

# PRACTICE GUIDELINE FOR THE Treatment of Patients With Acute Stress Disorder and Posttraumatic Stress Disorder

## WORK GROUP ON ASD AND PTSD

Robert J. Ursano, M.D., Chair  
Carl Bell, M.D.  
Spencer Eth, M.D.  
Matthew Friedman, M.D., Ph.D.  
Ann Norwood, M.D.  
Betty Pfefferbaum, M.D., J.D.  
Robert S. Pynoos, M.D.  
Douglas F. Zatzick, M.D.  
David M. Benedek, M.D., Consultant

*Originally published in November 2004. A guideline watch, summarizing significant developments in the scientific literature since publication of this guideline, may be available in the Psychiatric Practice section of the APA web site at [www.psych.org](http://www.psych.org).*

---

This guideline is dedicated to Rebecca M. Thaler Schwebel (1972–2004), Senior Project Manager at APA when this guideline was initiated. Becca's humor, generous spirit, and optimism will be missed.

# AMERICAN PSYCHIATRIC ASSOCIATION STEERING COMMITTEE ON PRACTICE GUIDELINES

John S. McIntyre, M.D.,  
*Chair*

Sara C. Charles, M.D.,  
*Vice-Chair*

Daniel J. Anzia, M.D.  
Ian A. Cook, M.D.  
Molly T. Finnerty, M.D.  
Bradley R. Johnson, M.D.  
James E. Nininger, M.D.  
Paul Summergrad, M.D.  
Sherwyn M. Woods, M.D., Ph.D.  
Joel Yager, M.D.

## AREA AND COMPONENT LIAISONS

Robert Pyles, M.D. (Area I)  
C. Deborah Cross, M.D. (Area II)  
Roger Peele, M.D. (Area III)  
Daniel J. Anzia, M.D. (Area IV)  
John P. D. Shemo, M.D. (Area V)  
Lawrence Lurie, M.D. (Area VI)  
R. Dale Walker, M.D. (Area VII)  
Mary Ann Barnovitz, M.D.  
Sheila Hafter Gray, M.D.  
Sunil Saxena, M.D.  
Tina Tonnu, M.D.

## STAFF

Robert Kunkle, M.A., *Senior Program Manager*  
Amy B. Albert, B.A., *Assistant Project Manager*  
Laura J. Fochtman, M.D., *Medical Editor*  
Claudia Hart, *Director, Department of Quality Improvement and  
Psychiatric Services*  
Darrel A. Regier, M.D., M.P.H., *Director, Division of Research*

# CONTENTS

Statement of Intent .....	5
Guide to Using This Practice Guideline .....	6
Development Process .....	7
Introduction .....	9
<b>Part A: Treatment Recommendations .....</b>	<b>11</b>
I. Executive Summary .....	11
A. Coding System .....	11
B. Summary of Recommendations .....	11
II. Formulation and Implementation of a Treatment Plan .....	14
A. Initial Assessment .....	14
B. Principles of Psychiatric Management .....	21
C. Principles of Treatment Selection .....	26
D. Specific Treatment Strategies .....	29
III. Specific Clinical Features Influencing the Treatment Plan .....	34
A. Age .....	34
B. Gender .....	35
C. Ethnic and Cross-Cultural Factors .....	35
D. Medical and Other Psychiatric Comorbidity .....	36
E. History of Previous Traumas .....	37
F. Aggressive Behavior .....	38
G. Self-Injurious and Suicidal Behaviors .....	38
<b>Part B: Background Information and Review of Available Evidence .....</b>	<b>39</b>
IV. Disease Definition, Epidemiology, and Natural History .....	39
A. Core Clinical Features .....	39
B. Associated Features .....	43
C. Differential Diagnosis .....	43
D. Epidemiology .....	43
E. Natural History and Course .....	51
V. Review and Synthesis of Available Evidence .....	51
A. Issues in Interpreting the Literature .....	51
B. Psychosocial Interventions .....	52
C. Pharmacotherapies .....	63
<b>Part C: Future Research Needs .....</b>	<b>67</b>
Individuals and Organizations That Submitted Comments .....	70
References .....	71



## STATEMENT OF INTENT

The American Psychiatric Association (APA) Practice Guidelines are not intended to be construed or to serve as a standard of medical care. Standards of medical care are determined on the basis of all clinical data available for an individual patient and are subject to change as scientific knowledge and technology advance and practice patterns evolve. These parameters of practice should be considered guidelines only. Adherence to them will not ensure a successful outcome for every individual, nor should they be interpreted as including all proper methods of care or excluding other acceptable methods of care aimed at the same results. The ultimate judgment regarding a particular clinical procedure or treatment plan must be made by the psychiatrist in light of the clinical data presented by the patient and the diagnostic and treatment options available.

This practice guideline has been developed by psychiatrists who are in active clinical practice. In addition, some contributors are primarily involved in research or other academic endeavors. It is possible that through such activities some contributors, including work group members and reviewers, have received income related to treatments discussed in this guideline. A number of mechanisms are in place to minimize the potential for producing biased recommendations due to conflicts of interest. Work group members are selected on the basis of their expertise and integrity. Any work group member or reviewer who has a potential conflict of interest that may bias (or appear to bias) his or her work is asked to disclose this to the Steering Committee on Practice Guidelines and the work group. Iterative guideline drafts are reviewed by the Steering Committee, other experts, allied organizations, APA members, and the APA Assembly and Board of Trustees; substantial revisions address or integrate the comments of these multiple reviewers. The development of the APA practice guidelines is not financially supported by any commercial organization.

More detail about mechanisms in place to minimize bias is provided in a document available from the APA Department of Quality Improvement and Psychiatric Services, “APA Guideline Development Process.”

This practice guideline was approved in June 2004 and published in November 2004.

# GUIDE TO USING THIS PRACTICE GUIDELINE

The *Practice Guideline for the Treatment of Patients With Acute Stress Disorder and Posttraumatic Stress Disorder* consists of three parts (Parts A, B, and C) and many sections, not all of which will be equally useful for all readers. The following guide is designed to help readers find the sections that will be most useful to them.

Part A, “Treatment Recommendations,” is published as a supplement to *The American Journal of Psychiatry* and contains general and specific treatment recommendations. Section I summarizes the key recommendations of the guideline and codes each recommendation according to the degree of clinical confidence with which the recommendation is made. Section II provides further discussion of the formulation and implementation of a treatment plan as it applies to the individual patient. Section III, “Specific Clinical Features Influencing the Treatment Plan,” discusses a range of clinical considerations that could alter the general recommendations discussed in Section I.

Part B, “Background Information and Review of Available Evidence,” and Part C, “Future Research Needs,” are not included in *The American Journal of Psychiatry* supplement but are provided with Part A in the complete guideline, which is available in print format, in guideline compendiums, from American Psychiatric Publishing, Inc. (<http://www.appi.org>), and online through the American Psychiatric Association (<http://www.psych.org>). Part B provides an overview of ASD and PTSD, including general information on natural history, course, and epidemiology. It also provides a structured review and synthesis of the evidence that underlies the recommendations made in Part A. Part C draws from the previous sections and summarizes areas for which more research data are needed to guide clinical decisions.

To share feedback on this or other published APA practice guidelines, a form is available at [http://www.psych.org/psych\\_pract/pg/reviewform.cfm](http://www.psych.org/psych_pract/pg/reviewform.cfm).

## DEVELOPMENT PROCESS

This practice guideline was developed under the auspices of the Steering Committee on Practice Guidelines. The development process is detailed in a document available from the APA Department of Quality Improvement and Psychiatric Services: the “APA Guideline Development Process.” Key features of this process include the following:

- A comprehensive literature review to identify all relevant randomized clinical trials as well as less rigorously designed clinical trials and case series when evidence from randomized trials was unavailable.
- Development of evidence tables that reviewed the key features of each identified study, including funding source, study design, sample sizes, subject characteristics, treatment characteristics, and treatment outcomes.
- Initial drafting of the guideline by a work group that included psychiatrists with clinical and research expertise in ASD and PTSD.
- Production of multiple revised drafts with widespread review; 11 organizations and 55 individuals submitted significant comments.
- Approval by the APA Assembly and Board of Trustees.
- Planned revisions at regular intervals.

Relevant literature was identified through a computerized search of MEDLINE and the Published International Literature on Traumatic Stress (PILOTS) database, produced by the National Center for Post-Traumatic Stress Disorder and available online (<http://www.ncptsd.org/publications/pilots/index.html>). An initial search of PubMed was conducted for the period from 1966 to 2002. Key words used were posttraumatic stress, stress disorder, acute stress disorder, posttraumatic stress disorder, and PTSD. Additional citations were identified by using key words emotional trauma, psychic trauma, posttraumatic, disaster, terrorism, rape, assault, physical abuse, sexual abuse, childhood abuse, combat, traumatic event, and traumatic incident and then limited to citations that included the key words stress, psychological sequelae, anxiety, and dissociation. In determining which of the identified citations related to treatment, key words used were treatment, management, therapy, psychotherapy, antidepressive agents, tranquilizing agents, anticonvulsants, debriefing, critical incident, eye movement desensitization, and EMDR. Citations were further limited to clinical trials or meta-analyses published in the English language and accompanied by abstracts. A total of 316 citations were found. When applied to the PILOTS database, this search strategy yielded a total of 587 citations, many of which were duplicates of those obtained in the PubMed search. Additional, less formal literature searches were conducted by APA staff and individual work group members. Other published guidelines for the treatment of ASD and PTSD were also reviewed (1, 2).

This guideline presents recommendations for the evaluation and treatment of adult patients with ASD or PTSD. The *Practice Parameters for the Assessment and Treatment of Children and Adolescents With Posttraumatic Stress Disorder* of the American Academy of Child and Adolescent Psychiatry (3) may be consulted for guidelines relating to the evaluation and treatment of children and adolescents.

This document represents a synthesis of current scientific knowledge and rational clinical practice. It strives to be as free as possible of bias toward any theoretical approach to treatment. Articles identified in the initial literature search were prioritized for review according to meth-

odological strength. Highest priority was given to randomized, placebo-controlled trials of psychotherapeutic and psychopharmacological interventions for individuals with a diagnosis of ASD or PTSD. The work group review process identified further citations that included randomized and open trials, literature reviews, meta-analyses, and other studies that were incorporated into evidence tables in an iterative manner. In interpreting the conclusions of these studies, consideration was given to factors that could limit the generalizability of the findings, including differences between individuals enrolled in well-controlled efficacy trials and individuals seen in clinical practice. Consequently, the recommendations for any particular clinical decision are based on the best available data and clinical consensus. The summary of treatment recommendations is keyed according to the level of confidence with which each recommendation is made. In addition, each reference is followed by a letter code in brackets that indicates the nature of the supporting evidence.



# INTRODUCTION

It has long been recognized that stressful life events may cause emotional and behavioral effects. In addition, the clinical phenomenon of PTSD has been known by various names, studied, and treated for centuries. In 1980, DSM-III delineated distinct criteria for the diagnosis of PTSD. The diagnosis of ASD was added to DSM-IV in 1995 to distinguish individuals with PTSD-like symptoms that lasted less than 1 month from persons who experienced milder or more transient difficulties following a stressor. The DSM-IV-TR diagnostic criteria for both disorders can be found in Section II.A.2.

Although 50% to 90% of the population may be exposed to traumatic events during their lifetimes (4, 5), most exposed individuals do not develop ASD or PTSD. ASD was introduced into DSM in an effort to prospectively characterize the subpopulation of traumatically exposed persons with early symptoms and identify those at risk for the development of PTSD. Research and clinical experience show that those with high levels of symptoms early on, including those with ASD, are at risk of subsequent PTSD; however, some patients with ASD do not develop PTSD, and a proportion of patients develop PTSD without first having met the criteria for ASD (6–8). Although research shows that individuals who are most highly exposed to a traumatic event are at greatest risk, there is still uncertainty about the patient- or trauma-specific factors that will predict the development of ASD (9) and about interventions that will mitigate against the evolution of ASD into PTSD.

The lifetime prevalence of ASD is unclear, but in the National Comorbidity Survey the estimated lifetime prevalence of PTSD was 7.8% (4). The prevalence of both disorders is considerably higher among patients who seek general medical care (10) and among persons exposed to sexual assault (4, 5) or mass casualties such as those occurring in wars or natural disasters (11–13). The lifetime prevalence of PTSD is also higher in women than in men and is higher in the presence of underlying vulnerabilities such as adverse childhood experiences or comorbid diagnoses (11, 12, 14, 15). Given the prevalence of ASD and PTSD and their associated distress and disability, psychiatrists must be prepared to recognize and treat these disorders.



# PART A

## TREATMENT RECOMMENDATIONS

### I. EXECUTIVE SUMMARY

---

#### ▶ A. CODING SYSTEM

Each recommendation is identified as falling into one of three categories of endorsement, indicated by a bracketed Roman numeral following the statement. The three categories represent varying levels of clinical confidence regarding the recommendation:

- [I] Recommended with substantial clinical confidence.
- [II] Recommended with moderate clinical confidence.
- [III] May be recommended on the basis of individual circumstances.

#### ▶ B. SUMMARY OF RECOMMENDATIONS

##### 1. Initial assessment

The initial step in identifying individuals with ASD or PTSD involves screening for recent or remote trauma exposure, although the clinical approach may vary depending on the recency of the traumatic event [I]. If eliciting vivid and detailed recollections of the traumatic event immediately after exposure enhances the patient's distress, the interview may be limited to gathering information that is essential to provide needed medical care [I]. The first interventions in the aftermath of an acute trauma consist of stabilizing and supportive medical care and supportive psychiatric care and assessment [I]. After large-scale catastrophes, initial psychiatric assessment includes differential diagnosis of physical and psychological effects of the traumatic event (e.g., anxiety resulting from hemodynamic compromise, hyperventilation, somatic expressions of psychological distress, fatigue) and identification of persons or groups who are at greatest risk for subsequent psychiatric disorders, including ASD or PTSD [I]. This identification may be accomplished through individual evaluation, group interviews, consultation, and use of surveillance instruments [I].

Diagnostic evaluation may be continued after the initial period has passed and a physically and psychologically safe environment has been established, the individual's medical condition has been stabilized, psychological reassurance has been provided, and, in disaster settings, necessary triage has been accomplished. It is important for this diagnostic assessment to include a complete psychiatric evaluation that specifically assesses for the symptoms of ASD and PTSD, including dissociative, reexperiencing, avoidance/numbing, and hyperarousal symptom clusters and their temporal sequence relative to the trauma (i.e., before versus after 1 month from the traumatic event) [I]. Other important components of the assessment process include functional assessment, determining the availability of basic care resources (e.g., safe housing, social support network, companion care, food, clothing), and identifying previous traumatic experiences and comorbid physical or psychiatric disorders, including depression and substance use disorders [I].

## **2. Psychiatric management**

Psychiatric management for all patients with ASD or PTSD includes instituting interventions and activities to ensure physical and psychological safety, required medical care, and availability of needed resources for self-care and recovery [I]. The patient's level of functioning and safety, including his or her risk for suicide and potential to harm others, are always important to evaluate during initial assessment and may determine the treatment setting [I]. The goals of psychiatric management for patients with ASD and PTSD also include establishing a therapeutic alliance with the patient; providing ongoing assessment of safety and psychiatric status, including possible comorbid disorders and response to treatment; and increasing the patient's understanding of and active adaptive coping with psychosocial effects of exposure to the traumatic event, such as injury, job loss, or loss of loved ones [I]. Additional goals of psychiatric management include providing education regarding ASD and PTSD, enhancing treatment adherence, evaluating and managing physical health and functional impairments, and coordinating care to include collaborating with other clinicians [I].

## **3. General principles of treatment selection**

The goals of treatment for individuals with a diagnosis of ASD or PTSD include reducing the severity of ASD or PTSD symptoms, preventing or treating trauma-related comorbid conditions that may be present or emerge, improving adaptive functioning and restoring a psychological sense of safety and trust, limiting the generalization of the danger experienced as a result of the traumatic situation(s), and protecting against relapse [I].

Patients assessed within hours or days after an acute trauma may present with overwhelming physiological and emotional symptoms (e.g., insomnia, agitation, emotional pain, dissociation). Limited clinical trial evidence is available in this area, as randomized designs are difficult to implement; however, clinical experience suggests that these acutely traumatized individuals may benefit from supportive psychotherapeutic and psychoeducational interventions [II]. Pharmacotherapy may be the first-line intervention for acutely traumatized patients whose degree of distress precludes new verbal learning or nonpharmacological treatment strategies [II]. Research has not consistently identified patient- or trauma-specific factors that predict the development of ASD or interventions that will alter the evolution of ASD into PTSD. However, early after a trauma, once the patient's safety and medical stabilization have been addressed, supportive psychotherapy, psychoeducation, and assistance in obtaining resources such as food and shelter and locating family and friends are useful [II].

Effective treatments for the symptoms of ASD or PTSD encompass psychopharmacology, psychotherapy, and psychoeducation and other supportive measures [I]. Although studies using a combination of these approaches for ASD and PTSD are not presently available, combination treatment is widely used and may offer advantages for some patients [II]. The psychotropic medications used in clinical practice and research for the treatment of ASD and PTSD were not specifically developed for these disorders but have been used in doses similar to those recommended or approved for other psychiatric illnesses.

For patients with ASD or PTSD, choice of treatment includes consideration of age and gender, presence of comorbid medical and psychiatric illnesses, and propensity for aggression or self-injurious behavior [I]. Other factors that may influence treatment choice include the recency of the precipitating traumatic event; the severity and pattern of symptoms; the presence of particularly distressing target symptoms or symptom clusters; the development of interpersonal or family issues or occupational or work-related problems; preexisting developmental or psychological vulnerabilities, including prior trauma exposure; and the patient's preferences [I].

When the patient's symptoms do not respond to a plan of treatment, selection of subsequent interventions will depend on clinical judgment, as there are limited data to guide the clinician. It is important to systematically review factors that may contribute to treatment nonresponse, including the specifics of the initial treatment plan and its goals and rationale, the patient's per-

ceptions of the effects of treatment, the patient's understanding of and adherence to the treatment plan, and the patient's reasons for nonadherence if nonadherence is a factor [I]. Other factors that may need to be addressed in patients who are not responding to treatment include problems in the therapeutic alliance; the presence of psychosocial or environmental difficulties; the effect of earlier life experiences such as childhood abuse or previous trauma exposures; and comorbid psychiatric disorders, including substance-related disorders and personality disorders [I].

#### **4. Specific treatment strategies**

##### **(a) Psychopharmacology**

Although it has been hypothesized that pharmacological treatment soon after trauma exposure may prevent the development of ASD and PTSD, existing evidence is limited and preliminary. Thus, no specific pharmacological interventions can be recommended as efficacious in preventing the development of ASD or PTSD in at-risk individuals.

For patients with ASD, there are few studies of pharmacological interventions. However, selective serotonin reuptake inhibitors (SSRIs) [II] and other antidepressants [III] represent reasonable clinical interventions that are supported by limited findings in ASD as well as by findings of therapeutic benefits in patients with PTSD.

SSRIs are recommended as first-line medication treatment for PTSD [I]. In both male and female patients, treatment with SSRIs has been associated with relief of core PTSD symptoms in all three symptom clusters (reexperiencing, avoidance/numbing, hyperarousal). Other antidepressants, including tricyclic antidepressants and monoamine oxidase inhibitors (MAOIs), may also be beneficial in the treatment of PTSD [II].

Benzodiazepines may be useful in reducing anxiety and improving sleep [III]. Although their efficacy in treating the core symptoms of PTSD has not been established, benzodiazepines are often used in trauma-exposed individuals and patients with PTSD. However, clinical observations include the possibility of dependence, increased incidence of PTSD after early treatment with these medications, or worsening of PTSD symptoms after withdrawal of these medications. Thus, benzodiazepines cannot be recommended as monotherapy in PTSD.

In addition to being indicated in patients with comorbid psychotic disorders, second-generation antipsychotic medications (e.g., olanzapine, quetiapine, risperidone) may be helpful in individual patients with PTSD [III]. Anticonvulsant medications (e.g., divalproex, carbamazepine, topiramate, lamotrigine),  $\alpha_2$ -adrenergic agonists, and  $\beta$ -adrenergic blockers may also be helpful in treating specific symptom clusters in individual patients [III].

##### **(b) Psychotherapeutic interventions**

Some evidence is available about the effectiveness of psychotherapeutic intervention immediately after trauma in preventing development of ASD or PTSD. Studies of cognitive behavior therapy in motor vehicle and industrial accident survivors as well as in victims of rape and interpersonal violence suggest that cognitive behavior therapies may speed recovery and prevent PTSD when therapy is given over a few sessions beginning 2–3 weeks after trauma exposure [II].

Early supportive interventions, psychoeducation, and case management appear to be helpful in acutely traumatized individuals, because these approaches promote engagement in ongoing care and may facilitate entry into evidence-based psychotherapeutic and psychopharmacological treatments [II]. Encouraging acutely traumatized persons to first rely on their inherent strengths, their existing support networks, and their own judgment may also reduce the need for further intervention [II]. In populations of patients who have experienced multiple recurrent traumas, there is little evidence to suggest that early supportive care delivered as a stand-alone treatment will result in lasting reductions in PTSD symptoms. However, no evidence suggests that early supportive care is harmful. In contrast, psychological debriefings or single-session techniques are not recommended, as they may increase symptoms in some settings and appear to be ineffective in treating individuals with ASD and in preventing PTSD.

No controlled studies of psychodynamic psychotherapy, eye movement desensitization and reprocessing (EMDR), or hypnosis have been conducted that would establish data-based evidence of their efficacy as an early or preventive intervention for ASD or PTSD.

For patients with a diagnosis of ASD or PTSD, available evidence and clinical experience suggest that a number of psychotherapeutic interventions may be useful. Patients with ASD may be helped by cognitive behavior therapy and other exposure-based therapies [II]. In addition, cognitive behavior therapy is an effective treatment for core symptoms of acute and chronic PTSD [I]. EMDR is also effective [II]. Stress inoculation, imagery rehearsal, and prolonged exposure techniques may also be indicated for treatment of PTSD and PTSD-associated symptoms such as anxiety and avoidance [II]. The shared element of controlled exposure of some kind may be the critical intervention.

Psychodynamic psychotherapy may be useful in addressing developmental, interpersonal, or intrapersonal issues that relate to the nature, severity, symptoms, or treatment of ASD and PTSD and that may be of particular importance to social, occupational, and interpersonal functioning [II].

Case management, psychoeducation, and other supportive interventions may be useful in facilitating entry into ongoing treatment, appear not to exacerbate PTSD symptoms, and in some pilot investigations have been associated with PTSD symptom reduction [II]. Present-centered and trauma-focused group therapies may also reduce PTSD symptom severity [III].

## **II. FORMULATION AND IMPLEMENTATION OF A TREATMENT PLAN**

---

### **▶ A. INITIAL ASSESSMENT**

#### **1. Initial clinical approach to the patient**

The timing and nature of initial assessments will be influenced by the type of the traumatic event (e.g., sexual assault versus natural disaster) and the scope of any destruction caused by the event. In large-scale catastrophes, the initial assessment may be the triage of individuals based on the presence of physical injury or psychological effects of the traumatic event, followed by the identification of individuals at greatest risk for psychiatric sequelae, including ASD or PTSD. Group interviews, consultation, or the administration of surveillance instruments may be part of this process. If local resources are overwhelmed by the catastrophe, psychiatric assessment will need to be prioritized so that the most severely affected individuals are seen first. Several self-rated and observer-based rating scales have been developed and validated to facilitate screening for possible PTSD; however, study of these scales in community-wide disasters with highly diverse populations has been limited. Such rating scales are most likely to be helpful after the acute event, when physical and cognitive functioning allow for a more complex assessment (16–18).

With individual traumas, the timing and nature of the first mental health contact may also vary. For individuals who have been sexually assaulted, for example, supportive psychological interventions may be initiated even before formal psychiatric assessment (e.g., use of educational materials on what to expect in the rape examination). In evaluations that occur shortly after exposure to the traumatic event, particularly in emergency settings, the initial clinical response consists of stabilizing and supportive medical care as well as supportive psychiatric care and assessment, including assessment of potential dangerousness to self or others. Addressing the individual's requirements for medical care, rest, nutrition, and control of injury-related pain is important for assuring the patient's physical health, enhancing the patient's experience

of safety, and initiating the therapeutic relationship. Such interactions with trauma-exposed individuals will always entail sensitivity to the patient's wishes and to the changing symptoms, fears, and interpersonal needs that unfold after trauma exposure.

Whenever possible, care should be given within a safe environment. This may not be feasible after large-scale traumatic events in which there may be additional or ongoing exposures (e.g., earthquakes, war zones, ongoing gang warfare). With other types of traumatic events, further assurances of safety may be possible and necessary. For example, with traumatic events such as domestic violence, specific efforts or engagement of law enforcement or social service agencies may be needed to address the patient's safety and reduce the likelihood of repeat traumatization.

During the first 48–72 hours after a traumatic event, some individuals may be very aroused, anxious, or angry, whereas others may appear minimally affected or “numb” as a result of injury, pain, or dissociative phenomena (19). In triage or emergency department settings, an in-depth exploration of the traumatic event and the patient's experiences may increase distress but may be required for medical or safety reasons. For example, after physical or sexual assault, recounting events in response to the evaluator's questions or the mere gender of the evaluator may have a distressing effect in some individuals. Similarly, after an event involving death or injury to a family member, a clinician may need to obtain or disclose upsetting information, while gauging the patient's response as part of the evaluation. Insensitive or premature exploration of recent life-threatening events or losses can be counterproductive, leading the patient to avoid medical care, whereas other individuals may find in-depth exploration of recent events helpful. Therefore, evaluators must respond to the patient's needs and capabilities. After mass disasters, triage assessments in a group setting may be used effectively to identify those in need of intervention. However, discussion of distressing memories and events in heterogeneously exposed groups may adversely affect those with little or no exposure when they hear of the frightening and terrifying experiences of others.

## **2. Assessing exposure to a traumatic event and establishing a diagnosis of ASD or PTSD**

By definition, ASD and PTSD are psychiatric disorders consisting of physiological and psychological responses resulting from exposure to an event or events involving death, serious injury, or a threat to physical integrity. Events such as natural disasters, explosions, physical or sexual assaults, motor vehicle accidents, or involvement with naturally occurring or terrorist-related disease epidemics are examples of events that may elicit the physiological and psychological response required by the diagnostic criteria of ASD and PTSD. Thus, screening for acute or remote event exposure is a necessary first step in identifying persons with either ASD or PTSD.

Table 1 and Table 2 provide the full criteria for the diagnosis of ASD and PTSD, respectively. For both disorders, DSM-IV-TR defines criterion A as exposure to a traumatic event in which both of the following conditions are present:

1. The person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others.
2. The person's response involved intense fear, helplessness, or horror.

Thus, for both ASD and PTSD, establishing a diagnosis requires consideration of the individual's response to the event as well as the nature of the event itself. It is important to note that for some individuals, initial assessment may occur in a triage setting immediately after the trauma and before all symptoms related to the trauma exposure are manifest. In addition, the presence of dissociative symptoms may prevent patients from recalling feelings of fear, helplessness, or horror and may require that clinical judgment be used in determining whether criterion A for diagnosis has been satisfied (20–22).

Clinical evaluation for ASD or PTSD requires assessment of symptoms within each of three symptom clusters: reexperiencing, avoidance/numbing, and hyperarousal. In addition, to meet the diagnostic criteria for ASD, a patient must exhibit dissociative symptoms either during or

**TABLE 1. DSM-IV-TR Diagnostic Criteria for Acute Stress Disorder (DSM-IV-TR code 308.3)**

- 
- A. The person has been exposed to a traumatic event in which both of the following were present:
    - 1. the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others
    - 2. the person's response involved intense fear, helplessness, or horror
  - B. Either while experiencing or after experiencing the distressing event, the individual has three (or more) of the following dissociative symptoms:
    - 1. a subjective sense of numbing, detachment, or absence of emotional responsiveness
    - 2. a reduction in awareness of his or her surroundings (e.g., "being in a daze")
    - 3. derealization
    - 4. depersonalization
    - 5. dissociative amnesia (i.e., inability to recall an important aspect of the trauma)
  - C. The traumatic event is persistently reexperienced in at least one of the following ways: recurrent images, thoughts, dreams, illusions, flashback episodes, or a sense of reliving the experience; or distress on exposure to reminders of the traumatic event.
  - D. Marked avoidance of stimuli that arouse recollections of the trauma (e.g., thoughts, feelings, conversations, activities, places, people).
  - E. Marked symptoms of anxiety or increased arousal (e.g., difficulty sleeping, irritability, poor concentration, hypervigilance, exaggerated startle response, motor restlessness).
  - F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or impairs the individual's ability to pursue some necessary task, such as obtaining necessary assistance or mobilizing personal resources by telling family members about the traumatic experience.
  - G. The disturbance lasts for a minimum of 2 days and a maximum of 4 weeks and occurs within 4 weeks of the traumatic event.
  - H. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition, is not better accounted for by Brief Psychotic Disorder, and is not merely an exacerbation of a preexisting Axis I or Axis II disorder.
- 

*Source.* Reprinted from *Diagnostic and Statistical Manual of Mental Disorders*, 4th Edition, Text Revision. Washington, DC, American Psychiatric Association, 2000. Copyright 2000, American Psychiatric Association. Used with permission.

immediately after the traumatic event. In PTSD, dissociative symptoms (e.g., inability to recall important aspects of the trauma) are not necessary to the diagnosis but are often observed.

By definition, ASD occurs within 4 weeks of the trauma and lasts for a minimum of 2 days. Consequently, it can be diagnosed within 2 days after the trauma exposure continuing to 4 weeks after the traumatic event. If symptoms are present 1 month after the trauma exposure, PTSD is diagnosed. Since diagnostic assessment may occur at any time following a traumatic event, the clinician must bear these essential distinctions in mind when evaluating the trauma-exposed individual.

### **3. Additional features of the initial assessment**

After it has been determined that the traumatically exposed individual is able to tolerate more extensive evaluation, it is important to obtain a detailed history of the exposure and the pa-



**TABLE 2. DSM-IV-TR Diagnostic Criteria for Posttraumatic Stress Disorder (DSM-IV-TR code 309.81)**

---

- A. The person has been exposed to a traumatic event in which both of the following were present:
  - 1. the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others
  - 2. the person's response involved intense fear, helplessness, or horror. Note: In children, this may be expressed instead by disorganized or agitated behavior
- B. The traumatic event is persistently reexperienced in one (or more) of the following ways:
  - 1. recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions. Note: In young children, repetitive play may occur in which themes or aspects of the trauma are expressed.
  - 2. recurrent distressing dreams of the event. Note: In children, there may be frightening dreams without recognizable content.
  - 3. acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated). Note: In young children, trauma-specific reenactment may occur.
  - 4. intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event
  - 5. physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event
- C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:
  - 1. efforts to avoid thoughts, feelings, or conversations associated with the trauma
  - 2. efforts to avoid activities, places, or people that arouse recollections of the trauma
  - 3. inability to recall an important aspect of the trauma
  - 4. markedly diminished interest or participation in significant activities
  - 5. feeling of detachment or estrangement from others
  - 6. restricted range of affect (e.g., unable to have loving feelings)
  - 7. sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span)
- D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:
  - 1. difficulty falling or staying asleep
  - 2. irritability or outbursts of anger
  - 3. difficulty concentrating
  - 4. hypervigilance
  - 5. exaggerated startle response
- E. Duration of the disturbance (symptoms in Criteria B, C, and D) is more than 1 month.

**TABLE 2. DSM-IV-TR Diagnostic Criteria for Posttraumatic Stress Disorder (DSM-IV-TR code 309.81) (continued)**

F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

*Specify if:*

Acute: if duration of symptoms is less than 3 months

Chronic: if duration of symptoms is 3 months or more

*Specify if:*

With Delayed Onset: if onset of symptoms is at least 6 months after the stressor.

---

*Source.* Reprinted from *Diagnostic and Statistical Manual of Mental Disorders*, 4th Edition, Text Revision. Washington, DC, American Psychiatric Association, 2000. Copyright 2000, American Psychiatric Association. Used with permission.

tient's early responses to the trauma as well as the responses of significant others. This history can provide important information for treatment and prognosis. Often, individuals provide negative responses to all-inclusive questions (e.g., "Have you ever been abused?"), and responses may also be affected by the timing and context of questioning. Consequently, it is helpful to ask more specific questions (e.g., "Have you ever been hit, beaten, or choked?") and attempt to elicit a history of trauma exposure at various points during the evaluation.

During the evaluation, the clinician obtains a longitudinal history of all traumatic experiences, including age at the time of exposure, duration of exposure (e.g., single episode, recurrent, or ongoing), type of trauma (e.g., motor vehicle accident, natural disaster, physical or sexual assault), relationship between the patient and the perpetrator (in cases of interpersonal violence), and the patient's perception of the effect of these experiences (on self and significant others). Other factors or interventions that may have intensified or mitigated the traumatic response should also be identified.

Clinical interviews may be combined with a variety of validated self-rated measures, including the PTSD Checklist (23), the Impact of Event Scale (24, 25) (available online at [www.mardihorowitz.com](http://www.mardihorowitz.com)), and the Davidson Trauma Scale (26), to assess the full range, frequency, and severity of posttraumatic symptoms and the related distress and impairment. Structured diagnostic interviews such as the Clinician-Administered PTSD Scale (27) and the Structured Interview for PTSD (28) have been used extensively in clinical research and are well-validated instruments for the diagnosis of PTSD.

In addition, a complete psychiatric evaluation should be conducted in accordance with the general principles and components outlined in APA's *Practice Guideline for the Psychiatric Evaluation of Adults* (29). These components include a history of the present illness and current symptoms; a psychiatric history, including a substance use history; medical history; review of systems and a review of prescribed and over-the-counter medications (including herbal products and supplements); personal history (e.g., psychological development, response to life transitions and major life events); social, occupational, and family history; history of prior treatments or interventions and their degree of success; mental status examination; physical examination; and diagnostic tests as indicated. Developmental and preexisting psychodynamic issues may make the patient especially vulnerable or reactive to a traumatic event. Old and dormant concerns may resurface and complicate or otherwise intensify the emotional response to a new trauma. Past exposure to traumatic events as well as previous patient and support network responses may affect the evaluation process and choice of and response to treatment. In the context of this complete psychiatric evaluation, certain areas of inquiry should receive additional attention and are described below. Table 3 summarizes the clinical domains relevant to the comprehensive assessment of ASD and PTSD.

### **a) Military and war-related traumatic event history**

Evaluation of exposure to traumatic events during military service or in war-torn areas is an important and often difficult part of clinical assessment. Past exposure to war environments increases the probability of exposure to traumatic events. In addition, past exposures to traumatic events or past PTSD may increase the likelihood of current PTSD from a new exposure (31–34). Persons who come from nations with past or ongoing histories of war and war atrocities may have substantial exposure to traumatic events. Military support troops in rear areas as well as combat troops are vulnerable to attacks and other life-threatening experiences. Those serving in the military or involved in humanitarian assistance may have been massively exposed to death and the dead and can have high rates of ASD and PTSD. Military service members may also be involved in or witness training accidents, including motor vehicle accidents or aircraft crashes.

For those with military service, it is often helpful to begin the evaluation by exploring why the patient joined the military and what he or she hoped to do. Specific data to be gathered that can assist in the evaluation of traumatic event exposures include the length of service (and whether this length of time was broken or unbroken), the presence or absence of any disciplinary charges, and military awards received. The patient should also be asked if he or she was ever referred for alcohol or other substance use counseling, family violence counseling, or a psychiatric evaluation. If the patient had a family while in the service, it is important to explore the frequency and effects of family separation on the service member, the spouse, and the children. With service members or veterans who report having been in combat, a description of the location and the events should be obtained. It is often helpful to obtain copies of service records to verify combat exposures.

Witnessing atrocities, seeing the death of children, seeing friends killed and wounded, and feeling responsible for the death of a friend are especially disturbing elements of some combat and war environments for both military and civilian persons. As in all traumas, the recovery environment (that is, whether family, friends, and the nation are welcoming or ashamed) plays a large role in how the experience is recalled and managed. Some immigrants have previously lived in war zones or have served as members of military, paramilitary, or insurgent units before immigration. Some may also have been victims of torture, maltreatment, or rape as part of a war environment. Immigrants who may have served for regimes that espoused strong anti-American politics may fear repercussions from an unsympathetic country. These contextual issues require clear and supportive discussion in the evaluation and assessment in order to obtain necessary clinical information.

### **b) Victims of crime and effects of legal system involvement**

Individuals with ASD or PTSD may be involved in legal actions either because they are involved in a civil case (e.g., motor vehicle accident) related to their psychiatric condition or because they were a victim of a crime. Some individuals may express distress through a variety of symptoms that may abate after the conclusion of legal proceedings or payment of damages. This pattern may represent the effects of retraumatization resulting from exposure to a perpetrator or recollection of traumatic events during depositions, trial preparation, or testimony, followed by the (at times, only transient) sense of “closure” that these proceedings provide. If the perpetrator is incarcerated as a result of legal proceedings, symptoms may reoccur when the victim learns of the perpetrator’s parole or release. Some persons may demonstrate waxing and waning symptoms regardless of the status of legal proceedings. Individuals may also fabricate or embellish symptoms. By raising the possibility that secondary gain, symptom exaggeration, or malingering may be part of the clinical picture, these factors can complicate assessment and treatment planning, as well as research (35). Confidentiality can also be compromised if the treating psychiatrist is in a dual role and is also required to communicate with members of the legal system. Some of the complexity of these cases can be managed by having the treatment and forensic evaluations performed by different psychiatrists, if possible (36, 37). As noted in DSM-IV-TR, the psychiatric assessment should address the possibility of malingering in situ-

**TABLE 3. Clinical Domains of Assessment for Acute Stress Disorder (ASD) and Posttraumatic Stress Disorder (PTSD)**

Clinical Domain	Component
Trauma history	Type, age, and duration
Safety	Threat of harm from others and dangerousness to self or others
Dissociative symptoms	Necessary for diagnosis of ASD: numbing, detachment, derealization/depersonalization, dissociative amnesia in acute response to trauma
ASD/PTSD symptoms	Reexperiencing, avoidance and numbing, hyperarousal as a consequence of trauma (PTSD is diagnosed if symptom onset is >30 days after the traumatic event; if <30 days and if dissociative symptoms are present, ASD is diagnosed)
Military history	Prior exposure(s), training and preparedness for exposure
Behavioral and health risks	Substance use/abuse, sexually transmitted diseases, preexisting mental illness, nonadherence to treatment, impulsivity, and potential for further exposure to violence
Personal characteristics	Coping skills, resilience, interpersonal relatedness/attachment, history of developmental trauma or psychodynamic conflict(s), motivation for treatment
Psychosocial situation	Home environment, social support, employment status, ongoing violence (e.g., interpersonal, disaster/war), parenting/caregiver skills or burdens
Stressors	Acute and/or chronic trauma, poverty, loss, bereavement
Legal system involvement	Meaning of symptoms, compensation based on disability determination or degree of distress

*Source.* Adapted with permission from “Posttraumatic Stress Disorder,” by Kathryn M. Connor and Marian I. Butterfield. *Focus* 2003; 1:247–262 (30). Copyright © 2003. American Psychiatric Association.

ations in which financial remuneration or benefit eligibility is at issue or when forensic determinations play a role in establishing the diagnosis of PTSD. Determining the temporal course of symptoms relative to the timing of legal initiatives is helpful in this process (38).

### **c) Identification of ASD and PTSD in the presence of common comorbid conditions**

In patients who present for evaluation after a traumatic event, exacerbations or relapse of pre-existing comorbid disorders may occur and require evaluation and treatment (see Section III.D, “Medical and Other Psychiatric Comorbidity”) (39, 40). Exacerbations or relapse of pre-existing PTSD may also occur with subsequent traumas or reminders of trauma.

For many individuals who have experienced a traumatic event but are presenting with other clinical needs, the diagnosis of ASD or PTSD may be missed entirely without a detailed evaluation. For example, individuals hospitalized on medical or surgical services after motor vehicle accidents, severe burns, or other major physical trauma have high rates of symptomatic distress, including ASD or PTSD, that often go unrecognized (34, 41–44). Patients with serious mental illness are exposed to high rates of physical assault and sexual abuse as well as other traumas (45–49). Mental health clinicians may fail to obtain this information unless they specifically inquire (50). Seriously mentally ill persons also have higher rates of PTSD (47–49, 51), compared to the general population (5). Individuals with psychotic disorders (48) and with borderline personality disorder (50, 52–54) are particularly likely to have experienced victimization in childhood and in adulthood. The associated PTSD often goes unrecognized. Histories of victimization and PTSD

are also common among individuals with substance-related disorders (55–58) and eating disorders (59–61). In addition, family members—particularly spouses—who present with symptoms of bereavement after the traumatic loss of a family member should be assessed for PTSD (62).

High rates of comorbid psychiatric and other medical diagnoses are observed in those with ASD and PTSD. For a number of reasons, the medical and neurological effects of traumatic events may not be immediately apparent. Acute psychological responses to trauma such as dissociation may also diminish the initial experience of physical pain. In the presence of overwhelming anxiety and distress, individuals may not be able to describe their mental and physical state to medical professionals in an articulate fashion. Individuals exposed to traumatic events, particularly events that include interpersonal assault and violence, can find the motives of well-intentioned evaluators suspect. Without the establishment of trust, patients may be unwilling or unable to provide a complete medical or psychiatric history.

Patients with PTSD often have comorbid major depressive disorders, anxiety disorders, and substance use disorders (use of alcohol, tobacco, and other substances). Physical complaints, which may result from injury, may also represent comorbid somatization disorder or other somatoform disorders (12, 63). Similarly, patients with preexisting personality disorders or maladaptive character traits, as well as those with unresolved psychodynamic developmental concerns or histories of childhood traumatic events, may be at higher risk for an accentuated response to further traumatic events. In the presence of prominent depressive symptoms, social withdrawal and avoidance may be increased, and suicide risk may be heightened. Thus, identification and treatment of comorbid psychiatric and other medical illnesses are important to an integrated treatment plan that addresses all of the patient's needs and contributes to recovery from PTSD.

## ► **B. PRINCIPLES OF PSYCHIATRIC MANAGEMENT**

Psychiatric management consists of a broad array of interventions and activities that may be instituted by psychiatrists for patients who have been exposed to extreme trauma. The specific components of psychiatric management that appear to mitigate the sequelae of trauma exposure and that are important to the treatment of patients with ASD or PTSD are described in more detail below.

### **1. Evaluating the safety of the patient and others**

As with all psychiatric patients, for patients exposed to trauma it is crucial to assess the risk for suicide and nonlethal self-injurious behavior as well as the risk for harm to others. Details of suicide assessment and estimation of suicide risk are described in APA's *Practice Guideline for the Assessment and Treatment of Patients With Suicidal Behaviors* (64). Although many factors have been associated with an increased risk of suicide attempts and suicide in groups of individuals, it is not possible to predict suicide in a given individual at a given point in time. Nonetheless, a number of factors should be taken into consideration in evaluating and estimating the patient's potential for self-injury or suicide.

In assessing suicide risk, it is essential to determine whether the patient has had thoughts of death, self-harm, or suicide and the degree to which the patient intends to act on any suicidal ideation, the extent of planning or preparation for suicide, and the relative lethality of any suicide methods that the patient has considered. The availability of the means for suicide, including firearms, should also be explored, and a judgment should be made concerning the lethality of those means.

Risk for suicide and for suicide attempts is also increased by the presence of previous suicide attempts, including aborted attempts. Thus, if a patient has a history of previous suicide attempts, the nature of those attempts should be determined. Individuals who experienced childhood abuse and who may have PTSD as a result of that experience sometimes exhibit self-harming behavior that is often repetitive but occurs in the absence of suicidal intent (65, 66).

Such behavior may progress to more serious forms of nonlethal self-harm but also confers an increased risk of suicidal behaviors. Patients should also be asked about suicide in their family and recent exposure to suicide or suicide attempts by others.

Depression, substance use, panic attacks, and severe anxiety are commonly present in individuals with ASD or PTSD and are associated with increased risk for suicide and suicidal behaviors. Other factors that deserve specific attention in individuals with ASD or PTSD include the presence of dissociative symptoms; high levels of shame or stigma (e.g., after rape); loss of family, friends, or employment as a result of the traumatic event; specific neurovegetative symptoms, including insomnia or weight/appetite loss; social withdrawal; social or cultural isolation with relocation or immigration; and preexisting psychological issues, personality traits, or patterns of coping that may indicate a heightened response to a specific trauma. Individuals who feel trapped within an inescapable and abusive relationship (e.g., situations involving domestic violence, marital rape, or child abuse) or who anticipate continued, imminent exposure to traumatic experiences or stimuli may be more likely to act on suicidal ideas. An association has also been observed between the number of previous traumatic events and the likelihood that an individual will attempt suicide (67, 68). Thus, a complete assessment of suicide risk should be individualized to the particular circumstances of the patient and should also include an evaluation of the patient's strengths, social support, and motivation to seek help (69–71).

Less is known about the risk factors for harm to others in the context of PTSD. Nonetheless, it is important to assess thoughts, plans, or intentions of harming others as part of the psychiatric evaluation. As with assessment of suicide risk, it is important to determine whether firearms or other lethal weapons are available that could be used for harming others. The presence of hallucinations, persecutory delusions about a particular individual or group, or the feeling of being trapped in a dangerous, abusive, and inescapable situation may augment risk of dangerousness to others.

## **2. Determining a treatment setting**

Treatment settings for patients with ASD or PTSD include the full continuum of levels of care. Treatment should be delivered in the setting that is least restrictive, yet most likely to prove safe and effective. In determining the appropriate treatment setting, multiple patient-specific factors are considered: symptom severity, comorbidity, suicidal ideation or behavior, homicidal ideation or behavior, level of functioning, and available support system. The determination of a treatment setting should also include consideration of the patient's personal safety, ability to adequately care for him- or herself, ability to provide reliable feedback to the psychiatrist, and willingness to participate in treatment. Here also, an important consideration is the patient's ability to trust clinicians and the treatment process; this ability may be limited as a consequence of traumatic events themselves, cultural barriers, or other factors. The choice of treatment setting and the patient's ability to benefit from a different level of care should be reevaluated on an ongoing basis throughout the course of treatment, as efficacy does not necessarily increase with increasing duration of treatment in a specific setting or level of care (72).

For the majority of individuals with ASD or PTSD, treatment on an outpatient basis is the most appropriate treatment setting. However, some patients, particularly those with comorbid psychiatric and other medical diagnoses, may require treatment on an inpatient basis. Patients who exhibit suicidal or homicidal ideation, plans, or intent require close assessment and monitoring. Hospitalization is generally indicated for patients who are considered to pose a serious threat of harm to themselves or others. If such patients refuse admission, they may be hospitalized involuntarily when their condition meets local jurisdictional criteria for emergency detention or involuntary hospitalization. Severely ill patients who lack adequate social support outside a hospital setting should also be considered for hospital admission, residential treatment, or participation in an intensive outpatient or day treatment program. For severely ill patients with repeated hospitalizations related to nonadherence, assertive community treatment may also be considered.

### **3. Establishing and maintaining a therapeutic alliance**

The therapeutic alliance is important and at times challenging to establish with patients who have experienced traumatic events.

Attention to the physician-patient interaction is important, even in settings such as emergency departments where the clinician may have only a single contact with the patient. Although more than 80% of victims of recent sexual or physical assault surveyed in an emergency department setting indicated interest in further mental health treatment (73), other studies have indicated that those with PTSD underuse or avoid mental health services (74). A positive experience may also make the patient more receptive to future evaluation or follow-up.

Evaluation and treatment should always be conducted with sensitivity and in a safe environment that facilitates the development of trust. The presence of ASD or PTSD may challenge the clinician's ability to ensure that the patient feels safe in the therapeutic relationship. Clinicians must acknowledge the patient's worst fears about reexposure to intolerable traumatic memories and recognize that treatment itself may be perceived as threatening or overly intrusive. The patient is often relieved when the therapist indicates that talking about traumatic life events can be distressing and that the patient will decide how deeply to explore the difficult events and feelings. This suggestion of flexibility helps the patient to maintain or restore a sense of control, which is often lost after exposure to traumatic events. In chronic PTSD, avoidant/numbing behaviors may have been present for many years or decades. Therefore, clinicians must be patient and ensure that therapy proceeds at a tolerable pace.

Many other components of the treatment of ASD and PTSD also require trust in the doctor-patient relationship as well as particular attention to the therapeutic alliance. Effective treatment of both of these disorders requires that patients understand educational or treatment plans and return for follow-up assessment and treatment. In addition, successful treatment may require patients to tolerate intense affect and/or disruptive or unpleasant medication side effects. To establish and maintain a therapeutic alliance, it is important for the psychiatrist to address the patient's concerns as well as treatment preferences. Developing a therapeutic alliance with a patient who has experienced significant traumatic events—particularly in childhood—may require considerable psychotherapeutic effort and require lengthening of treatment. Cultural factors may also impose barriers to developing a therapeutic relationship, since many non-Western cultures do not value traditional Western psychiatric interventions. Management of the therapeutic alliance also includes awareness of transference and countertransference issues, even if these issues are not directly addressed in treatment (75).

### **4. Coordinating the treatment effort**

Providing optimal treatment for patients with ASD and PTSD may require a team approach involving the coordinated effort of several clinicians. Patients may have a wide variety of comorbid psychiatric and/or physical disorders that need to be addressed. Family intervention or coordination of support services is often needed. One team member must assume the primary overall responsibility for the patient's treatment. This individual serves as the coordinator of the treatment plan, advocates for the appropriate level of care, oversees the family involvement, makes decisions regarding which potential treatment modalities are useful and which should be discontinued, helps assess the effects of medications, and monitors the patient's safety. Because of the diversity and depth of medical knowledge and expertise required for this oversight function, a psychiatrist may be optimal for this role, although this staffing pattern may not be possible in some health care settings. Ongoing coordination of the overall treatment plan is enhanced by clear role definitions, plans for the management of crises, and regular communication among the clinicians who are involved in the treatment. If team members work collaboratively with each other, with the patient, and with the patient's family and other social supports, the treatment has a better chance of helping the patient distinguish safe from dangerous and potentially retraumatizing situations, develop self-monitoring skills and coping strategies for anx-

xiety states related to reminders of his or her trauma, avoid abusive relationships, minimize alcohol and other drug misuse, and control impulsive, aggressive, or self-destructive behaviors.

Those who have experienced an acute traumatic injury or assault often require ongoing medical attention. Collaborating with physicians who are providing additional medical treatment to the patient is an important part of psychiatric treatment. Individuals with PTSD also often have high rates of somatic and somatoform (i.e., medically unexplained) symptoms that are not directly related to the traumatic event but that prompt visits to primary care physicians (76–79). In such settings, collaboration between the psychiatrist and the primary caregiver may facilitate appropriate medical assessment and management.

## **5. Monitoring treatment response**

During treatment, different features and symptoms of the patient's illness may emerge or subside. Monitoring the patient's status for the emergence of changes in destructive impulses toward self or others is especially crucial. For patients whose risk of such behaviors is found to be increased, additional measures such as hospitalization or more intensive treatment should be considered. Emergence of new symptoms, significant deterioration in functional status, or significant periods without response to treatment may suggest a need for diagnostic reevaluation. The psychiatrist should be particularly vigilant for comorbid medical conditions or substance-related disorders, for the emergence of symptoms such as interpersonal withdrawal or avoidance, and for the development or progression of symptoms of other disorders, including anxiety disorders or major depression.

## **6. Providing education**

For persons who seek care after traumatic events, it is helpful to provide education concerning the natural course of and interventions for ASD and PTSD as well as for the broad range of normal stress-related reactions. The APA Disaster Psychiatry web site (<http://www.psych.org/disasterpsych/>) provides educational materials and links to other online resources. Education should also be given to involved family members or significant members of the patient's support network. It is important to help patients understand that their symptoms may be exacerbated by reexposure to traumatic stimuli, perceiving themselves to be in unsafe situations, or remaining in abusive relationships and that they can learn methods for better managing their feelings when they are reminded of the traumatic event. Emphasizing that ASD and PTSD are conditions for which effective treatments are available may be crucial in educating patients who attribute their illness to a moral defect or in educating family members who are convinced that nothing is wrong with the patient. Education regarding available treatment options can also help patients (and family members) make informed decisions, anticipate side effects, and adhere to treatment regimens.

For individuals or groups whose occupation entails likely exposure to traumatic events (e.g., military personnel, police, firefighters, emergency medical personnel, journalists), ongoing educational efforts may decrease exposure to trauma (by reducing risk behaviors) or improve the likelihood that an individual in need will seek care. Awareness of the predictable initial psychological and physiological responses to traumatic events may also be reassuring when these responses occur and may vitiate new fears or expectations of disability. Such education can also aid in the accurate identification and support of colleagues who develop symptoms of ASD or PTSD (80, 81).

## **7. Enhancing adherence to treatment**

For patients who develop chronic PTSD, a long or indefinite duration of treatment may be needed. During acute exacerbations, patients with chronic PTSD may be easily discouraged and unduly pessimistic about their chances of recovery. In addition, the side effects or requirements of treatments may lead to nonadherence. Patients with PTSD who appear to have achieved



a stable and positive clinical response and those who appear to have recovered from ASD may exhibit sudden relapse when new events reactivate traumatic concerns and fears about the safety of their families or themselves. For patients involved in ongoing litigation related to the traumatic event and subsequent impairment, legal proceedings may similarly reactivate concerns or emotions surrounding the event and its aftermath. The patient's motivation for participating in PTSD treatment may also be altered by ongoing legal actions. Psychiatrists should recognize these possibilities, address them in therapy, and encourage the patient to discuss any concerns regarding adherence, personal safety, or reexposure to traumatic reminders.

Medication adherence may be improved by emphasizing to the patient 1) when and how often to take the medicine, 2) the expected time interval before beneficial effects of treatment may be noticed, 3) the necessity to take medication even after feeling better, 4) the need to consult with the physician before discontinuing medication, and 5) steps to take if problems or questions arise (82). Some patients, particularly those who are elderly, have achieved improved adherence when both the complexity of the medication regimen and the cost of treatments are minimized. Severe or persistent problems of nonadherence may represent psychological concerns, psychopathology, or disruptions in the doctor-patient relationship, for which additional psychotherapy should be considered. Family members who are supportive of medication and/or other treatment can also play an important role in improving adherence. Although models of care such as assertive community treatment have not been specifically studied in individuals with PTSD, they have demonstrated efficacy in decreasing symptom severity, reducing length of hospitalization, and improving living conditions in individuals with serious and persistent mental illness (83–86). Consequently, such approaches may be useful in improving adherence in individuals with PTSD who have repeated hospitalizations related to nonadherence, particularly in the presence of significant psychiatric comorbidity.

## **8. Increasing understanding of and adaptation to the psychosocial effects of the disorder**

While trauma itself often results in detrimental social, familial, academic, occupational, and financial phenomena, further effects may also stem from the symptoms of ASD or PTSD and may perpetuate these illnesses. For example, if one loses employment as a result of a disaster or because of missed work secondary to symptoms of ASD, the additional stressor of unemployment may increase the risk of developing PTSD (87). Consequently, the psychiatrist should assist the patient in addressing issues that may arise in various life domains, including family and social relationships, living conditions, general health, and academic and occupational performance, and help the patient to consider options that may be available to address such problems (e.g., consideration of alternative school or work schedules, other vocational options, financial or social supports). Working in collaboration with patients to set realistic and achievable short- and long-term goals can be useful. Patients can increase their sense of self-worth through achieving these goals, thereby reducing the demoralization that exacerbates or perpetuates illness. It may also be important to help the patient with ASD or PTSD obtain clinical assistance for family problems or for family members who may themselves require clinical intervention. Patients who have children may need help in assessing and meeting their children's needs, both during and in the wake of acute episodes.

*Resilience* has been alternately defined (by various researchers) as an individual trait or quality, an outcome, or a process. The concept of resilience may also encompass the ability to negotiate psychosocial and emotional changes after trauma exposure and in this way increase recovery possibilities. However, studies to date have identified no universal resilience factor or outcome (88, 89). Barnes and Bell (90) suggested that factors involved in resilience include 1) biological factors (intellectual and physical ability, toughness), 2) psychological factors (adaptive mechanisms such as ego resilience, motivation, humor, hardiness, and perceptions of self; emotional attributes such as emotional well-being, hope, life satisfaction, optimism, happiness, and trust; cognitive attributes such as cognitive styles, causal attribution such as an internal locus

of control and blame, world view or philosophy of life, and wisdom), 3) spiritual attributes, 4) attributes of posttraumatic growth, 5) social attributes (interpersonal skills, interpersonal relationships, connectedness, and social support), and 6) environmental factors such as positive life events and socioeconomic status. Some studies show that optimism can buffer the effects of life stress (91–97) and enable some individuals to mobilize protective factors such as adaptive coping skills, increased self-efficacy, ways of reinterpreting adverse experiences in a positive manner, and strategies for seeking social support (98–101). Although no published studies have assessed the effect of optimism training on the development or outcome of ASD or PTSD, a school-based community-wide screening followed by psychosocial intervention was able to effectively identify and reduce disaster-related trauma symptoms and facilitate psychological recovery in children (102). Thus, efforts to improve psychosocial functioning and resilience may help to minimize symptoms and enhance recovery and remission.

## **9. Evaluating and managing physical health and functional impairments**

Because ASD and PTSD are often the result of physically traumatic events, they are frequently associated with physical health problems and with functional impairments. Other mechanisms (e.g., hyperarousal, hypothalamic-pituitary-adrenal [HPA] axis dysregulation, poor self-care) may contribute to this association (103). In those who have experienced a trauma, medical problems may affect many aspects of health. Consequently, the presence, type(s), and severity of medical symptoms should be monitored continuously. Medical symptoms, symptoms of ASD or PTSD, and psychosocial or interpersonal relationship problems are each associated with impairments in a patient's ability to function. For such impairments to be addressed, level of functioning should also be assessed on an ongoing basis. For example, some patients may require assistance in scheduling absences from work or other responsibilities, whereas others may require encouragement to avoid major life changes during intensification of symptoms.

## **▶ C. PRINCIPLES OF TREATMENT SELECTION**

### **1. Goals of treatment**

The goals of treatment for individuals who have experienced a traumatic event and have received a diagnosis of ASD or PTSD include the following.

#### **a) Reduce the severity of ASD or PTSD symptoms**

Treatment aims include reducing the patient's overall level of emotional distress as well as reducing specific target symptoms that may impair social or occupational function. In general, the clinician attempts to assist the patient to better tolerate and manage the immediate distress of the memories of the traumatic experience(s) and to decrease distress over time. In addition, the clinician works to enhance the patient's ability to discriminate trauma cues and reminders from the original traumatic experience(s) by promoting adaptive coping with reexperiencing states and instilling the belief that the current response to triggers results from recall of a past danger that is no longer present. Thus, the aim of treatment is to prevent, ameliorate, and promote recovery from the presumed neurobiological alterations associated with ASD and PTSD. Symptom-specific goals include helping the patient reduce intrusive reexperiencing, psychological and physiological reactivity to reminders, trauma-related avoidant behaviors, nightmares and sleep disturbance, and anxieties related to fears of recurrence. Other targeted goals include reducing behaviors that unduly restrict daily life, impair functioning, interfere with decision making, and contribute to engagement in high-risk behavior.

#### **b) Prevent or reduce trauma-related comorbid conditions**

Little is known about the effects of comorbid disorders on the course of ASD. Depression, substance abuse, and other conditions can impede recovery in PTSD and carry additional risks for

psychiatric morbidity and functional impairment (4, 104). Medical disorders and somatic complaints are also common in war veterans (79, 105, 106) and persons with a history of sexual abuse (107–114). Thus, a major goal of treatment is to prevent secondary disorders and to appropriately diagnose and treat other concurrent conditions when present.

**c) Improve adaptive functioning and restore or promote normal developmental progression**

ASD and particularly PTSD are associated with a range of functional impairments in various areas of daily life (10, 12, 115–122). In addition to interventions that may be needed to address such impairments, related goals are to foster resilience and assist patients in adaptively coping with trauma-related stresses and adversities.

Traumatic experiences at any stage in the life cycle may impede the normal developmental progression. Posttraumatic stress symptoms can curtail current developmental achievements (for example, in dating, friendship, marriage, parenthood, educational achievement, occupational advancement, and retirement). Fears of event or symptom recurrence, avoidant behaviors, and restrictions on interpersonal life can also lead to lost developmental opportunities. As patients recover from PTSD, a therapeutic goal is to help identify and develop strategies to restore and promote normal developmental progression.

**d) Protect against relapse**

The course of acute and posttraumatic stress reactions can vary with symptomatic exacerbation relating to reminders of trauma or loss, additional life stresses or adversities, subsequent encounters with situations of danger or trauma, or discontinuation of psychotropic medication (123). Relapse prevention assists patients in anticipating such situations and in developing skills such as problem solving, emotional regulation, and the appropriate use of interpersonal support and professional help.

**e) Integrate the danger experienced as a result of the traumatic situation(s) into a constructive schema of risk, safety, prevention, and protection**

The danger or consequences associated with the original traumatic experience can skew personal beliefs, expectations, and constructs about the future, the risks of life, and safety. In addition, patients often search for the meaning of their life experience. The treatment of PTSD may include strategies to assist patients to constructively address these issues. As PTSD often evolves into a chronic illness, the meaning of the precipitating trauma in terms of its connections to past experience and its effects on subsequent perceptions of self-worth and interpersonal relationships may need to be addressed. Psychodynamic approaches and other psychotherapies may facilitate this integration (124–127).

## **2. Choice of initial treatment modality**

Patients assessed within hours or days after an acute trauma may present with overwhelming posttraumatic physiological and emotional symptoms that would appear to prevent or severely limit psychotherapeutic interchanges. Such presentations do not necessarily indicate impending development of ASD or PTSD. However, pharmacological intervention to relieve overwhelming physical or psychological pain, impairing insomnia, or extremes of agitation, rage, or dissociation may restore baseline function or may be a useful temporizing measure as the clinician monitors for the development of additional symptoms and considers additional psychotherapeutic intervention and/or medication treatment.

Treatment of ASD or PTSD symptoms includes three broad categories of intervention: pharmacological treatment, psychotherapeutic intervention, and education and supportive measures. While cognitive and behavior therapies and pharmacological intervention (particularly with SSRIs) have reasonable clinical evidence to support their efficacy in treating the core symptoms of PTSD (see Section II.D, “Specific Treatment Strategies”), few direct comparisons of specific interventions or studies of combinations of support/education, pharmacological intervention,

and psychotherapies are available. Nonetheless, consensus suggests that several factors, including the presence of specific target symptoms and individual patient characteristics, may guide decisions regarding initial treatment; these factors are reviewed in Section II.D, “Specific Treatment Strategies.”

For patients with ASD as well as for those without overt symptoms, single-session individual debriefing does not prevent PTSD and may impede recovery (128, 129). In ASD, early after a trauma, once the patient’s safety and medical stabilization have been addressed, supportive psychotherapy, case management, and assistance in obtaining resources such as food or shelter are useful (130, 131). Furthermore, in contrast to the findings for debriefing, there is no evidence to suggest that early supportive care is harmful (131–134). Preliminary evidence also suggests that ASD patients may be helped by cognitive behavior psychotherapy that incorporates exposure (135–137). Although there are few studies of pharmacological interventions in patients with ASD, treatment with SSRIs and possibly other antidepressants may represent reasonable initial clinical interventions.

In individuals with PTSD, evidence from randomized, controlled trials supports both psychotherapeutic and medication-based approaches to initial treatment. SSRIs are recommended as first-line medication treatment for PTSD, and other antidepressants may also be beneficial. In terms of psychotherapies, cognitive behavior therapy is an effective treatment for core symptoms of acute and chronic PTSD. EMDR is also effective. Stress inoculation, imagery rehearsal, and prolonged exposure techniques may also be employed in treating PTSD as well as associated symptoms such as anxiety and avoidance. The use of psychodynamic psychotherapy in treating PTSD is supported by a considerable number of descriptive studies and process-to-outcome analyses as well as substantial clinical experience. It may be useful in addressing developmental, interpersonal, or intrapersonal issues that may be of particular importance to social, occupational, and interpersonal functioning. It also appears to be useful in addressing the patient’s changes in beliefs, world expectations, generalization of threat experiences to other life events, and attempts to find meaning in her or his experience. Interpersonal issues that develop as a result of ASD or PTSD, including changes in interpersonal relationships, fears, avoidance, loss of trust, anger and aggression, and increasing generalization of fears and threat, should also be addressed psychotherapeutically.

The presence of a comorbid psychiatric disorder may also guide initial intervention. For example, substance misuse is a common concomitant of ASD or PTSD and signals a need for specific treatment for substance use disorder. In addition, individuals who are depressed may be at greater risk for further exposures to trauma. For example, when domestic partner violence is ongoing, low self-esteem or decreased energy accompanying depression may produce increased violence in the abusive partner or inadequate self-protective efforts in the patient. Thus, direct and vigorous treatment of underlying depression with psychotherapy and/or specific antidepressant pharmacotherapy may minimize the risk for additional trauma and development or prolongation of PTSD.

### **3. Approaches for patients who do not respond to initial treatment**

Because of the paucity of high-quality evidence-based studies of interventions for patients with treatment-resistant PTSD, treatment nonresponse cannot be addressed algorithmically. However, a systematic review of the factors that may be contributing to treatment nonresponse is possible. Since the initial treatment plan will have detailed each selected treatment, the rationale for its use, and the goals for treatment outcome, a review of this initial plan of care should help determine the extent to which therapeutic goals have been met. If interventions have been introduced sequentially, it will be easier to discern their individual effects. In reviewing the original plan, the clinician should explore with the patient which (if any) symptoms have improved, worsened, or remained the same. It is also important to determine whether the patient understands the plan and is adhering to it and, if nonadherence is present, the reasons for non-

adherence. For example, has the patient failed to do homework assignments or discontinued medications or skipped doses because of side effects or financial difficulties? The potential of other psychological disorders or underlying personality traits to interfere with the treatment should be reconsidered and addressed as needed. The therapist should inquire about any new psychosocial or other environmental factors that may be hindering therapy, such as a conflict at work or with family members.

If it appears that the therapist-patient relationship is not at issue and that the patient is adhering to the treatment, the therapist should explore other options. One strategy for nonresponse is to augment the initial treatment with another—for example, adding pharmacotherapy to psychotherapy, psychotherapy to a pharmacological intervention, or couples therapy to an individual psychotherapy. Generally, the therapist should first exhaust the treatments for which there is the best evidence of efficacy before trying more novel treatments. In some cases, the original treatment may need to be discontinued and a different modality selected, as in the case of a patient who is too overwhelmed by anxiety to tolerate exposure therapy. Because most therapies used for the treatment of PTSD or ASD are also indicated for other psychiatric conditions, a review of the literature on strategies for improving response in those situations may also be helpful. However, there are limited data to guide the clinician in the treatment of patients with treatment-resistant PTSD and ASD, and, at present, clinical judgment must prompt the selection of one path rather than another.

#### ► **D. SPECIFIC TREATMENT STRATEGIES**

Since patients with a diagnosis of ASD or PTSD experience a broad and complex range of symptoms, caring for patients with these disorders involves an array of approaches and should include consideration of the biopsychosocial diversity of the patient's clinical presentation. When choosing a specific strategy to treat ASD or PTSD, it is important to consider the weight of scientific evidence supporting each treatment option as well as the limitations of the current evidence base. There have been relatively few double-blind, randomized, controlled trials of treatments for patients with PTSD and even fewer such trials for patients with ASD. Many promising results still require replication, and some interventions that are commonly used, based on extensive clinical experience and consensus, have yet to be examined in more methodologically rigorous studies. In the studies that are available, treatment and follow-up durations are typically short, sample sizes are frequently small, and the possibility of a placebo response is often inadequately addressed (138). Furthermore, measured outcomes have often concentrated on more readily quantifiable changes in specific symptoms rather than focusing on the diagnosis of ASD and PTSD per se or on important short- and long-term outcomes such as social, occupational, and interpersonal functioning.

It is also likely that responses to specific treatments may differ depending on the type of trauma experienced (e.g., acute versus ongoing or cumulative, natural disaster versus interpersonal violence, community-wide versus individual traumatic event, presence versus absence of simultaneous physical injury) and the timing of treatment relative to the occurrence of the traumatic event. Since ASD, by definition, occurs in the 4 weeks immediately after a traumatic event, studies of treatment interventions during this period should be considered as treatment of ASD and potentially as preventive strategies for PTSD. Treatment strategies for symptoms occurring between 1 and 3 months after trauma exposure (acute PTSD) may be different than those for symptoms occurring (or reoccurring) more than 3 months after the traumatic event(s) (chronic PTSD), although the differential efficacies of specific strategies for treating acute versus chronic PTSD have not been well studied. Throughout the first 3 months after a traumatic event, recovery is the general rule (139), and this natural recovery period may extend up to 6 months (34, 140). Here, the clinician is guided by the expectation of recovery, the relief of suffering, and the use of interventions to speed recovery and to prevent additional exposure to the traumatic event, chronicity of symptoms, and relapse.

In choosing a specific treatment strategy, consideration should also be given to the patient's age, gender, and previous history (e.g., developmental history, past traumatic experiences, substance use disorders, other psychiatric diagnoses), current comorbid medical and psychiatric illnesses, propensity for aggression or self-injurious behavior (see Section III, "Specific Clinical Features Influencing the Treatment Plan"), or other factors that may vary widely across individuals. Although systematic study of these factors is rare, clinical experience suggests that these factors may also necessitate modification of the individual treatment plan. Specific treatment strategies should be selected to target the symptoms or symptom clusters (i.e., reexperiencing, avoidance/numbing, or hyperarousal) that are most disruptive for the patient and to take into account the time interval between trauma exposure and symptom development. Personality style and family interactions may affect symptom expression, persistence, or exacerbation.

Treatment for the symptoms of ASD or PTSD involves three approaches either alone or in combination: psychopharmacology, psychotherapy, and education and supportive measures. To date, no psychotropic medications have been developed specifically for use in ASD or PTSD. Therefore, in clinical practice and in pharmacotherapy research, medications have been used in doses similar to those recommended or approved for other psychiatric illnesses. While the clinical evidence to date for each of these interventions is limited, the efficacy of combinations of education/support, psychotherapy, and psychopharmacology has been even less well characterized. Clinical practice and consensus support combinations of these approaches based on several factors, such as specifically identified target symptoms, psychiatric and other medical comorbidity, and the patient's preferences. Medication therapy may also be initiated to address symptoms (e.g., physical pain, agitation, severe insomnia, or psychosis) that might otherwise limit the efficacy of psychotherapy. The sections that follow summarize specific psychopharmacological, psychotherapeutic, and educational and supportive approaches to the treatment of ASD and PTSD. Where efficacy has been established to a greater degree with regard to particular symptoms or clinical features or at particular time intervals after the trauma exposure, these findings are highlighted.

## **1. Psychopharmacology**

### **a) SSRIs**

Evidence from several large randomized, double-blind controlled trials suggests that SSRIs are first-line medication treatment for both men and women with PTSD (123, 141–147). There are four reasons that SSRIs are the current medications of choice for PTSD: 1) they ameliorate all three PTSD symptom clusters (i.e., reexperiencing, avoidance/numbing, and hyperarousal), 2) they are effective treatments for psychiatric disorders that are frequently comorbid with PTSD (e.g., depression, panic disorder, social phobia, and obsessive-compulsive disorder), 3) they may reduce clinical symptoms (such as suicidal, impulsive, and aggressive behaviors) that often complicate management of PTSD, and 4) they have relatively few side effects.

Reductions in the severity of core PTSD symptoms have been shown with fluoxetine, sertraline, and paroxetine in studies that were of relatively short duration (8–12 weeks) and included predominantly women with chronic PTSD resulting from rape or assault (123, 141–146, 148). While symptom reduction was generally observed within 2–4 weeks of treatment, symptoms of anger and irritability were reduced within the first week (149). In studies of fluoxetine, improvement in arousal, numbing, and avoidance (but not reexperiencing) and overall response were greater in women than in men. Other studies have demonstrated efficacy for these agents in intrusive, avoidance/numbing, and arousal symptoms. Smaller open-label studies of fluvoxamine have shown efficacy in sleep-related symptoms (including nightmares) in combat veterans (147, 150). Head-to-head comparisons between any of the SSRIs for ASD or PTSD symptoms have not been published; however, clinical consensus holds that these agents differ primarily in their pharmacokinetics, metabolic effects on other medications, and side effects rather than in their efficacy in treating ASD or PTSD.

### **b) Tricyclic antidepressants and MAOIs**

Studies of tricyclic antidepressants demonstrated efficacy for amitriptyline and imipramine (151, 152) but not desipramine (153). With the MAOIs, limited data suggest the efficacy of phenelzine and brofaromine (an MAOI available in Europe) (154, 155). In all of the trials, subjects were primarily male combat veterans, which limits the generalizability of findings. There do not appear to be studies of the effects of either MAOIs or tricyclic antidepressants specifically in women with PTSD or ASD.

### **c) Benzodiazepines**

While benzodiazepines can reduce anxiety and improve sleep, their efficacy in preventing PTSD or treating the core symptoms of PTSD has been neither established nor adequately evaluated (156, 157). Concerns about addictive potential in individuals with comorbid substance use disorders may prompt additional caution regarding the use of benzodiazepines. Worsening of symptoms with benzodiazepine discontinuation has also been reported (158). However, in a naturalistic study of more than 300 veterans with PTSD and comorbid substance abuse, treatment with benzodiazepines was not associated with adverse effects on outcome (159).

### **d) Anticonvulsants**

Open-label studies of divalproex, carbamazepine, and topiramate have demonstrated mixed or limited efficacy with regard to specific symptom clusters of PTSD (160–162), but these studies, as well as a single controlled trial of lamotrigine (163), have indicated benefit with regard to the reexperiencing symptoms.

### **e) Antipsychotics**

Psychotic symptoms are not included in the diagnostic criteria for either ASD or PTSD. Nonetheless, patients with these illnesses may also experience psychotic symptoms as part of a comorbid disorder. Before initiating antipsychotic treatment, careful diagnostic evaluation is required to appropriately address the potential contributions of delirium, dementia, primary thought disorders, brief psychotic reactions, delusional disorder, substance abuse, closed head injury, or other comorbid general medical conditions. Preliminary studies of the second-generation antipsychotic agents olanzapine (164–166), quetiapine (167), and risperidone (168) in patients with PTSD suggest a potential role for these medications in pharmacological treatment, particularly when concomitant psychotic symptoms are present or when first-line approaches have been ineffective in controlling symptoms.

### **f) Adrenergic inhibitors**

Agents acting on adrenergic receptors have also been proposed for the treatment of PTSD. Preliminary evidence has shown possible benefits with the  $\alpha_1$  antagonist prazosin (169) and with the  $\alpha_2$  agonist clonidine in combination with imipramine (170). However, there have been no large controlled studies of these agents to date.

While  $\beta$ -adrenergic blockers are at times prescribed for PTSD (171) and have been used in the treatment of performance anxiety, there have been no controlled studies of these agents for PTSD. Preliminary results suggest that acute administration of propranolol after trauma may reduce some later symptoms of PTSD (137, 172). Further controlled studies are necessary to evaluate this practice before it can be considered a part of the therapeutic armamentarium.

## **2. Psychotherapeutic interventions**

### **a) Cognitive and behavior therapies**

Cognitive behavior therapy in ASD or PTSD targets the distorted threat appraisal process (in some instances through repeated exposure and in others through techniques focusing on infor-

mation processing without repeated exposure) in an effort to desensitize the patient to trauma-related triggers. Distinctions may be drawn between psychotherapies that focus principally on aspects of cognitive processing and those that emphasize behavioral techniques. However, aspects of both are frequently combined, and studies that identify the effective components of these therapies or that distinguish one from another are not available. A course of cognitive behavior therapy generally begins with education about the symptoms of the disorder, as well as a rationale for asking the patient to recall painful experiences and relaxation training. After the therapist assesses the patient's ability to tolerate within-session anxiety and temporary exacerbations of symptoms, the patient is led through a series of sessions in which the traumatic event and its aftermath are imagined and described, and the patient is asked to focus on the negative affect and arousal until they subside. Reassurance and relaxation exercises aid the patient in progressing through these sessions, and homework assignments allow the patient to practice outside the sessions or while confronting triggers of anxiety (specific places or activities) in vivo (125, 173, 174). A limited number of well-designed studies demonstrate some success not only in speeding recovery but also in preventing PTSD when cognitive behavior therapy is given over a few sessions beginning 2–3 weeks after trauma exposure (135, 173, 175–178). Both stress inoculation and prolonged exposure techniques have demonstrated efficacy in women with PTSD resulting from assault or rape (179–181). Prolonged exposure (through imaginal and in vivo exposure to avoided situations associated with previous trauma) has been shown to be effective, particularly in the PTSD-associated symptoms of anxiety and avoidance (179, 182). However, several studies have noted that exposure may increase rather than decrease symptoms in some individuals (178, 183). Stress inoculation training involving breathing exercises, relaxation training, thought stopping, role playing, and cognitive restructuring has also proven effective alone and in combination with prolonged exposure in reducing PTSD symptoms (179). Survivors of rape, crime victims, and combat veterans have demonstrated improvement in overall PTSD symptoms and nightmares in response to imagery rehearsal (i.e., imaginal prolonged exposure) (184, 185). Clinical improvement (but not recovery) was also demonstrated in a group of PTSD patients with diverse trauma exposures who received either imaginal exposure or cognitive behavior therapy (186, 187). In group settings, cognitive processing therapy designed to correct distortions related to threat appraisal and safety through a facilitated study of the patient's written narrative of his or her traumatic experience has shown promise (188). Most of these trials have been short-term, and the extent to which improvement is maintained over time has not been assessed through follow-up study.

#### **b) Eye movement desensitization and reprocessing (EMDR)**

EMDR is a form of psychotherapy that includes an exposure-based therapy (with multiple brief, interrupted exposures to traumatic material), eye movement, and recall and verbalization of traumatic memories of an event or events. It therefore combines multiple theoretical perspectives and techniques, including cognitive behavior therapy. Some point to the use of directed eye movements as a feature markedly distinguishing this form of therapy from other cognitive behavior approaches. Others point to the fact that traumatic material need not be verbalized; instead, patients are directed to think about their traumatic experiences without having to discuss them. Like many of the studies of other cognitive behavior and exposure therapies, most of the well-designed EMDR studies have been small, but several meta-analyses have demonstrated efficacy similar to that of other forms of cognitive and behavior therapy (189–192). Studies also suggest that the eye movements are neither necessary nor sufficient to the outcome (193–195), but these findings remain controversial (196, 197). Although it appears that efficacy may be related to the components of the technique common to other exposure-based cognitive therapies, as in the previously described cognitive behavior therapies, further study is necessary to clearly identify the effective subcomponents of combined techniques. Follow-up studies are also needed to determine whether observed improvements are maintained over time.



### **c) Psychodynamic psychotherapy**

Psychodynamic therapy has, from its beginnings, been concerned with responses to traumatic events (198–200). There is an extensive body of research that includes descriptive designs, process-to-outcome correlational studies, and case studies. However, randomized, controlled research on psychodynamic psychotherapy in patients with ASD or PTSD is extremely limited. One controlled trial of psychodynamic therapy versus hypnotherapy or desensitization versus no therapy showed all interventions were superior to the control condition (no treatment) in decreasing avoidance and intrusive symptoms (201). Other controlled trials of hypnotherapy for ASD or PTSD have not been published, but descriptive studies and clinical consensus support its use—by appropriately trained individuals—in reducing symptoms of anxiety associated with acute distress and traumatic event cues and as a nonpharmacological adjunctive approach to anxiety reduction (202). A meta-analysis of controlled psychotherapy trials (including the study by Brom et al. [201]) also suggested the efficacy of hypnosis—particularly at the end of therapy (203).

The clinical research and narrative-based literatures on psychodynamic psychotherapy outline two major approaches to the treatment of traumatic stress disorders. The first views an individual's defenses and coping skills as a product of his or her biopsychosocial development and focuses on the meaning of the trauma for the individual in terms of prior psychological conflicts and developmental experience and relationships, as well as the particular developmental time of the traumatic occurrence(s). This approach examines the person's overall capacity to cope with memories of traumatic event(s) and their triggers and the coping style he or she uses to manage these memories (204, 205). The second approach focuses on the effect of traumatic experience on the individual's prior self-object experiences, overwhelmed self-esteem, altered experience of safety, and loss of self-cohesiveness and self-observing functions and helps the person identify and maintain a functional sense of self in the face of trauma (206, 207). Both approaches appear to be useful in addressing the subjective and interpersonal sustaining factors of the illness (e.g., shattered assumptions about attachments, issues of trust), as well as the changes in beliefs and world view and the widely altered threat perceptions often seen in chronic PTSD (21, 208, 209). Psychodynamic psychotherapists employ a mixture of supportive and insight-oriented interventions based on an assessment of the individual patient's symptoms, developmental history, personality, and available social supports as well as an ongoing assessment of the patient's ability to tolerate exploration of the trauma (210, 211). In chronic PTSD, issues of transference are often explored to help the patient understand conscious and unconscious concerns surrounding the meaning of recent and more remote traumatic events in his or her life as they appear in the treatment (212). Awareness of countertransference is a central component of treatment of traumatic experience in psychodynamic psychotherapy and in other therapies. The therapist's emotional response on hearing the patient describe the traumatic events can either facilitate or disrupt the therapeutic alliance, making ongoing attention to countertransference of particular importance in treating patients with ASD and PTSD.

### **d) Psychological debriefing**

Psychological debriefing was developed as an intervention aimed at preventing the development of the negative emotional sequelae of traumatic events, including ASD and PTSD. This staged, semistructured group (or, as often administered, individual) interview and educational process includes education about trauma experiences in general and about the chronological facts of the recently experienced traumatic event and exploration of the emotions associated with the event. Since debriefing has received considerable publicity, it may be expected (or specifically requested) by leaders or managers when a group confronts disaster. In the military, for example, group debriefings have been used as a means for describing normative responses to trauma exposures and educating individuals about pursuing further assistance if symptoms persist or cause significant dysfunction or distress. However, well-controlled studies of debriefing that have used single-session, individual, and group debriefing have not demonstrated efficacy

(128, 129, 213–216). Although some trauma survivors have reported that they experienced such debriefings as helpful, there is no evidence at present that establishes psychological debriefing as effective in preventing PTSD or improving social and occupational functioning. In some settings, it has been shown to increase symptoms (217–219). Its use may be most problematic with groups of unknown individuals who have widely varying trauma exposures or when it is administered early after trauma exposure, before safety and decreased arousal are established. Immediately after exposure, persons may not be able to listen attentively, absorb new information, or appreciate the nuances of the demands ahead in a manner that promotes recovery (220, 221). Also, in heterogeneous groups, some individuals will be increasing their exposure through group participation and obtain no added support after the group session, thereby potentially increasing their likelihood of later distress (19).

### **3. Psychoeducation and support**

Supportive interventions are often used as the control intervention in studies of more specific treatments. However, clinical experience indicates that both support and psychoeducation appear to be helpful as early interventions to reduce the psychological sequelae of exposure to mass violence or disaster. When access to expert care is limited by environmental conditions or reduced availability of medical resources, rapid dissemination of educational materials may help many persons to deal effectively with subsyndromal manifestations of trauma exposure. Such educational materials often focus on 1) the expected physiological and emotional response to traumatic events, 2) strategies for decreasing secondary or continuous exposure to the traumatic event, 3) stress-reduction techniques such as breathing exercises and physical exercise, 4) the importance of remaining mentally active, 5) the need to concentrate on self-care tasks in the aftermath of trauma, and 6) recommendations for early referral if symptoms persist. Encouraging persons who are acutely traumatized to first rely on their inherent strengths, their existing support networks, and their own judgment may reduce the need for further intervention. Although the efficacy of these measures alone in prevention of ASD or PTSD is unproven, emphasis on self-reliance and self-care should augment other strategies when and if they become necessary.

## **III. SPECIFIC CLINICAL FEATURES INFLUENCING THE TREATMENT PLAN**

---

### **▶ A. AGE**

Trauma exposure, and therefore ASD and PTSD, occurs in individuals of all ages, including infants. For all types of trauma, exposure varies with age (5), peaking in late adolescence. Although findings on the relationship between age and risk for developing PTSD are inconsistent (4, 33, 222), age and developmental stage may be important considerations in treatment. The meaning of the exposure to a traumatic event will differ depending on the developmental stage as well as the extent of any preexisting emotional problems or age-specific concerns of the patient. For example, an injury that causes a loss of a limb in early adulthood can raise issues of how to establish long-term intimate relations with a disability, while a similar injury late in life may raise fears of dependency, loss of mobility, and needs for care that may not be available in the family. Confrontation with the threat of the loss of one's life will also raise different concerns depending on the time of life. Since these meanings affect the patient in life planning, they should be addressed in psychotherapy or supportive treatment. Advancing age increases the probability of comorbid medical disorders (e.g., hypertension, renal failure, heart disease) and concomitant medication use that will influence psychopharmacological decisions.

## ▶ **B. GENDER**

Although overall exposure to trauma may be somewhat greater in men than in women (4, 5, 223), men and women differ in the types of traumatic events to which they are most likely to be exposed (4, 5, 223–226). That men are more likely to be exposed to combat and physical violence, whereas women are more likely to be exposed to rape and sexual assault, only partly accounts for the significantly higher lifetime prevalence rates of PTSD among women in the general population (4, 5, 222, 223, 226) as well as the longer duration of PTSD among women (5, 226). Differences in trauma exposures between men and women may also affect treatment considerations.

Initial assessment after sexual assault or rape requires a willingness to listen to the patient with an open mind to obtain necessary medical and investigative information and establish trust. Early attention to the therapeutic alliance may enhance the degree to which support and psychotherapy may be helpful in addressing later difficulties such as sexually transmitted diseases, pregnancy, difficult contraceptive choices, and feelings of loss of self-esteem, anger, rage, or guilt. Research neither supports nor refutes the prevalent notion that a treating clinician who is experienced as “different” from the perpetrator will more rapidly be accepted early on after the traumatic event. However, the gender of the treating clinician may be an issue for a specific patient under specific circumstances; therefore, the potential influence of the clinician’s gender on treatment response should be considered.

## ▶ **C. ETHNIC AND CROSS-CULTURAL FACTORS**

The likelihood of being exposed to traumatic events, as well as the likelihood of receiving a lifetime diagnosis of PTSD, differs by ethnic group. In general, clinicians who understand the importance of social and cultural dynamics will be sensitive to the need to treat patients with ASD and PTSD in such a manner as to not alienate them from their families and communities. Treatment must be knowledgeable and respectful of the culture, the cultural meaning of symptoms or illness, and cultural values of the patient and the patient’s family. Treatment must also recognize that the “cultural context” in which treatment occurs may affect the development of symptoms. That Central American refugees are viewed as immigrants rather than persons escaping combat and that Vietnam veterans were viewed with disdain rather than welcomed as heroes may help explain different aspects of these traumatized populations or their response to treatment, compared to others entering the United States in the aftermath of war. Clinicians must be sensitive to the idea that such societal views may also shape treatment response. An individual’s culture may be protective and contain a supportive system of values, roles, lifestyles, and knowledge that may buffer some of the effects of traumatic events (227). Protective influences of culture and social systems occur in part through provision of an acceptable context in which social support can be experienced and the traumatic event interpreted. The social and cultural context has the potential to provide a positive evaluation of the self, as well as to provide social support, both of which buffer the negative effects of stressful events (228). In other situations, cultural norms may contribute to the perception of an experience as traumatic (e.g., a rape victim may be shunned by family members for having “shamed” them). In addition, a disruption of social and cultural foundations can result in drastic changes in people’s expectations and views of the meaning of life, thus making individuals potentially more vulnerable to traumatic events. Consequently, therapy must be conducted in a manner that does not estrange the individual from his or her family and community (229). Thus, while psychosocial treatments that attempt to identify and process traumatic experiences may be effective for individuals from Western cultures, they may be contraindicated for some Southeast Asian populations and persons from other non-Western cultures (229).

No controlled studies have explored the extent to which specific religious groups or subgroups within the United States may be more or less likely to seek care for psychiatric symptoms related to trauma exposure. However, African American veterans may be less likely than European Amer-

ican veterans to use psychosocial care outside Department of Veterans Affairs (VA) clinical programs, even though both ethnic groups appear to respond similarly to treatment for PTSD (230).

Ethnicity is also relevant to the pharmacological treatment of patients with ASD or PTSD. Cultural values may affect a patient's decision to take medication or a patient's adherence to medication regimens. Moreover, genetic polymorphisms in hepatic cytochrome P450 (CYP) enzymes occur at varying frequencies across ethnic groups (231–234). Since most psychotropic medications are metabolized through the CYP system, polymorphisms will affect the likelihood that an individual patient will experience therapeutic benefits or adverse effects at a given dose of medication. For example, about 7% of Caucasians are poor metabolizers of CYP 2D6 substrates, and 3%–6% are poor metabolizers of CYP 2C19 substrates (233, 234). These patients would be expected to have disproportionately high blood levels of medications that are metabolized through these routes. In contrast, ultrarapid metabolism by means of CYP 2D6 enzymes is observed in 1%–3% of Middle Europeans but up to 29% of Ethiopians (232). Finally, because ethnic groups also differ in genetic polymorphisms affecting sites of psychotropic action (e.g., serotonin transporters), a drug's pharmacodynamic properties may also vary with ethnicity (235, 236). These findings emphasize the need to take ethnic and cultural factors into consideration in developing a plan of therapy with the patient.

#### ► **D. MEDICAL AND OTHER PSYCHIATRIC COMORBIDITY**

Individuals with ASD or PTSD present with a complex array of symptoms and comorbid conditions. Physical injury is common as a result of the exposure to traumatic events. Patients with PTSD may present with medical or somatic concerns. Indeed, a history of childhood physical and/or sexual abuse has been associated with a greater number of hospital admissions and surgical procedures, somatization, and hypochondriasis in adulthood (237). Victimization, particularly exposure to chronic trauma, has also been associated with chronic gastrointestinal symptoms (111, 114, 238–242), chronic pain syndromes (107–114), and fibromyalgia (243, 244). Thus, gender differences in the rates of childhood physical and/or sexual victimization may contribute to gender differences in associated medical comorbidity. In addition, physical disorders such as cardiac or neurological illnesses may mimic symptoms of traumatic stress (229), resulting in underdiagnosis of either ASD or PTSD. This confusion may result in inadequate treatment of posttraumatic anxiety disorders but may also result in inappropriate provision of medical or surgical care, including unnecessary prescribing of potentially addictive substances. Thus, in treating individuals with ASD or PTSD, coordination of care with other physicians is important in developing an appropriate plan of diagnostic assessment and therapy for concomitant somatic symptoms and medical disorders.

In intensive care and rehabilitation settings, ASD and later PTSD may be part of the complex medical picture of patients recovering from injuries ranging from burns and amputations to traumatic brain injury. Consideration of the patient's physical function, concurrent medications, and need for medical intervention is required for appropriate pharmacological management and psychotherapy. In the emergency department, life-sustaining measures as well as hydration, sleep, and nutrition must take precedence over psychosocial treatments. However, the longer the stay in the hospital, the more likely that ASD or PTSD symptoms will become a focus for treatment, as sleep disturbances, anxiety, depression, or fears of planning for the future become evident. Family members may also have substantial reactions to the traumatic events their loved ones have experienced. Family members should be afforded opportunities to discuss their concerns in an environment that fosters trust. They should receive available information about the condition or prognosis of loved ones, including discussion of the range of behavioral and emotional responses that may arise in the injured person(s) and in other family members. Often, indirectly affected family members will request advice about how to discuss or whether to discuss certain topics with the patient (e.g., death of a husband or wife in the same motor vehicle collision). These issues may further complicate the evaluation and manage-

ment of traumatized patients and must be taken into consideration when developing a treatment plan. Complicated evaluations may, by necessity, be initiated in an inpatient (intensive care or rehabilitative) setting but continue into outpatient care.

Patients who develop ASD or PTSD are also more likely to have other comorbid psychiatric disorders, including mood, dissociative, anxiety, substance-related, and personality disorders (171, 242, 245–254). Somatization disorder may also co-occur with ASD or PTSD, and in some individuals posttraumatic symptoms may represent somatization disorder psychopathology rather than ASD or PTSD (12). Thus, integrated treatment of ASD or PTSD and other psychiatric disorders is often required.

Among individuals with ASD or PTSD, depression and suicidal ideation or behavior require particular attention both pharmacologically and psychotherapeutically. Associated symptoms of depression, such as interpersonal withdrawal, survivor guilt, or shame, may be more amenable to psychosocial interventions than psychopharmacological interventions. Suicide risk may increase as the individual adjusts to physical losses or experiences guilt, shame, anger, or grief related to the loss of loved ones who may have been injured or may have died in the same traumatic event. While treatment targeted to specific symptoms of ASD or PTSD may also address these associated depressive features, such treatment may need to be continued beyond the time frame necessary to address ASD or PTSD alone.

Substance use may have a complex relation to ASD or PTSD after trauma. At times, substance use may contribute to the traumatic event itself (e.g., industrial or motor vehicle accident). Substance use may also be part of a preexisting substance use disorder or may reflect the patient's attempt to treat posttraumatic symptoms (e.g., sleep disturbance or anxiety). In fact, a period of increased substance (alcohol, tobacco, or drug of abuse) use often occurs early in ASD or PTSD, even when no substance use disorder existed before the trauma. In studies of large populations that have been exposed to trauma, higher rates of alcohol and tobacco use are observed after the event (255). Other studies of traumatized adults have reported high rates of alcohol and substance use (247, 250, 256, 257). Although increased usage does not equate to the presence of a substance use disorder, it remains a potential health concern and risk factor for other medical comorbidity. Substance misuse may also complicate psychiatric treatment of ASD or PTSD by producing symptoms that decrease the patient's ability to make use of psychotherapeutic treatments. Substance use also complicates pharmacological management and increases the risk of inadvertent patient overdose, somnolence, and behavioral problems. Thus, after a traumatic event, increased use of substances should be addressed as part of the treatment of ASD or PTSD, regardless of whether the criteria for a substance use disorder are met.

Patients with a large number of comorbid psychiatric and medical disorders are likely to have a greater severity of symptoms and a higher likelihood of developing a chronic course. It is prudent to realize that such individuals will often require long periods of treatment related to comorbid conditions and situational crises generated from these other illnesses. In addition, as a result of debilitation from both physical and mental conditions, these patients may require high levels of management and support to accomplish activities of daily living. They may be fragile, and some treatment interventions may prove either too exhausting or more disabling. Consequently, patients with chronic PTSD accompanied by comorbid medical and other psychiatric disorders need a graduated plan of treatment that begins with a primarily supportive approach and evolves into treatment that is more directed at restoring previous function. Very fragile patients may need hospitalization if they become dangerous to themselves or others or if they become so affectively labile that they experience significant functional impairment (229).

## ► **E. HISTORY OF PREVIOUS TRAUMAS**

Exposure to previous trauma may modify vulnerability to subsequent trauma (32, 33), influence the development of PTSD (32, 33, 223), and complicate treatment and recovery. Recent

loss—particularly if sudden or unexpected—is also associated with an increased prevalence of PTSD and may also complicate treatment (62). Although immediate illness may be precipitated by a recent trauma, symptoms of ASD or PTSD (sleep disturbance, irritability, hyperarousal) may in fact be directly related to the more remote traumatic experience(s), including childhood sexual abuse. Psychotherapeutic interventions aimed at integrating traumatic experience and diminishing the effect of intrusive recollections must therefore target not only the precipitating trauma but the remote trauma as well.

## ► **F. AGGRESSIVE BEHAVIOR**

More than a half-century ago, Kardiner (198) noted that some patients with PTSD had problems with aggressive behavior that was frequently impulsive and episodic. More recent studies have documented increases in domestic violence, child abuse, and delinquency after disasters (15, 258–260). It has been postulated that with the development of PTSD, an increased expectation of danger and potential trauma occurs and results in an “anticipatory bias” (261) or an increased readiness for “flight, fight, or freeze.” This increased readiness for aggression, as well as decreased sleep associated with PTSD, may produce a reduced ability to tolerate mild or moderate slights, resulting in acts of aggression that are disproportionate to the level of provocation (262).

Little evidence addresses the treatment of heightened aggressiveness in individuals with PTSD. Based on the use of SSRI antidepressants in treating PTSD, there is reason to suggest use of these medications in patients with aggression in the context of PTSD. Observation for symptomatic exacerbations is warranted in the early phases of treatment, before the therapeutic benefits of pharmacotherapy are manifest. Anticonvulsants are sometimes suggested for management of irritability and aggression, but evidence for their efficacy is similarly sparse, with only a single small-scale open-label trial that found a modest effect of carbamazepine on irritability/aggression (160).

To the extent that aggressive behavior occurs in the context of reexperiencing symptoms (e.g., flashbacks), treatment approaches targeting this symptom cluster may also reduce aggression. Since aggressive behaviors are associated with states of both intoxication and withdrawal, concurrent treatment of comorbid substance use disorders may also reduce the likelihood of aggressive behavior.

## ► **G. SELF-INJURIOUS AND SUICIDAL BEHAVIORS**

Both acute and chronic response to trauma exposure may include self-harming behaviors that range from self-mutilation to disordered eating behaviors to abuse of alcohol and other substances (256, 257, 263–270). This response may occur particularly when the trauma induces stigma, shame, or guilt. Children and adults who have been traumatized are likely to redirect onto themselves the feelings of aggression they have toward others (267, 271, 272). Furthermore, studies consistently show a significant relationship between childhood sexual abuse and various forms of self-injury later in life, particularly self-starving, cutting, and suicide attempts (267). In fact, PTSD has demonstrated the strongest association with suicidal behaviors of any of the anxiety disorders (273, 274). Specifically, PTSD is associated with a sixfold increase in the likelihood of an initial suicide attempt, an odds ratio that is double that for other anxiety disorders and about half that for mood disorders (275). In addition, individuals with PTSD appear to have an equal or greater odds ratio for making a suicide plan and for making impulsive suicide attempts, compared to those with mood disorders or other anxiety disorders (275). Anxiety disorders including PTSD are also associated with an increased risk for suicide per se (276, 277). Thus, it is apparent that patients with PTSD are at increased risk for developing self-harming and suicidal behaviors (269).

The possible utility of SSRI antidepressants in treating self-harming or suicidal behaviors in individuals with PTSD is suggested by the utility of SSRIs in treating PTSD in general (123, 141, 144–146, 148, 150, 278–280). Observation for symptomatic exacerbations is warranted

in the early phases of treatment, before the therapeutic benefits of pharmacotherapy are manifest. Other pharmacotherapies may also be useful, although evidence for their efficacy is sparse. For example, one study showed carbamazepine to be effective for treatment of self-destructive behaviors (281), and a single, relatively small study suggested that lithium carbonate may also be helpful (282). Finally, although opiate receptor blockers have not been studied specifically in patients with ASD or PTSD, limited evidence suggests that such agents may decrease self-destructive behaviors in other populations (283).

Regarding psychological treatments for suicidal behavior in patients with PTSD, few studies are available. In addition, most studies of PTSD specifically exclude acutely suicidal patients; therefore, clinical judgment must augment the research to date. Thus, although many studies show that cognitive behavior therapy is effective in treating psychiatric disorders such as depression and PTSD, which can increase the risk for suicide, few studies have shown cognitive behavior therapy to be effective for reducing actual suicidal behavior and intent (284). As in other mental disorders associated with suicidal behavior, involving the patient's family members and other sources of support in the treatment plan may increase awareness of and vigilance for indications of the potential for deliberate self-harm or suicide.

## **PART B**

# **BACKGROUND INFORMATION AND REVIEW OF AVAILABLE EVIDENCE**

## **IV. DISEASE DEFINITION, EPIDEMIOLOGY, AND NATURAL HISTORY**

---

### **▶ A. CORE CLINICAL FEATURES**

The DSM-IV-TR criteria for ASD and PTSD are shown in Table 1 and Table 2, respectively. Table 4 compares the specific criteria used in making these diagnoses. For both ASD and PTSD, essential features are exposure to a traumatic event that need not be outside the normal range of human experience but that arouses “intense fear, helplessness, or horror” (DSM-IV-TR, p. 463), followed by development of characteristic symptoms. Exposure can occur through direct experience or through witnessing or learning about a traumatic event that caused “actual or threatened death,” “serious injury,” or “threat to the physical integrity” of oneself or others (DSM-IV-TR, p. 463). Both natural and human-made traumatic events have the potential to evoke these symptoms. Naturally occurring stressors include, for example, tornadoes, earthquakes, and medical illnesses. Human-made events include accidents, domestic and community violence, rape, assault, terrorism, and war. Some of these are singular events; others involve chronic or repeated exposure. In general, human-made events have been believed to cause more frequent and more persistent psychiatric symptoms and distress.

**TABLE 4. Comparison of DSM-IV-TR Diagnostic Criteria for Acute Stress Disorder (ASD) and Posttraumatic Stress Disorder (PTSD)**

Description of Criterion	ASD Criterion	PTSD Criterion
Characteristics of traumatic exposure Exposure to a traumatic event in which both of the following conditions were present: <ol style="list-style-type: none"> <li>1. the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others</li> <li>2. the person's response involved intense fear, helplessness, or horror</li> </ol>	Criterion A	Criterion A
Dissociative symptom cluster Either while experiencing or after experiencing the distressing event, the individual has three (or more) of the following symptoms: <ol style="list-style-type: none"> <li>1. a subjective sense of numbing, detachment, or absence of emotional responsiveness</li> <li>2. a reduction in awareness of his or her surroundings (e.g., "being in a daze")</li> <li>3. derealization</li> <li>4. depersonalization</li> <li>5. dissociative amnesia (i.e., inability to recall an important aspect of the trauma)</li> </ol>	Criterion B	— <sup>a</sup>
Reexperiencing cluster The traumatic event is persistently reexperienced in one (or more) of the following ways: <ol style="list-style-type: none"> <li>1. recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions</li> <li>2. recurrent distressing dreams of the event</li> <li>3. acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated)</li> <li>4. intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event</li> <li>5. physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event</li> </ol>	Criterion C (except item 5)	Criterion B



**TABLE 4.** Comparison of DSM-IV-TR Diagnostic Criteria for Acute Stress Disorder (ASD) and Posttraumatic Stress Disorder (PTSD) (*continued*)

Description of Criterion	ASD Criterion	PTSD Criterion
<p>Avoidance/numbing of response cluster</p> <p>Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following characteristics:</p> <ol style="list-style-type: none"> <li>1. efforts to avoid thoughts, feelings, or conversations associated with the trauma</li> <li>2. efforts to avoid activities, places, or people that arouse recollections of the trauma</li> <li>3. inability to recall an important aspect of the trauma</li> <li>4. markedly diminished interest or participation in significant activities</li> <li>5. feeling of detachment or estrangement from others</li> <li>6. restricted range of affect (e.g., unable to have loving feelings)</li> <li>7. sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span)</li> </ol>	<p>Criterion D (requires only marked avoidance of stimuli that arouse recollections of the trauma)</p>	<p>Criterion C</p>
<p>Arousal cluster</p> <p>Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following symptoms:</p> <ol style="list-style-type: none"> <li>1. difficulty falling or staying asleep</li> <li>2. irritability or outbursts of anger</li> <li>3. difficulty concentrating</li> <li>4. hypervigilance</li> <li>5. exaggerated startle response</li> </ol>	<p>Criterion E (requires only marked symptoms of anxiety or increased arousal)</p>	<p>Criterion D</p>

**TABLE 4.** Comparison of DSM-IV-TR Diagnostic Criteria for Acute Stress Disorder (ASD) and Posttraumatic Stress Disorder (PTSD) (*continued*)

Description of Criterion	ASD Criterion	PTSD Criterion
Duration of disturbance	Minimum of 2 days, maximum of 4 weeks	Greater than 1 month (acute PTSD is diagnosed if duration is less than 3 months; chronic PTSD if duration is 3 months or greater)
Temporal relationship to traumatic event	Occurs within 4 weeks	Usually occurs within 3 months (if onset occurs more than 6 months after stressor, delayed onset is specified)
Distress or impairment in functioning: The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning (or inability to pursue some necessary task in ASD)	Criterion F	Criterion F
Exclusion of other conditions Not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition Not better accounted for by brief psychotic disorder Not an exacerbation of a preexisting axis I or axis II disorder	Criterion H	— <sup>a</sup>

<sup>a</sup>Not included in PTSD criteria.

The criteria for ASD overlap substantially with but are not identical to those for PTSD (Table 4). Although core symptoms fall into characteristic symptom clusters for both diagnoses, ASD and PTSD differ in the numbers of symptoms from each cluster that are required to establish a diagnosis. For example, in addition to three or more dissociative symptoms and “marked avoidance of stimuli that arouse recollections of the trauma,” the diagnosis of ASD requires at least one reexperiencing symptom as well as “marked” anxiety or increased arousal. On the other hand, for a diagnosis of PTSD to be made, DSM-IV-TR stipulates that there be at least one reexperiencing symptom, two arousal symptoms, and three avoidance/numbing symptoms and that these symptoms be temporally related to the stressor. Symptoms in the reexperiencing cluster include “recurrent and intrusive recollections” of the event, recurrent distressing trauma-related dreams, acting or feeling as if the event were reoccurring, “intense psychological distress” with exposure to trauma cues, and physiological reactivity to traumatic cues (DSM-IV-TR, p. 464). Within the avoidance/numbing cluster, purposeful actions as well as unconscious mechanisms may be present and may include efforts to avoid trauma-related thoughts, feelings, or conversations; efforts to avoid activities, places, or people reminiscent of the trauma; inability to recall important aspects of the trauma; greatly decreased “interest or participation in previously enjoyed activities”; feeling detached or estranged; restricted range of affect; and a “sense of a foreshortened future” (DSM-IV-TR, p. 464). Increased arousal includes sleep disturbance, “irritability or outbursts of anger,” difficulty concentrating, hypervigilance, and exaggerated startle response (DSM-IV-TR, p. 464), all of which are generalized arousal responses and are not precipitated by reminders of the stressor.

The two disorders also differ in the duration of the disturbance and its temporal relationship to the traumatic stressor. For ASD, the disturbance occurs within 4 weeks of the traumatic event and is from 2 days to 4 weeks in duration. To qualify for a diagnosis of PTSD, symptoms must be present for more than 1 month. If symptom duration is less than 3 months, acute PTSD is diagnosed, whereas chronic PTSD is diagnosed when symptoms persist for 3 months or longer. Although symptoms of PTSD usually begin within 3 months of exposure, DSM-IV-TR also allows for delayed onset with symptoms that appear months or even years after the event. Finally, for both ASD and PTSD, the severity of symptoms must be sufficient to cause “clinically significant distress” or impaired functioning (DSM-IV-TR, pp. 468, 472).

## ▶ **B. ASSOCIATED FEATURES**

A number of additional features may be associated with PTSD. According to DSM-IV-TR, these features include somatic complaints, shame, despair, hopelessness, impaired affect modulation, social withdrawal, survivor guilt, anger, impulsive and self-destructive behavior, difficulties in interpersonal relationships, changed beliefs, and changed personality. Difficulty seeking and sustaining medical care has also been observed (285). Symptoms such as inappropriate guilt, shame, or hopelessness may be indicative of comorbid depression that requires separate intervention, and other symptoms, such as somatic complaints, may represent common phenomena that are associated with anxiety disorders but are not necessary for the diagnosis of either ASD or PTSD. Finally, symptoms of trauma-related dissociation are essential to the diagnosis of ASD but are not necessary for the diagnosis of PTSD. Nonetheless, a previous history of peritraumatic dissociation (and ASD) may be of clinical significance in patients with PTSD, as studies have demonstrated that such a history predicts greater severity and chronicity of PTSD (7, 286, 287).

## ▶ **C. DIFFERENTIAL DIAGNOSIS**

The differential diagnosis of ASD and PTSD includes a broad range of psychiatric and physical diagnoses as well as normative responses to traumatic events. Individuals who are exposed to events that fulfill criterion A for ASD or PTSD often experience some transient symptoms that

differ from those of ASD or PTSD only in their duration or in the associated level of dysfunction or distress. In some professions (e.g., military, firefighters, police, emergency medical personnel), exposure to criterion A events is inevitable. If symptoms do not meet the criteria for ASD or PTSD but are persistent or associated with dysfunction or distress, a V code diagnosis (e.g., V62.2, occupational problem) may be appropriate.

Establishing a differential diagnosis also requires that ASD be differentiated from PTSD. For a single discrete traumatic event, ASD and PTSD can be readily distinguished from one another based on the time that has passed since the trauma. However, for less discrete or reoccurring traumas such as repetitive domestic violence, the distinctions between ASD and PTSD may be less clear. Although no convention or consensus exists regarding the classification of recurrent symptoms (for more than 1 month) during the course of repetitive episodic trauma, it may be best to conceptualize this symptom presentation as PTSD rather than as recurrent episodes of ASD. Clearly, eliminating the source or threat of continued violence and injury is critical to ultimate resolution of posttraumatic symptoms, regardless of diagnostic classification. As noted earlier, beyond duration of symptoms, the major distinguishing feature between ASD and PTSD is the emphasis in the former on dissociative symptoms. Although persons with ASD often develop PTSD, this is not invariably true. PTSD may also occur in persons who manifest few or even no symptoms of ASD in the period immediately after trauma (6, 7, 9). In patients with subthreshold or full symptoms of PTSD for less than 1 month who do not experience dissociative symptoms sufficient to meet the DSM-IV-TR criteria for ASD, the illness would be best characterized as an adjustment disorder in DSM terms. Such patients would also meet the diagnostic criteria for acute stress reaction, as defined by ICD-10. The differential diagnosis also includes medical disorders as well as a number of other psychiatric disorders (Table 5).

The fact that many of these disorders occur comorbidly with ASD or PTSD further complicates diagnosis. For example, a substantial proportion of trauma-exposed veterans (20, 247), refugees (292), and civilians (12, 293) develop symptoms consistent with major depressive disorder. Mood disorders are also an established risk factor for the development of PTSD in newly exposed individuals (12, 14, 34). Symptoms such as insomnia, poor concentration, and diminished interest in activities may be present with ASD and PTSD as well as with major depression. In addition, the restricted affective range that may accompany the numbing of responses with PTSD may resemble the restricted affect seen in depressed patients. It is important to note that if the DSM-IV-TR criteria are met, a major depressive episode can be diagnosed in conjunction with ASD or PTSD.

Trauma-exposed populations and patients with PTSD frequently experience comorbid substance-related disorders (256, 257, 294–299). Patients with PTSD also manifest increased physical complaints (76–79, 300, 301) and comorbid medical conditions (302). Although DSM-IV excluded complicated or prolonged grief as an axis I diagnosis (because of a lack of empirical evidence regarding symptoms), some investigators have proposed criteria for a diagnosis of complicated grief disorder based on patterns of prolonged bereavement characterized by persistence, intensity, intrusive recollections or images of the death, preoccupation with the loss, and avoidance of reminders (303). Furthermore, there is evidence that these symptoms may be more distressing after an unnatural or violent death. Such symptoms overlap with both major depressive disorder and PTSD, but persons may acknowledge these symptoms without meeting the criteria for either diagnosis. Here, preoccupation with the suddenness, violence, or catastrophic aspects of traumatic loss may be independent from and may interfere with the normal bereavement process (304). Consensus criteria for “traumatic grief” have been developed; these criteria overlap with those of complicated grief but incorporate additional symptoms of distress related to cognitive reenactment of the death, terror, and avoidance of reminders (289). Once again, studies that address treatment for these phenomena distinct from treatment for PTSD or depression are presently lacking. Nonetheless, complicated or traumatic grief as well as bereavement must be considered in the differential diagnosis for persons who have experienced a traumatic loss.

Finally, since childhood trauma may be a common antecedent to the development of personality (particularly cluster B) disorders in adulthood, and associated features of personality disorders and PTSD overlap (e.g., difficulty with affect modulation, impulsivity, irritability, comorbid substance abuse), PTSD symptoms may be “masked” by an underlying personality disorder. Numerous reports describe childhood trauma in adults with borderline personality disorder, and other reports describe childhood trauma as a root cause of adult PTSD. However, the extent to which symptoms may be misattributed to either PTSD or a personality disorder has not been well studied. Therefore, personality disorders must be considered in the differential diagnosis either as the primary etiology for symptoms or as comorbid illnesses.

## ► D. EPIDEMIOLOGY

Exposure to a traumatic event, the essential element for development of ASD or PTSD, is a relatively common experience, although the specific rates of such experiences within a population sample will vary with the criteria used to define a potential trauma as well as with the sample characteristics and the interviewing method (e.g., telephone survey versus face-to-face interview, clinician versus lay interviewer, structured versus unstructured interview), as reviewed by Brewin and colleagues (222). For example, using DSM-III-R criteria, which required that the event be outside the range of normal human experience, researchers in the National Comorbidity Survey (4) assessed 5,877 individuals ages 15–54 years with the Diagnostic Interview Schedule (DIS) and the Composite International Diagnostic Interview, administered by experienced nonclinician interviewers. They found that more than one-half of the subjects had experienced a traumatic event during their lifetime, with most people having experienced more than one. Giaconia and colleagues (305) also used the DSM-III-R version of the DIS and found that by age 18 years, more than two-fifths of youths in a community sample had been exposed to an event that was severe enough to qualify for a diagnosis of PTSD. Using structured telephone interviews in a national sample of 4,008 adult women, Resnick and colleagues (306) found a lifetime rate of exposure to any type of traumatic event of 69%. Using the DSM-IV version of the DIS, Breslau and colleagues (5) examined trauma exposure and the diagnosis of PTSD in a telephoned community sample of 2,181 individuals in the Detroit area and found that the lifetime prevalence of trauma exposure was 89.6%. The most prevalent types of events were the sudden unexpected death of a close relative or friend (60.0%) or learning of trauma to a close relative or friend (62.4%).

Overall exposure to traumatic events may be somewhat greater in men than in women (4, 5), although the gender difference in the lifetime prevalence of such exposure is relatively small (60.7% for men versus 51.2% for women in the study of Kessler and colleagues [4], and 92.2% for men versus 87.1% for women in the study of Breslau and colleagues [5]). In addition, men and women differ in the types of events to which they are exposed. For example, in the National Comorbidity Survey, 0.7% of men versus 9.2% of women had a lifetime experience of being raped, whereas 19.0% of men but only 6.8% of women had been threatened with a weapon and 6.6% of men but no women had experienced combat (4). In the Detroit Area Survey of Trauma (5), a similar pattern was noted, with women being more likely than men to report rape (9.4% versus 1.1%) or other sexual assault (9.4% versus 2.8%) and men being more likely than women to report other types of assaultive violence, including being mugged or threatened with a weapon (34.0% versus 16.4%) and being shot or stabbed (8.2% versus 1.8%).

Exposure to traumatic events also varies with age, showing consistent declines with age across multiple studies. For example, Norris (307) found a strong trend for decreases in both past-year and lifetime exposure with increasing age in a nonrandom sample of 1,000 individuals from four cities in southeastern states. Bromet and colleagues (14) analyzed data from the National Comorbidity Survey and found that the risk of experiencing a traumatic event was greatest in the 15- to 24-year-old cohort and decreased in subsequent age cohorts. Similarly, Breslau and colleagues (5)

**TABLE 5. Psychiatric Diagnoses Often Applicable to Injured Trauma Survivors Treated in the Acute Care Medical Setting**

Diagnosis <sup>a</sup>	Diagnostic Considerations			
	Symptomatic Criteria	Functional Criteria	Time Course	Acute Care Considerations
Acute stress disorder (ASD)	<p>A. Exposure to a traumatic event in which the person experienced or witnessed a life-threatening event that was associated with intense emotions (e.g., physical injury)</p> <p>B. Either while experiencing the event or after, the person experiences three or more dissociative symptoms.</p> <p>C. The event is reexperienced.</p> <p>D. Avoidance of reminders of the event</p> <p>E. Symptoms of arousal</p>	Symptoms are associated with clinically significant impairments in social, occupational, or physical function.	Diagnosis can be made between 2 and 30 days after the event.	Not all injured patients with immediate distress will experience three dissociative symptoms.
46 Posttraumatic stress disorder (PTSD)	<p>A. Exposure to a traumatic event in which the person experienced or witnessed a life-threatening event that was associated with intense emotions (e.g., physical injury)</p> <p>B. The event is persistently reexperienced</p> <p>C. Persistent avoidance of reminders of the event</p> <p>D. Persistent arousal symptoms</p>	Symptoms are associated with clinically significant impairments in social, occupational, or physical function.	Diagnosis must be made at least 1 month after the event.	Patient's symptoms frequently appear before the 1-month point.
Major depressive episode	Five or more of the following symptoms: depressed mood, <sup>b</sup> diminished interest in pleasurable activities, <sup>b</sup> weight loss or gain, insomnia or hypersomnia, agitation or retardation, fatigue or energy loss, feelings of worthlessness, poor concentration, and suicidal ideation	Symptoms are associated with clinically significant impairment in social, occupational, or physical function.	Symptoms must be present for 2 weeks.	Major depressive episode can be diagnosed in conjunction with ASD or PTSD. Injured trauma survivors frequently present with multiple symptoms of a depressive episode early on (i.e., before 2 weeks after the traumatic injury).

**TABLE 5. Psychiatric Diagnoses Often Applicable to Injured Trauma Survivors Treated in the Acute Care Medical Setting (continued)**

Diagnosis <sup>a</sup>	Diagnostic Considerations			
	Symptomatic Criteria	Functional Criteria	Time Course	Acute Care Considerations
Traumatic grief	This evolving diagnostic category can be used when the events that lead to a patient's or relative's visit to the acute care setting involve sudden unanticipated loss. The symptoms of traumatic grief involve distressing thoughts and experiences related to reunion, longing, and searching for the deceased loved one (289–291).	The disturbance causes clinically significant impairment in social, occupational, or other important areas of functioning.	Duration of disturbance is at least 2 months.	Traumatic grief is applicable to patients who have experienced the death of a significant other.
Adjustment disorder	A. Development of emotional or behavioral symptoms in response to an identifiable stressor. Symptoms can include depression, anxiety, conduct disturbance, or other emotional disturbance. B. The symptoms or behaviors are clinically significant, as evidenced by marked distress.	Emotional or behavioral symptoms are associated with marked impairment in social, role, or physical function.	Onset occurs within 3 months after the traumatic injury.	DSM-IV-TR suggests that the adjustment disorder diagnosis be used for patients who develop a symptom pattern that is not entirely consistent with the criteria for ASD/PTSD. Nonspecific symptomatic requirements make adjustment disorder a useful diagnosis for the many patients who experience posttraumatic behavioral and emotional disturbances that include symptoms that do not fit into other diagnostic rubrics (e.g., patients who present with marked somatic symptom amplification).

*Source.* Adapted with permission from *Diagnostic and Statistical Manual of Mental Disorders*, 4th Edition, Text Revision. Washington, DC, American Psychiatric Association, 2000. Copyright 2000. American Psychiatric Association; and from *Seminars in Clinical Neuropsychiatry*, vol. 8, Zatzick D., "Posttraumatic stress, functional impairment, and service utilization after injury: a public health approach," pp. 149–157, Copyright 2003, with permission from Elsevier.

<sup>a</sup>Often, posttraumatic symptoms may be present that are insufficient to meet criteria for the diagnoses listed in this table. In such cases, V code diagnoses would be indicated, as would supportive therapy, psychoeducation, and continued monitoring for the development of further psychiatric disorder(s). <sup>b</sup>At least one of the five symptoms must be either depressed mood or diminished interest in pleasurable activities and shall not be due to a general medical condition or mood-incongruent delusions or hallucinations.

found that in all classes of traumas studied, peak exposures to traumatic events occurred in persons ages 16–20 years, with subsequent declines in exposure rates with age.

The lifetime prevalence of ASD is unclear, but a number of community-based studies have examined the prevalence of PTSD. Here, too, the reported rates vary with the specific diagnostic criteria employed, the interviewing method, and the sample characteristics. For example, in a study of the data for 2,985 participants from a central North Carolina community who were assessed as part of the Epidemiologic Catchment Area (ECA) survey, Davidson and colleagues (242) found a lifetime prevalence for DSM-III PTSD of 1.3%. Helzer and colleagues (308) found a lifetime PTSD prevalence of 1% in the St. Louis ECA sample. Using DSM-III-R criteria, Kessler and colleagues (4) found an estimated lifetime prevalence of PTSD of 7.8% in the National Comorbidity Survey, whereas Giaconia and colleagues (305) found that more than 6% of youths in a community sample met the criteria for a lifetime diagnosis of PTSD.

The likelihood of developing PTSD after having been exposed to a traumatic event (i.e., the conditional risk of PTSD) varies widely with the specific experience. Overall in the Detroit Area Survey of Trauma, for example, 9.2% of trauma-exposed persons developed PTSD, but PTSD developed in about half of those who were raped or held captive, tortured, or kidnapped, compared to only 2.2% of those who learned of the rape, attack, or injury of a close relative (5). In the women studied by Resnick and colleagues (306), rates of PTSD were significantly greater in crime victims than in noncrime victims (25.8% versus 9.4%).

General population studies typically find a significantly higher lifetime prevalence of PTSD in women, with rates that are consistently about twice those seen in men (4, 5, 222, 242, 308). The absolute rates for a lifetime diagnosis of PTSD again vary with the definition and severity of the traumatic stressor. Using the DSM-III criteria as part of the ECA survey, Helzer and colleagues (308) found that 1.3% of women and 0.5% of men met the criteria for a lifetime diagnosis of PTSD, and Davidson and colleagues (242) found lifetime rates of PTSD of 1.8% in women and 0.9% in men. In contrast, using the DSM-III-R criteria in the National Comorbidity Survey, Kessler and colleagues (4) found a lifetime prevalence for PTSD of 10.4% in women and 5.0% in men, and Breslau and colleagues (5, 223), using the DSM-IV criteria, found the lifetime prevalence of PTSD to be 13.0% in women, compared to 6.2% in men. In terms of the relative likelihood of developing PTSD after having experienced a traumatic event, Kessler and colleagues (4) found more than a twofold increase in the conditional risk of PTSD in women, compared with men (20.4% versus 8.1%). These gender differences in rates of PTSD do not necessarily imply that women are more likely to develop PTSD, *per se*; the differences may be explained by other factors that increase risk for women (15), such as the greater likelihood of women's experiencing rape and other sexual assaults, which carry a high conditional risk of developing PTSD. In addition, since a history of mood disorder increases the subsequent risk of developing PTSD in response to a stressor (14), the greater prevalence of such disorders among women may influence their likelihood of developing PTSD. Furthermore, specific aspects of the traumatic event, such as fear, threat, surprise, and meaning, may influence the victim's response (309).

The literature provides inconsistent information on the relationship between age and the risk of developing PTSD. Breslau and colleagues (33), in a representative community sample in southeast Michigan, found no relationship between age and risk of PTSD. In the National Comorbidity Survey, Kessler and colleagues (4) found some variations in the lifetime prevalence of PTSD by birth cohort, but men had the highest rates in the 45- to 54-year-old cohort, whereas women had the highest rates in the 25- to 34-year-old cohort. In terms of the conditional risk of developing PTSD after adjustment for the type of trauma exposure, a subsequent analysis of the National Comorbidity Survey data also showed variations in risk with age among men but a greater risk for PTSD among women in younger age cohorts (14). Brewin and colleagues (222) found weak effects of age in a meta-analysis of risk factors for PTSD but suggested that the differences may reflect confounding factors.



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 through 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 or to a waiting-list control group. A total of 114 subjects completed follow-up at 3 and/or 6 months. The treatment groups experienced significant reductions in the number of nightmares per week and significant improvement in sleep, relative to the control group. These improvements were noted at the 3-month follow-up and were sustained without further intervention or contact between 3 and 6 months. Furthermore, PTSD symptoms decreased in a majority of treated subjects but remained the same or worsened in a majority of control subjects. Forbes et al. (185) employed the same intervention for combat-related nightmares in 12 Vietnam veterans with PTSD and found significant reductions in targeted nightmares and improvements in PTSD symptoms. These changes persisted at 12-month follow-up (370). Similar success in female rape victims with chronic PTSD was reported in a study comparing cognitive processing therapy (another non-exposure-based cognitive therapy) to prolonged exposure and a waiting-list condition (181).

Group exposure therapy has also been found to be more effective than minimal attention groups. Falsetti et al. (371) reported on the results of a pilot study of a manualized treatment that included multiple cognitive and behavioral strategies, which they called multiple channel exposure therapy (M-CET), for patients with PTSD and comorbid panic disorder. They compared M-CET with a minimal attention group. Subjects recruited from an outpatient department and a local rape crisis center were randomly assigned to either 12 weeks of once-weekly M-CET group therapy or to a minimal attention group that received bimonthly supportive telephone counseling. Women in the control condition were offered free treatment after completing their participation in that condition. Posttreatment, only 8.3% of the subjects in the M-CET condition met the Clinician-Administered PTSD Scale diagnostic criteria for PTSD, compared with 66.7% of the control subjects. Ninety-three percent of the control group reported at least one panic attack in the past month, compared with only 50% of the treatment group. Glynn et al. (372) examined a behavioral family therapy for 42 Vietnam veterans with combat-related PTSD and a family member for each veteran (typically the veteran's wife). Three conditions were used: a course of twice-weekly direct exposure therapy, the same course of exposure followed by 16 sessions of behavioral family therapy, or a waiting-list condition.

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 90-minute 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

1 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.

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 pre-treatment 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 4-month 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, Bor-

dow 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 antidepressant 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).

## **3. 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, placebo-controlled 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 step-wise 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 self-harmful, 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.

## INDIVIDUALS AND ORGANIZATIONS THAT SUBMITTED COMMENTS



Jon G. Allen, Ph.D.  
 William Arroyo, M.D.  
 J. Wesson Ashford, M.D., Ph.D.  
 Donald Banzhaf, M.D.  
 Romano Biancoli, Ph.D.  
 Evelyn Bromet, Ph.D.  
 David W. Brook, M.D.  
 Bonnie J. Buchele, Ph.D.  
 Fredric Busch, M.D.  
 Peter Roy Byrne, M.D.  
 Judy Cohen, M.D.  
 Mary Ann Cohen, M.D.  
 Mirean Coleman, M.S.W., L.I.C.S.W., C.T.  
 Joan Cook, M.D.  
 Jonathan Davidson, M.D.

Cleto DiGiovanni, Jr., M.D.  
 Lois T. Flaherty, M.D.  
 Jane Meschan Foy, M.D.  
 Terry Fullerton, Ph.D.  
 Leslie Hartley Gise, M.D.  
 Robert M. Goisman, M.D.  
 Michael Good, M.D.  
 Tina Haynes, M.T.-B.C.  
 Al Herzog, M.D.  
 Mardi Horowitz, M.D.  
 Craig L. Katz, M.D.  
 Harold Kudler, M.D.  
 Barry Landau, M.D.  
 Melvin Lansky, M.D.  
 Dori Laub, M.D.

Fred M. Levin, M.D., S.C.  
Brett Litz, Ph.D.  
John Markowitz, M.D.  
Julia Matthews, Ph.D., M.D.  
Miles McFall, Ph.D.  
Thomas A. Mellman, M.D.  
Thomas Neylan, M.D.  
Carol S. North, M.D.  
Andrei Novac, M.D.  
John D. O'Brien, M.D.  
Herbert S. Peysner, M.D.  
Roger Pitman, M.D.  
Charles W. Portney, M.D.

Murray Raskind, M.D.  
Barbara R. Rosenfeld, M.D.  
Barbara O. Rothbaum, M.D.  
Patricia Rowell, R.N., Ph.D., C.N.P.  
Diane H. Schetky, M.D.  
David Servan-Schreiber, M.D., Ph.D.  
Jonathan Shay, M.D., Ph.D.  
Bradley D. Stein, M.D., Ph.D.  
Robert Stern, M.D., Ph.D.  
Nicholas E. Stratas, M.D.  
Elisa G. Triffleman, M.D.  
Bessel A. van der Kolk, M.D.  
Sidney Zisook, M.D.

American Academy of Child and Adolescent Psychiatry  
American Academy of Clinical Psychiatrists  
American Academy of Pediatrics  
American Academy of Psychoanalysis and Dynamic Psychiatry  
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.

1. 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](http://www.oqp.med.va.gov/cpg/PTSD/PTSD_Base.htm) [G]
2. 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]
3. 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]
4. 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]
5. 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]
6. 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]
10. 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]
14. 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]
15. 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]
20. Shalev AY, Peri T, Rogel-Fuchs Y, Ursano RJ, Marlowe D: Historical group debriefing after combat exposure. *Mil Med* 1998; 163:494–498 [B]
21. 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]
24. 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]
26. 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 self-rating 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]
40. Beckham JC: Smoking and anxiety in combat veterans with chronic posttraumatic stress disorder: a review. *J Psychoactive Drugs* 1999; 31:103–110 [F]
41. 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]
52. 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, Poccock H, Pottle S: Deliberate self-harm: clinical and socio-economic characteristics of 368 patients. *Br J Psychiatry* 1975; 127:564–574 [G]
66. 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]
99. 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–]
102. 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]
104. Breslau N, Davis GC, Peterson EL, Schultz L: Psychiatric sequelae of posttraumatic stress disorder in women. *Arch Gen Psychiatry* 1997; 54:81–87 [D]
105. McCarroll JE, Ursano RJ, Fullerton CS, Liu X, Lundy A: Somatic symptoms in Gulf War mortuary workers. *Psychosom Med* 2002; 64:29–33 [G]
106. 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]
109. 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, Lonnberg 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]
145. 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, flexible-dosage trial. *J Clin Psychiatry* 2001; 62:860–868 [A]
146. 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]
147. 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]
149. 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]
150. 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]
160. 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]
165. 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 open-label study. *Int Clin Psychopharmacol* 2001; 16:331–337 [B]
166. 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]
167. 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]
180. 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]
186. 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–]
189. 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]
201. 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](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]
205. 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]
226. 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]
245. 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]
246. 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]
249. 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]
252. 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]

263. van der Kolk BA, Fislis 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]
265. 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]
267. 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]
271. 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]
274. 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 Heringen K. Chichester, UK, John Wiley & Sons, 2000, pp 179–192 [F]
277. Institute of Medicine: *Reducing Suicide: A National Imperative*. Washington, DC, National Academies Press, 2002 [E]
278. 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]
279. Londeborg 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 tranlycypromine. *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]
  309. 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]
  313. 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 posttraumatic 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, Phommavanh 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]



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]
351. 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]
359. 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]
361. 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 disorder 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 posttraumatic 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]
395. 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 JE, 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, Tarrrier 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, double-blind 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, Kopsi 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 posttraumatic 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 posttraumatic 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 DE, 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]

