:234–241 [G]
- 462. Rothbaum BO, Mellman TA: Dreams and exposure therapy in PTSD. J Trauma Stress 2001; 14:481–490 [F]
- 463. Foa EB, Riggs DS, Massie ED, Yarczower M: The impact of fear activation and anger on the efficacy of exposure treatment for posttraumatic stress disorder. Behav Ther 1995; 26:487–499 [C]