VA/DoD CLINICAL PRACTICE GUIDELINE FOR MANAGEMENT OF POST-TRAUMATIC STRESS

Department of Veterans Affairs

Department of Defense

Prepared by:

The Management of Post-Traumatic Stress Working Group

With support from:
The Office of Quality and Safety, VA, Washington, DC
&
Quality Management Division, United States Army MEDCOM

QUALIFYING STATEMENTS

The Department of Veterans Affairs (VA) and The Department of Defense (DoD) guidelines are based on the best information available at the time of publication. They are designed to provide information and assist in decision-making. They are not intended to define a standard of care and should not be construed as one. Also, they should not be interpreted as prescribing an exclusive course of management.

Variations in practice will inevitably and appropriately occur when providers take into account the needs of individual patients, available resources, and limitations that are unique to an institution or type of practice. Every healthcare professional who is making use of these guidelines is responsible for evaluating the appropriateness of applying them in any particular clinical situation.

Version 2.0 – 2010

Table of Contents

	Page
INTRODUCTION	**4**
Guideline Update Working Group	**11**
ALGORITHMS AND ANNOTATIONS	
CORE Module: Post-Traumatic Stress, Screening	**15**
Module A: Management of Acute Stress Reaction and Prevention of PTSD	**30**
Module B: Management of Post-Traumatic Stress Disorder (PTSD)	**57**
TREATMENT INTERVENTIONS	
Module I: Treatment Interventions for Post-Traumatic Stress	**102**
I1 – Early Interventions to Prevent PTSD	
I2 – Treatment of PTSD	
I3 – Management of Specific Symptoms	
APPENDICES	
Appendix A. Guideline Development Process	**199**
Appendix B. Acronym List	**206**
Appendix C. PTSD Screening Tools	**209**
Appendix D. Participant List	**213**
Appendix E. Bibliography	**222**

LIST OF TABLES

Table CORE - 1 Common Signs & Symptoms Following Exposure to Trauma 25

Table A - 1 Early Interventions after Exposure to Trauma (<4 days after exposure).....35
Table A - 2 Key Elements of Psychological First Aid (PFA) 37
Table A - 3 Diagnostic criteria for 308.3 Acute Stress Disorder (DSM-IV) 41
Table A - 4 Early Interventions after Exposure to Trauma (4 to 30 days after exposure) 46

Table B - 1 Common Symptoms following Exposure to Trauma 59
Table B - 2 Components of Functional Assessment 70
Table B - 3 Diagnostic criteria for Post-Traumatic Stress Disorder (DSM-IV) 79

Table I - 1 Early Interventions after Exposure to Trauma (4 to 30 days after exposure) 102
Table I - 2 Brief Psychotherapy Studies to Prevent the Development of PTSD 109
Table I - 3 Pharmacological Studies to Prevent the Development of PTSD 113
Table I - 4 Psychotherapy Interventions for Treatment of PTSD 115
Table I - 5 Group Therapy in PTSD (Shea et al., 2009) 136
Table I - 6 Pharmacotherapy Interventions for Treatment of PTSD 149
Table I - 7 Pharmacological Studies for Treatment of PTSD 156
Table I - 8 Symptom Response by Drug Class and Individual Drug (based on controlled trials) 160
Table I - 9 Drug Details 161
Table I - 10 Adjunctive Problem-Focused Method/Services 168
Table I - 11 Pharmacological Studies - Prazosin for Sleep Disturbances 188

INTRODUCTION

This update of the Clinical Practice Guideline for the Management of Post-Traumatic Stress was developed under the auspices of the Veterans Health Administration (VHA) and the Department of Defense (DoD), pursuant to directives from the Department of Veterans Affairs (VA). VHA and DoD define clinical practice guidelines as:

> *"Recommendations for the performance or exclusion of specific procedures or services derived through a rigorous methodological approach that includes:*
>
> *Determination of appropriate criteria, such as effectiveness, efficacy, population benefit, or patient satisfaction and a literature review to determine the strength of the evidence in relation to these criteria."*

This 2010 VA/DoD Post-Traumatic Stress guideline update builds on the 2004 VA/DoD *Clinical Practice Guideline for the Management of Post-Traumatic Stress.* The 2004 Post-Traumatic Stress Guideline was the first effort to bring evidence-based practice to clinicians providing care to trauma survivors and patients with stress disorders in the VA and DoD. The development of the Guideline originated with recognition of the need to diagnose and treat Post-Traumatic Stress among the military and veteran population. The Guideline presented evidence-based recommendations that were thoroughly evaluated by practicing clinicians and reviewed by clinical experts from the VHA and DoD.

Algorithms:

The VA/DoD also utilized an algorithmic approach for the 2004 Guideline for the Management Post-Traumatic Stress. This guideline update has also been developed using an algorithmic approach to guide the clinician in determining the care and the sequencing of the interventions on a patient-specific basis. The clinical algorithm incorporates the information that is presented in the guideline in a format that maximally facilitates clinical decision-making. The use of the algorithmic format was chosen because such a format improves data collection, facilitates clinical decision-making, and changes in patterns of resource use. However, *this should not prevent providers from using their own clinical expertise in the care of an individual patient.* Guideline recommendations are intended to support clinical decision-making and should never replace sound clinical judgment.

During the past 6 years, a number of well-designed randomized controlled trials of pharmacological and psychotherapeutic interventions for post-traumatic stress have been conducted. Therefore, the goal of this update is to integrate the results of this research and update the recommendations of the original guideline to reflect the current knowledge of effective treatment intervention. As in the original guideline, this update will explore the most important research areas of intervention to prevent the development of PTSD in persons who have developed stress reaction symptoms after exposure to trauma.

Target Population:

This guideline applies to adult patients with post-traumatic stress who are treated in any VA or DoD clinical setting.

Audiences:

The guideline is relevant to all healthcare professionals who are providing or directing treatment services to patients with post-traumatic stress at any VA/DoD healthcare setting.

Post-Traumatic Stress:

Post-traumatic stress consists of a spectrum of traumatic stress disorders—hence, this Clinical Practice Guideline for the Management of Post-Traumatic Stress. These disorders can be arranged along a temporal axis, from Acute Stress Reaction, to Acute Stress Disorder, Acute PTSD, and Chronic PTSD. Each of these

may be associated with serious mental and physical co-morbidities. Some survivors will experience only a part of this spectrum, while others will progress through the entire range.

Acute stress reaction (ASR) is not a DSM IV diagnosis and is used in this guideline to refer to a range of transient conditions that develop in response to a traumatic event. Onset of at least some signs and symptoms may be simultaneous with the trauma itself or within minutes of the traumatic event and may follow the trauma after an interval of hours or days. In most cases symptoms will disappear within days (even hours). **Combat and Operational Stress Reaction** (COSR) reflects acute reactions to a high-stress or combat-related event. ASR/COSR can present with a broad group of physical, mental, and emotional symptoms and signs (e.g., depression, fatigue, anxiety, decreased concentration/memory, hyperarousal, and others) that have not resolved within 4 days after the event, and after other disorders have been ruled out.

Acute stress disorder (ASD), a diagnosis defined by DSM IV, occurs when the individual has experienced trauma(s) as described above, has symptoms lasting more than two days, but less than one month after exposure to the trauma (may progress to PTSD if symptoms last more than one month), and exhibits re-experiencing, avoidance, increased arousal and at least three out of five dissociative symptoms.

Post-traumatic stress disorder (PTSD) is a clinically significant condition with symptoms continuing more than one month after exposure to a trauma that has caused significant distress or impairment in social, occupational, or other important areas of functioning. Patients with PTSD may exhibit persistent re-experiencing of the traumatic event(s), persistent avoidance of stimuli associated with the trauma, numbing of general responsiveness (not present before the trauma), and persistent symptoms of increased arousal (not present before the trauma). PTSD can also have a delayed onset, which is described as a clinically significant presentation of symptoms (causing significant distress or impairment in social, occupational, or other important areas of functioning) at least 6 months after exposure to trauma.

PTSD is further sub-divided into Acute PTSD (symptoms lasting more than one month, but less than three months after exposure to trauma) and Chronic PTSD (symptoms lasting more than three months after exposure to trauma). PTSD can appear alone (presenting with common symptoms of PTSD) or more commonly with other co-occurring conditions (persistent difficulties in interpersonal relations, mood, chronic pain, sleep disturbances, somatization, and profound identity problems) or psychiatric disorders (meeting DSM criteria for another disorder, such as substance abuse, depression, and anxiety disorder).

OEF/OIF veterans and service members who have sustained a concussion (mild-TBI) in the combat environment are often at significantly greater risk of PTSD. Moreover, the diagnosis of either condition may be complicated by the fact that PTSD is associated with generalized health symptoms, including neurocognitive impairment and other symptoms in the persistent post-concussion syndrome definition.

Evidence-based practices to prevent and treat PTSD include screening, cognitive behavioral therapies, and medications. There are many new strategies involving enhancement of cognitive fitness and psychological resilience to reduce the detrimental impact of trauma. In terms of screening, evidence suggests that identifying PTSD early and quickly referring people to treatment can shorten their suffering and lessen the severity of their functional impairment. Several types of cognitive behavioral therapies, counseling, and medications have been shown to be effective in treating PTSD.

The VA and DoD Healthcare systems have undergone significant changes in the past 10-15 years that are transforming the two into an integrated system that provides high-quality care. In response to the increased demands for services to treat OEF/OIF veterans with PTSD, these systems have invested resources in expanding outreach activities, enhancing the availability and timeliness of specialized PTSD services..

Post-Traumatic Stress in VA population:

The numbers of veterans seeking and receiving treatment for post-traumatic stress in general and PTSD, in particular, continue to increase. In a follow-up to a study by Dohrenwend et al. (2006), 9.1 percent of Vietnam veterans sampled still suffered from symptoms of PTSD in 1990. During a five year span (2004-2008), the number of unique veterans seeking help for PTSD in the VA system increased from 274,000 to 442,000. Also, according to a review of several studies investigating the prevalence of PTSD in U.S. Veterans of the first Persian Gulf War, the Board on Population Health and Public Health Practice at the National Academies of Science (2008) reported that the PCL-based prevalence of PTSD in a sample of

11,441 veterans was 12.1 percent. This review also cited evidence that ten years after the 1990 Gulf War, 6.2 percent of a sample of veterans still suffered from PTSD.

The number of Iraq and Afghanistan veterans who have been separated from service, seen in U.S. Veteran's Administration healthcare facilities, and diagnosed with PTSD was reported by Seal and colleagues (2007). Of the 103,788 veterans included in this review, the overall prevalence of PTSD was 13 percent, higher than any other mental health diagnostic category reported by these authors. The VA's Uniform Services Handbook sets standards for mental healthcare across VA facilities and is intended to both improve quality of care and facilitate implementation of evidence-based practices. In recent years, the exponential increase in clinical services for veterans with PTSD has been driven by the combination of improved diagnostic and treatment techniques for all stress-related disorders, the needs of veterans from past wars as far back as World War II, the co-morbid conditions many veterans experience in addition to PTSD (chronic medical conditions, SUD), and the ongoing nature of the current wars in Iraq and Afghanistan.

Post-Traumatic Stress in DoD population:

A number of studies have been conducted to estimate the prevalence and incidence of PTSD in military personnel during the Iraq and Afghanistan wars. These studies have shown high consistency in rates, when grouped according to study population (e.g., studies involving Army or Marine combat infantry units versus studies involving samples of the deploying population at large, including personnel from support units or services not involved in direct combat).

One of the first and most cited epidemiological surveys to provide estimates of PTSD prevalence in military personnel who served in Afghanistan or Iraq was published by Hoge et al. (2004). The prevalence of PTSD 3 months post-deployment among infantry soldiers and Marines who returned from high-intensity combat in Iraq was 12.9 percent and 12.2 percent, respectively (n = 894 soldiers, 815 Marines), based on a stringent definition for PTSD supported in a study by Terhakopian et al. (2008) (PCL score of at least 50 combined with DSM criteria). By comparison, the rate among soldiers who had deployed to Afghanistan, where there was very low-intensity combat at that time, was 6.2 percent, and the baseline rate in a group of soldiers before deployment was 5 percent. This study also highlighted the impact that stigma and barriers to care have on willingness to receive help. Less than half of the soldiers in need of mental health services received care, and many reported concerns that they would be treated differently by peers or leaders if they sought care.

In a subsequent survey involving active and National Guard brigade combat teams (infantry), rates of 15 percent were documented at three months post-deployment and rose to 17-25 percent at twelve months post-deployment using the same definitions as in the 2004 article (Thomas et al., 2010).

In-theater assessments of personnel in ground combat units have been conducted on nearly an annual basis in Iraq and several times in Afghanistan since the start of the wars (Army Mental Health Advisory Team Assessments–MHATs). These studies have found rates of acute stress or PTSD (based on a PCL $\geq$50 points) of 10-20 percent, with a strong correlation to combat frequency and intensity. Rates in units exposed to minimal combat were similar to baseline rates in the population (5 percent), and there was a linear increase up to 25 percent in units involved in the highest-intensity combat. The Afghanistan theater showed lower rates earlier in the war (7 percent in 2005), but they increased to levels comparable with Iraq in 2007 and thereafter.

In addition to studies based on infantry samples, there have been a number of studies based on post-deployment health assessments, healthcare utilization records, and random samples of military or veteran populations, including those not engaged in direct combat (Hoge et al., 2006; Milliken et al., 2007; Tanielian et al., 2008; Smith et al.,2008; Fear et al., 2010; and others). General population samples that do not focus specifically on combat units have resulted in lower rates than reported in infantry samples, but estimates approach infantry samples when analyses are restricted to Army or Marine personnel with combat experience. While most studies have focused on point prevalence of PTSD, one study has looked at the 3-year incidence in a large representative population sample (Smith et al., 2008). The cumulative incidence was 9 percent in Army personnel who had experienced combat, which equates to a prevalence of approximately 12 percent, including those excluded for PTSD at baseline. Overall, baseline pre-deployment rates in military samples have ranged from 3-6 percent, comparable to civilian rates reported in the

National Co-morbidity Study (Kessler et al., 2005), and post-deployment rates have ranged from 6-20 percent. The strongest predictors of increased prevalence post-deployment have been combat frequency and intensity. There are also many other types of traumatic experience that service members encounter, both in their professional military occupations and in their pre-military or off-duty time, including exposure to accidents, assault, rape, natural disasters, and other experiences.

Outcome Measures:

The Working Group (WG) agreed on the following health-related outcomes for management of post-traumatic stress:

- Improvement in quality of life (social and occupational functioning)
- Reduced morbidity/mortality
- Improvement over long term
- Patient Satisfaction
- Co-morbidity
- Improvement of symptoms.

Guideline Goals:

The most important goal of the VA/DoD Clinical Practice Guideline for the Management of Post-Traumatic Stress is to provide scientific evidence-based practice evaluations and interventions. The guideline was developed to assist facilities in implementing processes of care that are evidence-based and designed to achieve maximum functionality and independence, as well as improve patient and family quality of life. The related specifics are:

- To identify the critical decision points in the management of patients with post-traumatic stress disorder
- To allow flexibility so that local policies or procedures, such as those regarding referrals to or consultation with specialty care (mental healthcare), can be accommodated
- To decrease the development of complications and co-morbidity
- To improve patient outcomes—i.e., reduce symptoms, decrease co-morbidity, increase functional status, and enhance the quality of life.

Development Process:

The development process of this guideline follows a systematic approach described in "Guideline-for-Guidelines," an internal working document of the VA/DoD Evidence-Based Practice Working Group that requires an ongoing review of the work in progress. Appendix A clearly describes the guideline development process followed for this guideline.

The Offices of Quality and Performance and Patient Care Service of the VA, in collaboration with the network Clinical Managers, and the Medical Center Command of the DoD identified clinical leaders to champion the guideline development process. During a preplanning conference call, the clinical leaders defined the scope of the guideline and identified a group of clinical experts from the VA and DoD that formed the Guideline Development Working Group.

At the start of the update process, the clinical leaders, guideline panel members, outside experts, and experts in the field of guideline and algorithm development were consulted to determine which aspects of the 2004 guideline required updating. These consultations resulted in the following recommendations that guided the update efforts: (1) update any recommendations from the original guideline likely to be affected by new research findings; (2) provide information and recommendations on health system changes relevant to the management of post-traumatic stress; (3) address content areas and models of treatment for which little data existed during the development of the original guideline; and (4) review the performance and lessons learned since the implementation of the original guideline

Review of Literature and Evidence:

Recommendations for the performance or inclusion of specific procedures or services in this guideline were derived through a rigorous methodological approach that included the following:

- Determining appropriate criteria, such as effectiveness, efficacy, population benefit, and patient satisfaction
- Performing a comprehensive literature search and selection of relevant studies from January 2002 to August 2009 to identify the best available evidence and ensure maximum coverage of studies at the top of the hierarchy of study types
- Reviewing the selected studies to determine the strength of the evidence in relation to these criteria
- Formulating the recommendations and grading the level of evidence supporting each recommendation.

This 2010 update builds on the 2004 version of the guideline and incorporates information from the following existing evidence-based guidelines/reports identified by the Working Group as appropriate seed documents:

- ISTSS (2009) - Effective Treatments for PTSD: Practice Guidelines from the International Society for Traumatic Stress Studies. Foa EB, Keane TM, Friedman MJ. Cohen J (Eds) 2009. New York: Guilford Press.
- IOM (2007) - Institute of Medicine (IOM). 2008. Treatment of post-traumatic stress disorder: An assessment of the evidence. Washington, DC: The National Academies Press.
- APA (2009) - Practice Guideline for the Treatment of Patients with Acute Stress Disorder and Posttraumatic Stress Disorder: Guideline Watch, March 2009.

Literature searches were conducted, covering the period from January 2002 through August 2009, that combined terms for post-traumatic stress, acute stress reaction (ASR), acute stress disorder (ASD), acute post-traumatic stress disorder, and chronic post-traumatic stress disorder. Electronic searches were supplemented by reference lists, and additional citations were suggested by experts. The identified and selected studies on those issues were critically analyzed, and evidence was graded using a standardized format, based on the system used by the U.S. Preventive Services Task Force (USPSTF, 2007).

If evidence exists, the discussion following the recommendations for each annotation includes an evidence table identifying the studies that have been considered, the quality of the evidence, and the rating of the strength of the recommendation [SR]. The Strength of Recommendation, based on the level of the evidence and graded using the USPSTF rating system (see Table: Evidence Rating System), is presented in brackets following each guideline recommendation.

Evidence Rating System

SR	
A	A strong recommendation that clinicians provide the intervention to eligible patients. Good evidence was found that the intervention improves important health outcomes and concludes that benefits substantially outweigh harm.
B	A recommendation that clinicians provide (the service) to eligible patients. At least fair evidence was found that the intervention improves health outcomes and concludes that benefits outweigh harm.
C	No recommendation for or against the routine provision of the intervention is made. At least fair evidence was found that the intervention can improve health outcomes but concludes that the balance of benefits and harms is too close to justify a general recommendation.
D	Recommendation is made against routinely providing the intervention to asymptomatic patients. At least fair evidence was found that the intervention is ineffective or that the harms outweigh benefits.
I	The conclusion is that the evidence is insufficient to recommend for or against routinely providing the intervention. Evidence that the intervention is effective is lacking, of poor quality, or conflicting, and the balance of benefits and harms can not be determined.

SR = Strength of recommendation

Where existing literature was ambiguous or conflicting, or where scientific data was lacking on an issue, recommendations are based on the clinical experience of the Working Group. Although several of the recommendations in this guideline are based on weak evidence, some of these recommendations are strongly recommended, based on the experience and consensus of the clinical experts and researchers of the Working Group. Recommendations that are based on a consensus of the Working Group include a discussion on the given topic. No [SR] is presented for these recommendations. A complete bibliography of the references in this guideline can be found in Appendix E

This Guideline is the product of many months of diligent effort and consensus-building among knowledgeable individuals from the VA and DoD and a guideline facilitator from the private sector. An experienced moderator facilitated the multidisciplinary Working Group. The draft document was discussed in two face-to-face group meetings. The content and validity of each section were thoroughly reviewed in a series of conference calls. The final document is the product of those discussions and has been approved by all members of the Working Group.

The list of participants is included in Appendix D to the guideline.

Implementation:

The guideline and algorithms are designed to be adapted by individual facilities in consideration of local needs and resources. The algorithms serve as a guide that providers can use to determine best interventions and timing of care for their patients in order to optimize quality of care and clinical outcomes.

Although this guideline represents the state-of-the-art practice on the date of its publication, medical practice is evolving, and this evolution requires continuous updating of published information. New technology and more research will improve patient care in the future. The clinical practice guideline can assist in identifying priority areas for research and optimal allocation of resources. Future studies examining the results of clinical practice guidelines such as these may lead to the development of new practice-based evidence.

KEY POINTS ADDRESSED BY THIS GUIDELINE

1. Triage and management of acute traumatic stress
2. Routine primary care screening for trauma and related symptoms
3. Diagnosis of trauma syndromes and co-morbidities
4. Evidence-based management of trauma-related symptoms and functioning
5. Collaborative patient/provider decision-making, education, and goal-setting
6. Coordinated and sustained follow-up
7. Identification of major gaps in current knowledge
8. Outline for psychological care in ongoing military operations
9. Proactive strategies to promote resilience and prevent trauma-related stress disorders
10. Standardized longitudinal care (DoD/VA, Primary Care/Mental Health)

OVERVIEW OF GUIDELINE UPDATE

This clinical practice guideline updates the 2004 version of the VA/DoD Guideline on Management of Post-Traumatic Stress. The Working Group (WG) developed a revised, comprehensive clinical algorithm. The objective of the VA/DoD Working Group in developing this revision was to incorporate the accumulating experience in the field and information from the original guideline recommendations into a format that would maximally facilitate clinical decision-making. Randomized controlled trials and systematic reviews were identified and have been carefully appraised and included in the analysis of the evidence for this update. Promoting evidence-based treatment ultimately enhances and optimizes treatment outcomes, thus contributing to optimal care across institutional boundaries and promoting a smooth transition of care between the DOD and the VA healthcare systems.
The current revision incorporates the four Modules of the 2004 guideline into a CORE module and two management Modules: 1) Acute Stress Reaction and early interventions to prevent PTSD; and 2) Management of PTSD. Where evidence suggests differences in the management of Acute Stress Reactions (ASR), Acute Stress Disorder (ASD), and Post-Traumatic Stress Disorder (PTSD), specific treatment intervention recommendations are provided.

The VA/DoD Working Group reviewed the International Society for Traumatic Stress Studies clinical practice guideline (Foa et al., 2009) and made the decision to adopt several of their evidence-based recommendations. In addition, identified randomized controlled trials and systematic reviews published in the past 7 years have been carefully appraised and included in the analysis of the evidence for this update.

The first Module incorporates the assessment, diagnosis, and management of symptoms of **Acute Stress Reaction (ASR)** in the immediate period after exposure to trauma, the management of **Acute Stress Disorder (ASD**), and the effective early interventions to prevent progression of stress reactions to full PTSD. Additional recommendations were added for the assessment and management of **Combat and Operational Stress Reaction (COSR),** addressing specific actions that the WG considered to be of importance for providers caring for service members with symptoms.

The second Module addresses the diagnosis and management of patients with **Post-Traumatic Stress Disorder (PTSD**). The WG revised the algorithm for this module in a patient-centered approach that emphasizes the decisions and interventions shown to be effective in treating PTSD, regardless of the treatment setting. This approach should allow for the use of the guideline as a starting point for innovative plans that improve collaborative efforts and focus on key aspects of care. The recommendations outlined in this guideline should serve as a framework for the care that is provided in both, specialty mental healthcare settings and primary care. The optimal setting of care for the individual patient will depend on patient preferences, the level of expertise of the provider, and available resources.

The WG recognizes that PTSD is often accompanied by other psychiatric conditions. Such co-morbidities require clinical attention at the point of diagnosis and throughout the process of treatment. Disorders of particular concern are substance use disorder, major depression, and post-concussive symptoms attributed to mild TBI. The WG also recognizes the fact that few trials have been published that can provide guidance on how to manage PTSD that is complicated by co-morbid illness. The revised guideline includes recommendations based on the experience and opinion of the experts, providing suggestions for the approach to treatment of PTSD in the presence of co-morbid psychiatric conditions.

Working Group consensus-based recommendations are added to the 2010 revision of the CPG regarding specific adjunct treatment interventions that target specific symptoms frequently seen in patients with acute stress reactions (beyond the core symptoms of ASD/PTSD). These include sleep disturbance, pain, and anger. These consensus-based recommendations are aimed to help the primary care practitioners and others to provide brief symptom-focused treatment.

Finally, clinicians following these updated guidelines should not limit themselves only to the approaches and techniques addressed in the guideline. All current treatments have limitations—not all patients respond to them, patients drop out of treatment, or providers are not comfortable using a particular intervention. Creative integration of combined treatments that are driven by sound evidence-based principles is encouraged in the field.

VA/DoD GUIDELINE WORKING GROUP

<table>
<tr><th>VHA</th><th>DoD</th></tr>
<tr><td>Ron Acierno, PhD
Kathleen Chard, PhD
Daniella David, MD
Matt Friedman, MD (Co-Chair)
Matt Jeffreys, MD
Terry Keane, PhD
Harold Kudler, MD
Todd Semla, PharmD
Sheila Rauch, PhD
Josef Ruzek, PhD (Co-Chair)
Steve Southwick, MD
Murray Stein, MD

Reviewers and Contributors:

Nancy Bernardy, PhD
LTC Edward Brusher, LCSW
Bruce Capehart, MD, MBA
Michael Clark, PhD
Kent Drescher, PhD
Carolyn Green, PhD
Barbara Hermann, PhD
Julia Hoffman, Psy.D
Dan Kivlahan, PhD
Eric Kuhn, PhD
Walter Penk, PhD
Paula Schnurr, PhD
James Spira, PhD, MPH
Jennifer Vasterling, PhD</td><td>Curtis Aberle, NP
LT Justin Campbell, PhD
MAJ Debra Dandridge, PharmD
COL Charles Engel, MD
Capt Joel Foster, PhD
CDR Stella Hayes, MD
Charles Hoge, MD
MAJ Kenneth Hyde, PA
CAPT Robert Koffman, MD
COL James Liffrig, MD
COL Patrick Lowry, MD (Co-Chair)
LTC Sandra McNaughton, NP
David Orman, MD
Alan Peterson, PhD
Miguel Roberts, PhD
CAPT Mark Stephens, MD
CAPT Frances Stewart, MD
MAJ Christopher Warner, MD
Lt Col Randon Welton, MD
LTC Robert Wilson, PhD</td></tr>
<tr><td>Office of Quality and Performance
Carla Cassidy, RN, MSN, NP
Kathryn J. Dolter, RN, PhD</td><td>US Army Medical Command
Ernest Degenhardt, MSN, RN, ANP-FNP
Marjory Waterman, MN, RN</td></tr>
<tr><td colspan="2">Guideline Facilitator: Oded Susskind, MPH</td></tr>
<tr><td>Research and Evidence Appraisal
Hayes Inc. Lansdale, PA
Susan A. Levine, DVM, PhD
Arlene Mann, R.N.</td><td>Healthcare Quality Informatics:
Martha D’Erasmo, MPH
Rosalie Fishman, RN, MSN, CPHQ
Sue Radcliff</td></tr>
</table>

TABLE OF CONTENTS

CORE MODULE: ALGORITHM 14

CORE MODULE: ANNOTATIONS 15

1. PRIMARY PREVENTION 15
A. Education and Training to Foster Resilience 15

2. POPULATIONS AT-RISK FOR DEVELOPING PTSD 17
B. Person Exposed to Trauma 17

3. SECONDARY PREVENTION 18
C. Screen for PTSD Symptoms 18
D. Are Trauma-Related Symptoms Present? 20
E. Educate About Additional Care If Needed; Provide Contact Information 25

MODULE A: ACUTE STRESS REACTION (ASR) and PREVENTION OF POST-TRAUMATIC STRESS DISORDER (PTSD) 28

MODULE A: ALGORITHM 29

MODULE A: ANNOTATIONS 30

1. ASSESSMENT & TRIAGE 30
A. Trauma Exposure (within the past 30 days) 30
B. Assess Briefly Based on General Appearance and Behavior 31
C. Unstable, Dangerous to Self or Others, or Need for Urgent Medical Attention 32
D. Ensure Basic Physical Needs Are Met 34
E. Person has Trauma-Related Symptoms, Significant Impaired Function, or Diagnosis of ASD 39
F. Assess Medical and Functional Status 42
G. Assess Pre-Existing Psychiatric and Medical Conditions 43
H Assess Risk Factors for Developing ASD/PTSD 43

2. TREATMENT 45
I. Provide Education and Normalization / Expectancy of Recovery 45
J. Initiate Brief Intervention 45
K. Acute Symptom Management 49
L1. Facilitate Spiritual Support 50
L2. Facilitate Social Support 50

3. RE-ASSESSMENT 51
M. Reassess Symptoms and Function 51

4. FOLLOW-UP 52
N. Persistent (>1 Month) or Worsening Symptoms, Significant Functional Impairment, or High Risk for Development of PTSD. 52
O. Monitor and Follow-Up 54

MODULE B: ALGORITHM 56

MODULE B: ANNOTATIONS 58

1. ASSESSMENT 58
A. Assessment of Stress Related Symptoms 58
B. Assessment of Trauma Exposure 60
C. Assessment of Dangerousness to Self or Others 61
D. Obtain Medical History, Physical Examination, Laboratory Tests and Psychosocial Assessment 65
E. Assessment of Function, Duty/Work Responsibilities and Patient's Fitness (In Relation To Military Operations) 68
F. Assessment of Risk/Protective Factors 71

2. TRIAGE 78
G. Diagnosis of PTSD or Clinical Significant Symptoms Suggestive of PTSD? 78
H. Assess for Co-Occurring Disorders 81
I. Educate Patient and Family 84
J. Determine Optimal Setting for Management of PTSD and Co-Occurring Disorders 86
J1. Management of PTSD with Co-morbidity 86
J2. Management of Concurrent PTSD and Substance Use Disorder 88
J3. The Role of the Primary Care Practitioner 91

3. TREATMENT 91
K. Initiate Treatment Using Effective Interventions for PTSD 91
L. Facilitate Spiritual Support 93
M. Facilitate Social Support 93

4. RE-ASSESSMENT 94
N. Assess Response to Treatment 94
O. Follow-Up 95

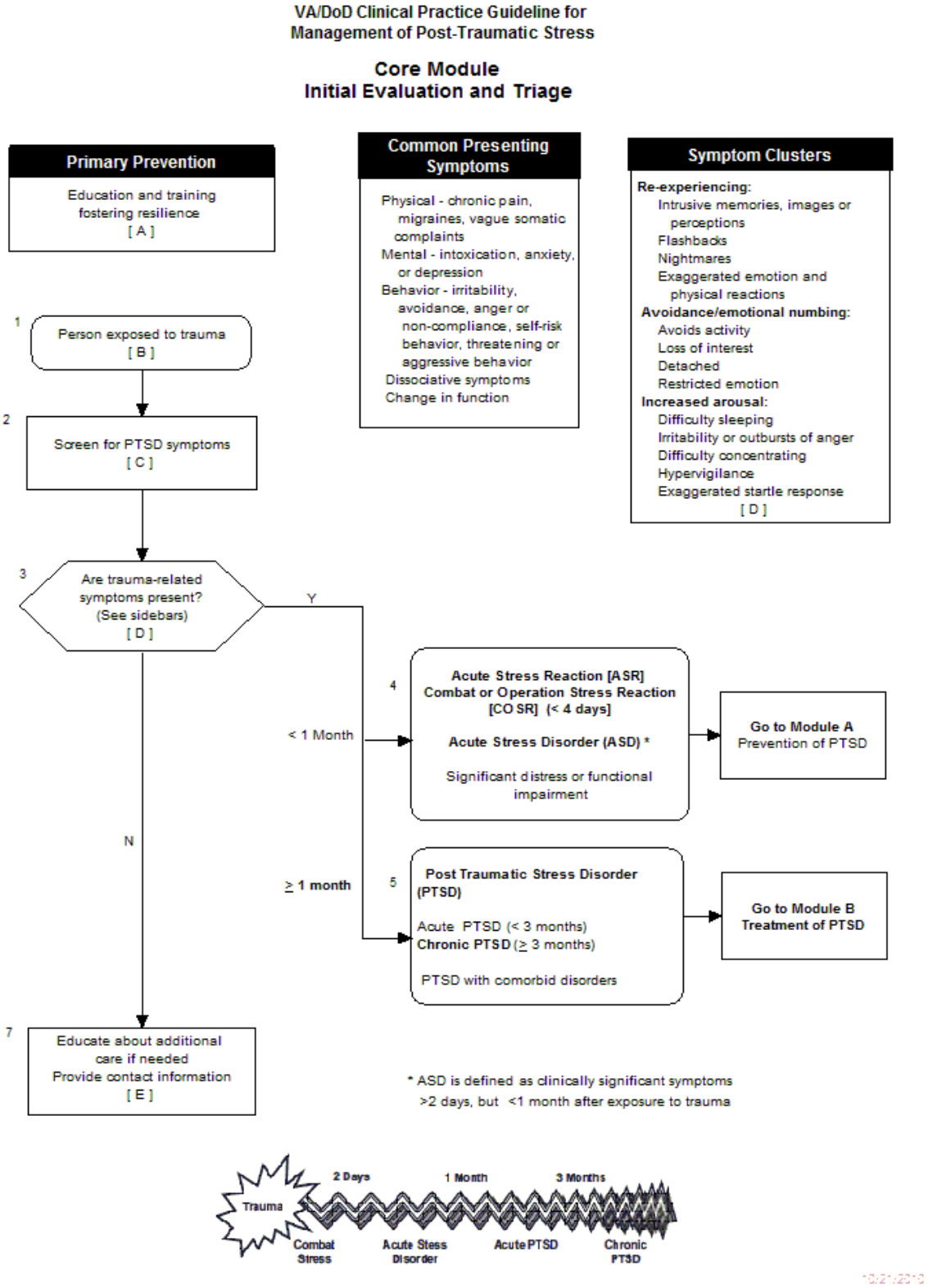
MODULE I: TREATMENT INTERVENTIONS 101

Module I-1. EARLY INTERVENTIONS TO PREVENT PTSD 102
A. PSYCHOTHERAPY 104
A1. Psychological Debriefing 104
A2. Brief Early Cognitive-Behavioral Intervention 108
A3. Other Early Interventions 109
B. Early Pharmacotherapy Interventions to Prevent PTSD 110

Module I-2. TREATMENT FOR PTSD 114
A. Selection of Therapy for PTSD 114
B. PSYCHOTHERAPY INTERVENTIONS FOR PTSD 115
B1. Therapies that More Strongly Emphasize Cognitive Techniques (CT) 119
B2. Exposure Therapy (ET) 123
B3. Stress Inoculation Training (SIT) 126
B5. Imagery Rehearsal Therapy (IRT) 130
B6. Psychodynamic Therapy 132
B7. Patient Education 133
B9. Dialectical Behavior Therapy 140
B10. Hypnosis 142
B11. Behavioral Couples Therapy 143
B12. Telemedicine and Web-based Interventions 144
C. PHARMACOTHERAPY FOR PTSD 149
D. ADJUNCTIVE SERVICES 167
D1. Psychosocial Rehabilitation 167
D2. Spiritual Support 172
E. SOMATIC TREATMENT 173
E1. Biomedical Somatic Therapies 173
E2. Acupuncture 175
F. COMPLEMENTARY AND ALTERNATIVE MEDICINE 176
F1. Natural Products (Biologically Based Practices) 178
F2. Mind-Body Medicine 179
F3. Manipulation and Body-Based Practices (Exercise and Movement) 180
F4. Energy medicine 180
F5. Whole Medical Systems 181
F6. Other Approaches 182

Module I-3. MANAGEMENT OF SPECIFIC SYMPTOMS 183
A. Sleep Disturbances 183
B. Pain 189
C. Irritability, Severe Agitation, or Anger 194

CORE MODULE: ALGORITHM



CORE MODULE: ANNOTATIONS

1. PRIMARY PREVENTION

A. Education and Training to Foster Resilience

OBJECTIVE

Prepare individuals and groups for exposure to potentially traumatic experiences in ways that minimize the likelihood of development of Post-Traumatic Stress Disorder (PTSD) and other trauma-related problems.

BACKGROUND

Because exposure to traumatic stressors is part of the expected work experience of some occupations (e.g., military personnel and emergency services workers), it is sensible to make efforts to prepare individuals in these professions for their encounters with traumatic events. This preparation is not explicitly undertaken in most workplaces, with some exceptions (e.g., some military training environments). To date, research has not examined our capacity to prepare individuals or communities for trauma exposure. However, general principles of preparation can be outlined that are consistent with theoretical models of the development of PTSD, research on risk factors for development of PTSD, and emerging concepts of resilience and hardiness.

RECOMMENDATIONS

1. In high-risk occupations, for which the probability of trauma exposure is moderate or high, efforts should be undertaken to increase the psychological resilience of workers to the negative effects of trauma exposure.

DISCUSSION

Although little is directly known about our capacity to prepare individuals or communities for trauma exposure, it is possible to identify principles of preparation that are consistent with empirical research on risk and resilience factors and with current theories of PTSD development. Such pre-trauma preparation can include attention to both the ability to cope during the trauma itself and shaping the post-trauma environment so that it will foster post-trauma adaptation.

Some influential theories of PTSD posit that a process of classical fear conditioning can lead to development of chronic PTSD symptomatology. In this process, stimuli associated with the traumatic experience can elicit responses similar to those experienced during the trauma itself (e.g., intense anxiety). Other theories suggest that individuals who develop negative trauma-related beliefs (e.g., about personal guilt) will be more likely to experience continuing trauma reactions, because such beliefs will maintain a sense of threat and personal incompetence. Research on risk factors for PTSD indicates that post-trauma social support and life stress affect the likelihood of development of the disorder. Protective factors have also been identified that mitigate the negative effects of stress. Research is beginning to delineate the psychological processes that moderate an individual's response to stress and to explore training programs for increasing resilience to stress. Hardiness (Kobasa et al., 1982) is one personality factor that has been demonstrated to buffer against traumatic stress and PTSD in military veterans (King et al., 1998; Bartone, 2000). Zach, Raviv & Inbar (2007) found that hardiness levels increased for Special Forces

trainees over the course of a stressful training/selection program in which challenges were gradually more difficult, and leaders were consistently supportive and encouraged trainees to view failures as learning opportunities. Hardiness is characterized by three key attributes: ability to perceive *control* over life's events; ability to make strong *commitment* to tasks; and ability to see stressful experiences as a *challenge* to be overcome. Training programs, personnel policies, and leadership strategies that promote hardiness may thereby increase an individual's ability to resist the negative effects of traumatic stress.

Such findings and theories are consistent with the following principles of preparation:

1. *Provide realistic training* that includes vicarious, simulated, or actual exposure to traumatic stimuli that may be encountered. Examples of application of this principle in military training include exposure to live weapons fire, survival training, or, for subgroups of military personnel, mock captivity training. This principle can be applied to many work roles—for example, those likely to be involved in body handling might be trained in mortuary environments. It is consistent with classical conditioning theories, in that this can help reduce arousal or anxiety associated with particular traumatic stimuli.
2. *Strengthen perceived ability to cope* during the trauma and with the aftermath. Realistic training contributes to this goal. Instruction and practice in the use of a variety of coping skills (e.g., stress inoculation training, problem-solving, assertion, and cognitive restructuring) may be helpful in enabling workers to tolerate stressful work environments. In addition, individuals can be trained to cope with acute stress reactions that are common following trauma exposure. Such training experiences help to maximize expectations of mastery of traumatic situations and their physical and emotional sequelae. Use of positive role models (leaders and peers) is also an effective tool for building up the sense of ability to cope. The training must include specific, practical actions to change the threatening or horrifying situation for the better. Without such positive action learning, "simulated" terrifying or horrifying situations and stimuli can induce feelings of helplessness that make the training itself traumatizing.
3. *Create supportive interpersonal work environments* that are likely to provide significant social support during and after traumatic events. Efforts to build teams and establish group cohesion among work group members are important in this regard. Identification and training of peer stress management consultants and training and practice in the provision of peer social support may also be useful. Families are crucial in post-trauma support and can be given information about, and training in, ways of providing social support. Finally, competent, ethical leadership at all levels of the organization helps protect against traumatization.
4. *Develop and maintain adaptive beliefs* about the work role and traumatic experiences that may be encountered within it. Key beliefs will be related to realistic expectancies about the work environment, confidence in leadership, confidence in the meaningfulness or value of the work role, positive but realistic appraisals of one's coping ability, and knowledge about the commonness and transitory nature of most acute stress reactions. It may be useful to identify and discuss negative beliefs that sometimes arise in the specific work environment in order to "inoculate" against such beliefs.
5. *Develop workplace-specific comprehensive traumatic stress management programs.* Such programs can be a significant source of post-trauma support (e.g., via Chaplains or mental health professionals) that can minimize trauma-related problems among workers. It is important to take steps to increase

awareness of such services and to de-stigmatize and reduce the potential negative consequences of their use. For example, employees should be helped to understand that seeking help in confronting symptoms and problems early in their development is likely to be more effective than avoiding them or keeping them secret from others but that even long-hidden or persisting PTSD can be treated.

Comprehensive preparation programs that target and incorporate these principles and that are integrated themselves into existing unit/community programs and support systems may be expected to be most helpful (Gist & Lubin, 1999).

2. POPULATIONS AT-RISK FOR DEVELOPING PTSD

B. Person Exposed to Trauma

OBJECTIVE

Assess the nature of the traumatic event and other potential stressors.

BACKGROUND

A number of sufferers with PTSD may recover with no or limited interventions. However, without effective treatment, many people may develop chronic problems over many years. The severity of the initial traumatic response is a reasonable indicator of the need for early intervention. Families and care-givers have a central role in supporting people with stress symptoms. Depending on the nature of the trauma and its consequences, many families may also need support for themselves.

RECOMMENDATIONS

1. Persons exposed to trauma should be assessed for the type, frequency, nature, and severity of the trauma. [B]
    a. Assessment should include a broad range of potential trauma exposures in addition to the index trauma.
    b. Trauma Exposure Assessment Instruments may assist in evaluating the nature and severity of the exposure.
    c. Assessment of existing social supports and ongoing stressors is important.

DISCUSSION

Although exposure to trauma is common, several risk factors for the development of PTSD have been identified. *Trauma*-related risks include the nature, severity, and duration of the trauma exposure. For example, life-threatening traumas, such as physical injury or rape, pose a high risk of PTSD (Kilpatrick, 1989). A prior history of trauma exposure conveys a greater risk of PTSD from subsequent trauma (Breslau et al., 1999).

Post-trauma risks include poor social support and life stress (Brewin et al., 2000). A greater risk for developing PTSD may be conveyed by post-trauma factors (e.g., lack of social support and additional life stress) than pre-trauma factors.

3. SECONDARY PREVENTION

C. Screen for PTSD Symptoms

OBJECTIVE

Identify possible cases of post-traumatic stress

BACKGROUND

Patients do not often self-identify as suffering with PTSD, and patients with unrecognized PTSD are often difficult to treat because of poor patient/provider rapport, anger and distrust, a focus on somatic symptoms, and other trauma-related problems. Research supports the utility of brief screening tools for identifying undiagnosed cases of PTSD. Identification of PTSD may help facilitate development of rapport, suggest treatment options, and potentially improve outcomes for these patients.

RECOMMENDATIONS

1. All new patients should be screened for symptoms of PTSD initially and then on an annual basis or more frequently if clinically indicated due to clinical suspicion, recent trauma exposure (e.g., major disaster), or history of PTSD. [B]
2. Patients should be screened for symptoms of PTSD using paper-and-pencil or computer-based screening tools. [B]
3. There is insufficient evidence to recommend one PTSD screening tool versus another. However, the following screening tools have been validated and should be considered for use. For example: (See Appendix C)
    - Primary Care PTSD Screen (PC-PTSD)
    - PTSD Brief Screen
    - Short Screening Scale for DSM IV PTSD.
    - PTSD Checklist (PCL)
4. There is insufficient evidence to recommend special screening for members of any cultural or racial group or gender. [I]

DISCUSSION

The benefit of screening is well established for diseases with high prevalence. In one study (Taubman et al., 2001), 23 percent of patients presenting in the primary care setting reported exposure to traumatic events, and 39 percent of those met criteria for PTSD. Screening strategies should, however, balance efficacy with practical concerns (e.g., staffing, time constraints, and current clinical practices). Brevity, simplicity, and ease of implementation should encourage compliance with recommended screening. Care should be exercised in implementing screening in ways that avoid social stigmatization and adverse occupational effects of positive screens.

Brewin (2005) reviewed published screening instruments for civilian PTSD, consisting of 30 items or fewer, that were validated against structured clinical interviews. Thirteen instruments were identified as meeting these criteria, all consisting of symptoms of traumatic stress. The review concluded that the performance of some currently available instruments is near their maximal potential effectiveness and that instruments with fewer items, simpler response scales, and simpler scoring methods perform as well as, if not better, than longer and more complex measures.

Screening Tools: *(See Appendix C)*

Primary Care PTSD Screen (PC-PTSD): This is a 4-item screen that was designed for use in primary care and other medical settings and is currently used to screen for PTSD in veterans at the VA. The screen includes an introductory sentence to cue respondents to traumatic events. The authors suggest that in most circumstances, the results of the PC-PTSD should be considered "positive" if a patient answers "yes" to any 3 items. Those who screen positive should then be assessed with a structured interview for PTSD. The screen does not include a list of potentially traumatic events (Prins et al., 2003). Internal consistency (KR20=.79) and test-retest reliability (r=.84) of the PC-PTSD were found to be good (Prins et al., 1999). The operating characteristics of the screen suggest that the overall efficiency (i.e., optimal sensitivity and specificity =.87) is best when any two items are endorsed. The PC-PTSD screen has been validated in a military population (Bliese et al., 2008) and has been used extensively in post-deployment screening efforts (Hoge, 2004).

PTSD Brief Screen: The PTSD Brief Screen was developed using the rationally derived approach, based on data from the National Co-morbidity Survey. Construct validity has generally been adequate. The overall efficiency of this screen was good (.78), whereas the correlations were significantly lower or negative for other mental disorders, indicating good construct validity (Leskin et al., 1999).

PTSD Checklist (PCL): The PCL has been used extensively in military and civilian populations, and there are numerous validation studies, including studies in military populations (Terhakopian et al., 2008).

Special Screening of Cultural or Racial Groups:

Research has centered on three broadly defined groups—Hispanics, Blacks/African-Americans, and Whites/Caucasians—in the attempt to answer two questions: First, are members of one or more groups more susceptible to developing PTSD? Second, are the symptoms shown by members of any group more severe or otherwise different from symptoms shown by other veterans with PTSD?

There are data to suggest that Blacks/African-Americans and Hispanics experience higher rates of PTSD than do Whites/Caucasians (Frueh et al., 1998; Ortega & Rosenheck, 2000). But, as Frueh and his colleagues note in a systematic review, “secondary analyses within the existing epidemiological studies suggest that differential rates of PTSD between racial groups may be a function of differential rates of traumatic stressors and other pre-existing conditions. This finding, in combination with the general paucity of empirical data and certain methodological limitations, significantly moderates the conclusions that should be reached from this body of literature.” Studies in military samples have generally shown no or minimal race/ethnic differences in PTSD prevalence.

In terms of symptom severity and clinical course, the evidence is also mixed. Among the studies reviewed here, the following conclusions were reached:

- Two studies found Black/African-American veterans to be more severely affected than Hispanics or Whites/Caucasians (Frueh et al., 1996; Penk et al., 1989)
- The National Vietnam Veterans Readjustment Study (NVVRS) found higher PTSD prevalence among Hispanic veterans than among Whites or Blacks after controlling for combat exposure (Kulka et al., 1990; Schlenger et al., 1992).
- One study found Hispanics to be more severely affected than Whites/Caucasians but not to suffer from higher functional impairment levels than Whites/Caucasians (Ortega and Rosenheck, 2000).

- Three studies found no significant clinical differences between Black/African-American veterans and White/Caucasian veterans (Frueh et al., 1997; Rosenheck and Fontana, 1996; Trent et al., 2000).
- One review found no clinical differences among Hispanics, Blacks/African-Americans, and Whites/Caucasians (Frueh et al., 1998)
- One study found that American-of-Japanese Ancestry Vietnam Veterans had lower PTSD prevalence than Caucasians (Friedman et al., 2004).
- Among Vietnam Veterans, American Indians and Native Americans have higher rates than Caucasian veterans whereas American of Japanese ancestry have lower PTSD prevalence than Caucasians (Beals et al., 2002; Friedman et al., 2004).

These results support Frueh et al. (1998) in their conclusion that "despite the prevailing zeitgeist and clinical lore, the limited extant empirical evidence suggests that veterans of different races are more similar to each other than they are different when it comes to the clinical manifestation and response to treatment of combat-related PTSD and associated features."

EVIDENCE

	Evidence	Sources	LE	QE	SR
1	Screening all patients for PTSD symptoms.	Breslau et al., 1999a Leskin & Westrup, 1999 Prins et al., 1999 Taubman et al., 2001	II-2	Fair	B
2	Screening tools: Primary Care PTSD Screen PTSD Brief Screen Short Screening Scale for DSM IV PTSD Checklist (PCL)	Breslau et al., 1999a Leskin & Westrup, 1999 Prins et al., 1999 Terhakopian, et al 2008	II-2	Fair	B
3	Special screening for members of any cultural or racial group	Frueh et al., 1996, 1997, 1998 Ortega & Rosenheck, 2000 Penk et al., 1989 Rosenheck & Fontana, 1996 Trent et al., 2000 Friedman 2004	III	Poor	I

LE – Level of Evidence; QE = Quality of Evidence; SR = Strength of Recommendation (see Appendix A)

D. Are Trauma-Related Symptoms Present?

OBJECTIVE

Identify people exposed to trauma who are at risk for developing acute stress reactions (ASR), acute stress disorder (ASD), or Post-Traumatic Stress Disorder (PTSD).

BACKGROUND

Warning Signs of Trauma-Related Stress (APA)

Individuals who have experienced a traumatic event often experience psychological stress reactions related to the incident. In most instances, these are common normal reactions to extreme situations. Individuals who feel they are unable to regain control of their lives or who experience the following symptoms for more than a

month should consider seeking outside professional mental health assistance. Some symptoms to watch out for include:

- Recurring thoughts, mental images, or nightmares about the event
- Having trouble sleeping
- Changes in appetite
- Experiencing anxiety and fear, especially when exposed to events or situations reminiscent of the trauma
- Feeling on edge, being easily startled, or becoming overly alert
- Feeling depressed or sad and having low energy
- Experiencing memory problems, including difficulty in remembering aspects of the trauma
- Feeling "scattered" and unable to focus on work or daily activities
- Having difficulty making decisions
- Feeling irritable, easily agitated, or angry and resentful
- Feeling emotionally "numb," withdrawn, disconnected, or different from others
- Spontaneously crying, feeling a sense of despair and hopelessness
- Feeling extremely protective of, or fearful for, the safety of loved ones
- Not being able to face certain aspects of the trauma and avoiding activities, places, or even people that remind you of the event.

RECOMMENDATIONS

1. Individuals who are presumed to have symptoms of PTSD or who are positive for PTSD on the initial screening should receive a more detailed assessment of their symptoms.
2. Useful symptom-related information may include details, such as time of onset, frequency, course, severity, level of distress, and degree of functional impairment.
3. The elapsed time since the exposure to trauma should be considered when assessing the risk of developing PTSD and determining the diagnosis and appropriate intervention.

The following definitions will help providers select the appropriate treatment algorithm:

Stress-Related Disorders and Syndromes Definitions

Trauma

An extreme traumatic stressor involving direct personal experience of an event that involves actual or threatened death or serious injury or another threat to one's physical integrity; witnessing an event that involves death, injury, or a threat to the physical integrity of another person; or learning about unexpected or violent death, serious harm, or threat of death or injury experienced by a family member or other close associate. According to DSM-IV-TM criteria, the person's response to the event must involve intense fear, helplessness, or horror. However, there is evidence that military personnel do not always respond in the same way as civilian victims of traumatic events, and the criteria for “fear, helpless, or horror” are being reconsidered in the proposed future DSM criteria (Adler, 2008).

Acute Stress Reaction (ASR)

Acute stress reaction is a transient condition that develops in response to a traumatic event. Onset of at least some signs and symptoms may be simultaneous with the trauma itself or within minutes of the traumatic event and may follow the trauma after an interval of hours or days. In most cases, symptoms will disappear within days (even hours). Symptoms include a varying mixture of the following:

A broad group of physical, mental, and emotional signs and symptoms that result from heavy mental and emotional work during exposure to difficult potentially traumatic conditions.

Symptoms may include depression, fatigue, anxiety, decreased concentration/memory, hyperarousal, or any of the four categories of reactions (See Table CORE - 1) that have not resolved within four days after the event, after a rule-out of other disorders.

The traumatic events that can lead to an acute stress reaction are of similar severity to those involved in post-traumatic stress disorder.

Combat and Operational Stress Reaction (COSR) during an Ongoing Military Operation

COSR is the term used to describe an acute stress reaction in the combat environment and can include virtually any symptom and sign, including physical and neurological symptoms, resulting from exposure to extremely stressful events or combat experiences. It may result from specific traumatic experiences in combat or exhaustion due to the cumulative effects of one or more factors, including sleep deprivation, extreme physical stress, poor sanitary conditions, limited caloric intake, dehydration, or extremes of environmental conditions.

Acute Stress Disorder (ASD)

ASD refers to clinically significant (causing significant distress or impairment in social, occupational, or other important areas of functioning) symptoms >2 days but <1 month after exposure to a trauma, as defined above (may progress to PTSD if symptoms last >1 month). Criteria for diagnosis include:

- Exposure to trauma, as defined above
- Either while experiencing or after experiencing the distressing event, the individual has at least three of the following dissociative symptoms:
    - A subjective sense of numbing, detachment, and/or absence of emotional responsiveness
    - A reduction in awareness of his/her surroundings (e.g., "being in a daze").
    - Derealization (the feeling that familiar surroundings or people are unreal or have become strange)
    - Depersonalization (the feeling in an individual that (s)he is no longer him/herself. His/Her personality, body, external events, and the whole world may no longer appear to be real)
    - Dissociative amnesia (i.e., the inability to recall an important aspect of the trauma).
- The traumatic event is persistently re-experienced 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.

- Marked avoidance of stimuli that arouse recollections of the trauma (e.g., thoughts, feelings, conversations, activities, places, people, sounds, smells, or others).
- Marked symptoms of anxiety or increased arousal (e.g., difficulty sleeping, irritability, poor concentration, hypervigilance, exaggerated startle response, and motor restlessness).

Post-Traumatic Stress Disorder (PTSD)

Clinically significant symptoms that are causing significant distress or impairment in social, occupational, or other important areas of functioning and occur more than one month after exposure to a trauma. Symptoms may include:

The traumatic event is **persistently re-experienced** in one (or more) of the following ways:

- Recurrent and intrusive recollections of the event, including images, thoughts, or perceptions
- Recurrent distressing dreams of the event
- 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)
- Intense psychological distress on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event
- Physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

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:

- Efforts to avoid thoughts, feeling, or conversations associated with the trauma
- Efforts to avoid activities, places, or people that arouse recollections of the trauma
- Inability to recall an important aspect of the trauma
- Markedly diminished interest or participation in significant activities
- Feeling of detachment or estrangement from others
- Restricted range of affect (e.g., unable to have loving feelings)
- Sense of foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span).

Persistent symptoms of increased **arousal** (not present before the trauma), as indicated by at least two of the following:

- Difficulty falling or staying asleep
- Irritability or outbursts of anger
- Difficulty concentrating
- Hypervigilance
- Exaggerated startle response.

Acute PTSD

The clinically significant symptoms above continue to cause significant distress or impairment in social, occupational, or other important areas of functioning, lasting more than one month but less than 3 months after exposure to trauma.

Chronic PTSD

The clinically significant symptoms above cause significant distress or impairment in social, occupational, or other important areas of functioning. The symptoms last more than 3 months after exposure to trauma. Chronic PTSD is unlikely to improve without effective treatment.

- Some PTSD patients may exhibit persistent difficulties in interpersonal relations, mood, somatization, and profound identity problems. Such presentation may be often associated with sustained or repeated trauma during childhood or adolescence (such as longstanding incest or physical abuse), but it may also be associated with sustained trauma in later life or may appear as a late consequence of chronic PTSD, even if the original traumatic stressor was a single event.
- Co-morbid – also meeting DSM criteria for another disorder, such as substance use disorder, major depression disorder, other anxiety disorder, and mTBI among military personal.

PTSD with Delayed Onset

Onset of the clinically significant symptoms above, causing significant distress or impairment in social, occupational, or other important areas of functioning at least 6 months after exposure to trauma.

Figure 1. Stress Reaction Timeline.

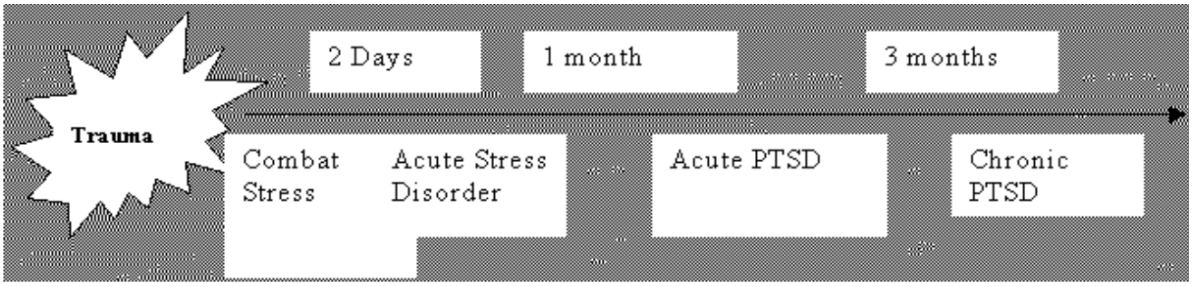


Table CORE - 1 Common Signs & Symptoms Following Exposure to Trauma

Physical	Cognitive / Mental	Emotional	Behavioral
• Chills • Difficulty breathing • Dizziness • Elevated blood pressure • Fainting • Fatigue • Grinding teeth • Headaches • Muscle tremors • Nausea • Pain • Profuse sweating • Rapid heart rate • Twitches • Weakness	• Blaming someone • Change in alertness • Confusion • Hyper-vigilance • Increased or decreased awareness of surroundings • Intrusive images • Memory problems • Nightmares • Poor abstract thinking • Poor attention • Poor concentration • Poor decision-making • Poor problem solving	• Agitation • Anxiety • Apprehension • Denial • Depression • Emotional shock • Fear • Feeling overwhelmed • Grief • Guilt • Inappropriate emotional response • Irritability • Loss of emotional control	• Increased alcohol consumption • Antisocial acts • Change in activity • Change in communication • Change in sexual functioning • Change in speech pattern • Emotional outbursts • Inability to rest • Change in appetite • Pacing • Startle reflex intensified • Suspiciousness • Social withdrawal

E. Educate About Additional Care If Needed; Provide Contact Information

OBJECTIVE

Provide normalization for survivors and responders whose reactions are not clinically significant

BACKGROUND

Trauma survivors and responders who are NOT experiencing signs or symptoms or who are experiencing few symptoms should receive education. It should emphasize that the observed reactions in the symptomatic survivors are common in the aftermath of trauma and do not signify personal inadequacy, health problems, mental illness, or other enduring negative consequences.

Contemporary approaches to early intervention following trauma exposure emphasize the importance of “normalization” of acute stress reactions. Survivors or responders who show distressing symptoms or disturbed behavior should be educated to understand that their reactions are common, normal responses to the extreme events. Such an approach follows from the common clinical observation that individuals experiencing acute stress reactions often interpret their reactions as signs of “personal weakness” or evidence that they are “going crazy,” which increases their demoralization and distress. Normalization is undermined if survivors or responders who are not experiencing disruptive distress show a derogatory or punitive attitude to others who are.

Also, the persons with distress who most strongly deny or dissociate from their distress may be at increased risk for developing acute stress disorder (ASD) and subsequent PTSD. The education and normalization may therefore help them recognize how to protect themselves better and to seek care early if symptoms do interfere with their “self-control.” Even those who go on to develop PTSD may benefit from an understanding that their symptoms do not represent “personal weakness”

and that although their symptoms may be severe and painful, they are not losing control of their minds.

RECOMMENDATIONS

1. Pre- and post-trauma education should include helping the asymptomatic trauma survivor or responder understand that the acute stress reactions of other people are common and probably transient and do not indicate personal failure or weakness, mental illness, or health problems.
2. Education should include sufficient review of the many ways that post-traumatic problems can present, including symptoms in the ASD/PTSD spectrum, behavioral problems with family and friends, occupational problems, and the potential impact of alcohol or other substance misuse/abuse.
3. Education should also include positive messages by identifying and encouraging positive ways of coping, describing simple strategies to resolve or cope with developing symptoms and problems, and setting expectations for mastery and/or recovery.
4. Provide contact information, should post-traumatic symptoms emerge later.
5. Routine debriefing or formal psychotherapy is not beneficial for asymptomatic individuals and may be harmful. [D]

DISCUSSION

Individuals who do not exhibit symptoms may have family members or close friends who are symptomatic. The clinician should educate them about their role in supporting their loved ones and emphasize that normalization is a concept that can incorporate helping asymptomatic survivors to:

- View other people's (and their own possible future) stress reactions as normal, common, and expectable responses to trauma
- Recognize that sometimes peoples' inadequate attempts to cope with their reactions are also within the range of "normal" for the strange situation
- See that it is natural for them to wonder how they are doing and to be surprised or upset by the intensity, duration, or uncontrollability of their reactions.

The evidence base for the utility of normalization is weak. Few studies have attempted to assess the degree of normalization of survivor attitudes and establish a relationship with PTSD and other outcomes. Also unstudied is whether reassurance of normality and likely recovery, provided by co-survivor peers or helpers, actually serves to promote normalization. Nonetheless, the concept of normalization is consistent with theories of the development and maintenance of PTSD and with research showing a relationship between negative reactions to symptoms and PTSD (Steil & Ehlers, 2000).

Recent literature in the area of trauma has highlighted the potential for interventions to exacerbate trauma reactions. Asymptomatic survivors should not be offered services that extend beyond delivery of Psychological First Aid and education. Psychotherapy intervention may actually cause harm in persons not experiencing symptoms of acute stress (Roberts, Kitchiner et al., 2009b). The general rule of "do no harm" should apply not only to professionals but volunteers alike.

EVIDENCE

	Recommendation	Sources	LE	QE	SR
1	Providing pre- and post-trauma education can help individuals understand and cope with exposure experiences.	Working Group Consensus	III	Poor	I
2	Routine single, or multiple, psychological interventions for **asymptomatic** trauma survivors are NOT effective and may be harmful	Roberts, Kitchiner et al., 2009b	I	Good	D
	Psychological debriefings are not effective	See module I-1: Early Interventions			

LE =Level of Evidence; QE = Quality of Evidence; SR = Strength of Recommendation (see Appendix A)

1

MODULE A: ACUTE STRESS REACTION (ASR) and PREVENTION OF POST-TRAUMATIC STRESS DISORDER (PTSD)

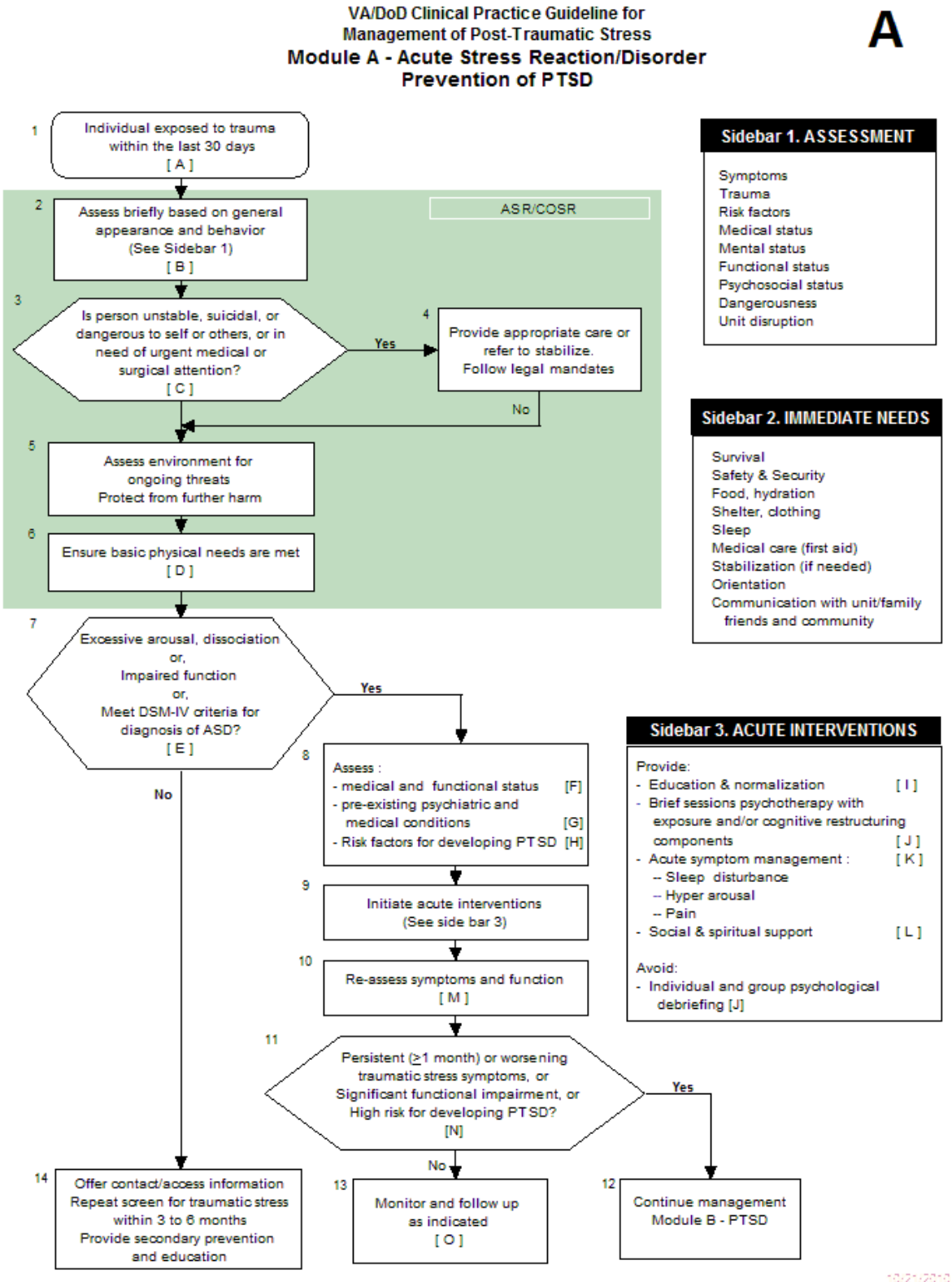
Although acute stress reaction (ASR) is not defined in the DSM-IV, there has long been recognition among mental health professionals that individuals who experience a traumatic event react in certain predictable ways. A key point in the World Health Organization definition (WHO, 1992) of ASR is the assertion that "the symptoms usually appear within minutes of the impact of the stressful stimulus or event and disappear within 2-3 days (often within hours)." This view is echoed in a Guideline for Evidence-Based Early Psychological Intervention for Victims/Survivors of Mass Violence, released in 2002 by a collaborative group of Federal Departments and the American Red Cross: "a sensible working principle in the immediate post-incident phase is to expect normal recovery" (NIMH, 2002).

Screening and needs assessments for individuals, groups, and populations are important for the provision of informed early intervention following a major incident or traumatic event. Initial reactions following trauma are varied, complex, and unstable.

The authors of this guideline have formulated the recommendations discussed below for the management of persons with acute stress reaction (ASR) following a traumatic event. Most of the recommendations in this module are based on group consensus. When available, the evidence and supporting research are presented in evidence tables.

The approach to triage in the immediate response to traumatic exposure for service members with symptoms during Ongoing Military Operations may vary from the management of civilians exposed to traumatic events. Combat and Operational Stress Reaction (COSR) management is targeted to preserve the fighting force and return the service member (SM) to functional status. The annotations for this Module include, when appropriate, specific recommendations addressing the service members with COSR.

MODULE A: ALGORITHM



MODULE A: ANNOTATIONS

1. ASSESSMENT & TRIAGE

A. Trauma Exposure (within the past 30 days)

Acute Stress Reaction (ASR) is a transient condition that often develops in response to a traumatic event. Traumatic events are events that cause a person to fear that he/she may die or be seriously injured or harmed. These events also can be traumatic when the person witnesses them happening to others. Such events often create feelings of intense fear, helplessness, or horror for those who experience them. The traumatic events that can lead to an acute stress reaction are of similar severity to those involved in post-traumatic stress disorder (PTSD).

Combat or Operational Stress Reaction (COSR) is an acute stress reaction of service members during Ongoing Military Operations. COSR specifically refers to a reaction to high-stress events and potentially traumatic event exposure. This reaction is not attributed to an identified medical/surgical condition that requires other urgent treatment (a service member can have COSR concurrent with minor wounds/illnesses).

Among the common types of traumatic events are:

- Combat in a war zone
- Ongoing military operations
- Rape, sexual, or other physical assault
- Natural disaster (e.g., hurricanes, floods, or fires)
- Child physical and/or sexual abuse
- Domestic violence (battering)
- Motor vehicle accidents (MVAs)
- Exposure to the sudden or unexpected death of others
- Sudden life-threatening physical illness (e.g., heart attack or cancer)
- Continuous or reoccurring exposure to traumatic event(s).

Events specific to COSR:

- Intense emotional demands (e.g., rescue personnel and caregivers searching for possibly dying survivors or interacting with bereaved family members)
- Extreme fatigue, weather exposure, hunger, sleep deprivation
- Extended exposure to danger, loss, emotional/physical strain
- Exposure to environmental hazards, such as toxic contamination (e.g., gas or fumes, chemicals, radioactivity)
- While a COSR can result from a specific traumatic event, it generally emerges from cumulative exposure to multiple stressors.

Onset of at least some signs and symptoms may be simultaneous with the trauma itself or may follow the trauma after an interval of hours or days. Symptoms may include depression, fatigue, anxiety, decreased concentration/memory, irritability, agitation, and exaggerated startle response.

B. Assess Briefly Based on General Appearance and Behavior

OBJECTIVE

Identify individuals who may be at risk for endangering themselves or others due to emotional distress or functional incapacity.

BACKGROUND

The transient symptoms or problems that often develop in response to exposure to trauma begin within minutes of the traumatic event and disappear within days (even hours). Symptoms vary greatly but can include a mixture of:

- Anxiety symptoms (e.g., sweating, increased heart rate, and flushing)
- An initial state of 'daze' - narrowing of attention
- Reduced levels of consciousness - disorientation
- Agitation or over-activity
- Depression
- Withdrawal.

There are a number of possible reactions to a traumatic situation, which are considered within the "norm" for persons experiencing traumatic stress. These reactions are considered 'normal' in the sense of affecting most survivors, being socially acceptable, psychologically effective, and self-limited. In the early stage (the first four days after the trauma exposure), it is important not to classify these reactions as "symptoms" in the sense of being indicative of a mental disorder.

RECOMMENDATIONS

1. Identification of a patient with ASR symptoms is based on observation of behavior and function; there is insufficient evidence to recommend a specific screening tool.
2. Individuals exhibiting the following responses to trauma should be screened for ASR:
    a. Physical: exhaustion, hyperarousal, somatic complaints (GI, GU, MS, CV, Resp, NS), or symptoms of conversion disorder
    b. Emotional: anxiety, depression, guilt/hopelessness
    c. Cognitive/mental: amnestic or dissociative symptoms, hypervigilance, paranoia, intrusive re-experiencing
    d. Behavioral: avoidance, problematic substance use.
3. Individuals who experience ASR should receive a comprehensive assessment of their symptoms to include details about the time of onset, frequency, course, severity, level of distress, functional impairment, and other relevant information.
4. Assess for capability to perform routine functions.

Assessment specific to COSR:

5. Assess service member's functional status, to include:
    a. Any changes in productivity
    b. Co-worker or supervisor reports of recent changes in appearance, quality of work, or relationships

c. Any tardiness/unreliability, loss of motivation, or loss of interest

d. Forgetful or easily distracted

e. Screening for substance use.

6. Document symptoms of COSR and obtain collateral information from unit leaders, coworkers, or peers about stressors, function, medical history, and absence or impairment in operation or mission.

7. Consider the service member's role and functional capabilities and the complexity and importance of his/her job.

DISCUSSION

An acute stress reaction (ASR/COSR) may appear concurrent with other wounds or illnesses. Providers should confirm that the symptoms are not due to identified medical/surgical conditions requiring other urgent treatment. ASR may result from a specific traumatic event or from series of events.

In the aftermath of any extreme stressful event, most of those suffering from acute traumatic stress reactions will be easy to spot. Those who have been injured will be obvious. Among the uninjured there will also be many who look stunned, appear pale and faint, or can be seen to be shaking. Some of those who appear to be suffering from trauma may not even be the actual victims of the disaster but witnesses or rescuers who may be deeply affected by what they are seeing. Some may not be immediately identifiable as traumatized, because they may be highly active - looking for others or looking after others and organizing help and rescue. A percentage of these may, in the next days or weeks, develop post-traumatic stress disorder (PTSD).

Practitioners who are managing service members suffering from stress reactions or COSR should consider a variety of factors when deciding when a service member is ready to return to duty including the severity of the condition, the level of occupational impairment, nature and complexity of the occupation and level of social support.

C. Unstable, Dangerous to Self or Others, or Need for Urgent Medical Attention

OBJECTIVE

Protect individuals who may be at risk for endangering themselves or others due to emotional distress or functional incapacity.

BACKGROUND

Emergency treatment, administered to an injured person before professional medical care is available, can be applied to stress reactions of the mind as well as to physical injuries of the body. Acute interventions can be envisioned as the mental health correlate of physical first aid, with the goal being to "stop the psychological bleeding." The first, most important measure should be to eliminate (if possible) the source of the trauma or to remove the victim from the traumatic, stressful environment. Once the patient is in a safe situation, the provider should attempt to reassure the patient, encourage a professional healing relationship, encourage a feeling of safety, and identify existing social supports. Establishing safety and assurance may enable people to get back on track, and maintain their pre-trauma stable condition.

RECOMMENDATIONS

1. Address acute medical/behavioral issues to preserve life and avoid further harm by:
    a. Providing appropriate medical/surgical care or referring to stabilize
    b. Evaluating the use of prescribed medications
    c. Preventing possible biological or chemical agent exposure
    d. Managing substance intoxication or withdrawal
    e. Stopping self-injury or mutilation
    f. Addressing inability to care for oneself.
2. Arrange a safe, private, and comfortable environment for continuation of the evaluation:
    a. Assess danger to self or others (e.g., suicidal, or homicidal behavior)
    b. Establish a working treatment alliance with the patient
    c. Maintain a supportive, non-blaming, non-judgmental stance throughout the evaluation
    d. Assist with the removal of any ongoing exposure to stimuli associated with the traumatic event
    e. Minimize further traumas that may arise from the initial traumatic event
    f. Assess and optimize social supports
    g. Secure any weapons and explosives.
3. Legal mandates should be followed:
    a. Reporting of violence, assault
    b. Confidentiality for the patient
    c. Mandatory testing
    d. Attending to chain of evidence in criminal cases (e.g., rape, evaluation)
    e. Involuntary Commitment procedures if needed.
4. Carefully consider the following potential interventions to secure safety:
    a. Find safe accommodation and protect against further trauma
    b. Voluntary admission if suicidal
    c. Restraint/seclusion only if less restrictive measures are ineffective
    d. Provide medications managing specific symptoms as needed (e.g., sleep, pain).
5. Educate and “normalize” observed psychological reactions to the chain of command.
6. Evacuate to next level of care if unmanageable, if existing resources are unavailable, or if reaction is outside of the scope of expertise of the care provider.

DISCUSSION

Foa et al. (2000) rank “suicidality” among factors that will affect treatment decisions for PTSD. This factor must also be considered in the immediate post-trauma period: “self-destructive and impulsive behaviors, while not part of the core PTSD symptom complex, are recognized as associated features of this disorder that may profoundly affect clinical management. Therefore, the routine assessment of all patients

presenting with acute stress symptoms after exposure to a traumatic stressor should include a careful evaluation of current suicidal ideation and past history of suicidal attempts. Risk factors for suicide should also be assessed, such as current depression and substance abuse. If significant suicidality is present, it must be addressed before any other treatment is initiated." Likewise, the patient must be assessed for any signs of violence toward others, or threat of violence in the home environment (e.g., ongoing battering), and any risk of violence should be an indication for immediate treatment.

While there is little research on these issues for acute stress reaction per se, the literature suggests some general trends for persons with PTSD that may inform clinical management of ASR. For example, individuals with sub-threshold PTSD are at high risk for suicidal ideation (Marshall et al., 2001) and, for women, suicide attempts (Breslau, 2001; Ferrada-Noli et al., 1998; Kaslow et al., 2000; Prigerson et al., 1999). For young adults, aggressive symptoms may be predictive of suicidality in men and elevated symptoms of PTSD and/or depression may be more predictive in women (Prigerson et al., 1999). Some individuals with stress reactions could be at risk for violence toward others. This can be manifested through explosivity and anger problems and may predict risk for violent behavior.

Optimizing existing social supports is helpful in settings of acute stress and may decrease risk of suicidality in PTSD (Kotler et al., 2001).

For extended discussion of dangerousness to self or others, see Module B: Annotation C – Assessment of Dangerousness.

D. Ensure Basic Physical Needs Are Met

OBJECTIVE

Ensure that trauma-exposed persons with acute stress symptoms have their basic needs met.

BACKGROUND

Trauma victims often have significant disruption to their routines for sleep, nutrition, exercise, access to finances, and healthcare. Their normal shelter, clothing, and other basic resources may be destroyed or inaccessible. These disruptions can be additionally traumatizing.

Early interventions should typically seek to address the needs of the individual person, with the aim of promoting normal recovery, resiliency, and personal growth and avoiding additional harm (see Table A1-Early Interventions).

Individual persons who were exposed to trauma as members of a group/unit that existed prior to the trauma event (e.g., police units, firefighters, or military units), may also benefit from interventions addressing the collective outcomes such as social order and community or unit cohesion. Some of the acute interventions, such as psychoeducation, may be provided in a group format to maintain unit integrity and promote continuity with established relationships.

Table A - 1 Early Interventions after Exposure to Trauma (<4 days after exposure)

	Balance of Benefit and Harm			
SR	**Significant Benefit**	**Some Benefit**	**Unknown Benefit**	**No Benefit Potential Harm**
I	--	Psychological First Aid Psychoeducation and normalization Social support	Spiritual support	--
D				Psychological debriefing

SR = Strength of recommendation (see Appendix A)

RECOMMENDATIONS

1. Acute intervention should ensure that the following needs are met:
    a. Safety/security/survival
    b. Food, hydration, clothing, hygiene, and shelter
    c. Sleep
    d. Medications (i.e., replace medications destroyed/lost)
    e. Education as to current status
    f. Communication with family, friends, and community
    g. Protection from ongoing threats/toxins/harm. If indicated, reduce use of alcohol, tobacco, caffeine, and illicit psychoactive substances.

2. Provide Psychological First Aid to:
    a. Protect survivors from further harm
    b. Reduce physiological arousal
    c. Mobilize support for those who are most distressed
    d. Keep families together and facilitate reunion with loved ones
    e. Provide information and foster communication and education
    f. Use effective risk communication techniques.

Interventions Specific for Members of Pre-existing Group (e.g., COSR):

3. Treat according to member's prior role and not as a "patient."
4. Assure or provide the following, as needed:
    a. Reunion or ongoing contact with group/unit
    b. Promote continuity with established relationships (e.g., primary group)
    c. Respite from intense stress
    d. Comfortable environment (e.g., thermal comfort)
    e. Consider psychoeducation and discussion in a group format
    f. Assign job tasks and recreational activities that will restore focus and confidence and reinforce teamwork (limited duty).

DISCUSSION

Psychological first aid should be envisioned as the mental health correlate of physical first aid, with the goal being to "stop the bleeding." The patient should be removed from the traumatic situation. When the patient is in a safe situation, the clinician should attempt to reassure the patient and encourage a feeling of safety.

In their Disaster Mental Health Response Handbook (Raphael, 2000), a group of PTSD experts propose three stages of care:

Protect:

Find ways to protect survivors from further harm and from further exposure to traumatic stimuli. If possible, create a "shelter" or safe haven for them, even if it is symbolic. The fewer traumatic stimuli people see, hear, smell, taste, or feel, the better off they will be.

Direct:

Kind and firm direction is needed and appreciated. Survivors may be stunned, in shock, or experiencing some degree of dissociation. When possible, direct ambulatory survivors:

- Away from the site of destruction
- Away from severely injured survivors
- Away from continuing danger.

Connect:

Survivors who are encountered will usually have lost connection to the world that was familiar to them. A supportive, compassionate, and nonjudgmental verbal or nonverbal exchange between you and survivors may help to give the experience of connection to the shared societal values of altruism and goodness. Help survivors connect:

- To loved ones
- To accurate information and appropriate resources
- To locations where they will be able to receive additional support
- To unit comrades and mission, fostering vertical and horizontal cohesion.

Triage:

A majority of survivors experience normal stress reactions. However, some may require immediate crisis intervention to help manage intense feelings of panic or grief. Signs of panic are trembling, agitation, rambling speech, and erratic behavior. Signs of intense grief may be loud wailing, rage, or catatonia. In such cases, attempt to quickly establish therapeutic rapport, ensure the survivor's safety, acknowledge and validate the survivor's experience, and offer empathy. Medication may be appropriate and necessary, if available.

Psychological First Aid: *(See Table A-2)*

Psychological first aid was coined in Raphael's book, 'When Disaster Strikes: How Individual and Communities Cope with Catastrophe' (1986). It is included as part of the *Fundamental Criteria for First Aid* knowledge and skills that soldiers should be trained in order to save themselves or other soldiers in casualty situations.

Table A - 2 Key Elements of Psychological First Aid (PFA)

1. *Contact and Engagement* - Respond to contacts initiated by affected persons, or initiate contacts in a non-intrusive, compassionate, and helpful manner
2. *Safety and Comfort* - Enhance immediate and ongoing safety, and provide physical and emotional comfort
3. *Stabilization (if needed)* - Calm and orient emotionally overwhelmed or distraught survivors
4. *Information Gathering - Current Needs and Concerns* - Identify immediate needs and concerns, gather additional information, and tailor PFA interventions
5. *Practical Assistance* - Offer practical help to the survivor in addressing immediate needs and concerns
6. *Connection with Social Supports* - Help establish opportunities for brief or ongoing contacts with primary support persons or other sources of support, including family members, friends, and community helping resources
7. *Information on Coping* - Provide information (about stress reactions and coping) to reduce distress and promote adaptive functioning
8. *Linkage to Collaborative Services* - Link survivors with needed services and inform them about available services that may be needed in the future.

These core goals of PFA constitute the basic objectives of providing early assistance (e.g., within days or weeks following an event). The amount of time spent on each goal will vary from person to person and with different circumstances, according to need.

The complete document describing PFA components can be found at: http://www.vdh.state.va.us/EPR/pdf/PFA9-6-05Final.pdf

The FM 21-11 First Aid for Soldiers document (1991) states: (www.medtrng.com/Fm21_11/fm211_8.htm)

> "The psychological first aid is most needed at the first sign that a soldier can not perform the mission because of emotional distress. Stress is inevitable in combat, in hostage and terrorist situations, and in civilian disasters, such as floods, hurricanes, tornadoes, and industrial and aircraft catastrophes. Most emotional reactions to such situations are temporary, and the person can still carry on with encouragement. Painful or disruptive symptoms may last for minutes, hours, or a few days. However, if the stress symptoms are seriously disabling, they may be psychologically contagious and endanger not only the emotionally upset individual but also the entire unit. In such situations, you may be working beside

someone who cannot handle the impact of disaster. Even when there is no immediate danger of physical injury, psychological harm may occur."

"Psychological first aid really means assisting people with emotional distress whether it results from physical injury, disease, or excessive traumatic stress. Emotional distress is not always as visible as a wound, a broken leg, or a reaction to pain from physical damage. However, overexcitement, severe fear, excessive worry, deep depression, misdirected aggression, or irritability and anger are signs that stress has reached the point of interfering with effective coping."

"Psychological first aid should go hand in hand with physical first aid. The discovery of a physical injury or cause for an inability to function does not rule out the possibility of a psychological injury (or vice versa). A physical injury and the circumstances surrounding it may actually cause an emotional injury that is potentially more serious than the physical injury; both injuries need treatment. The person suffering from pain, shock, fear of serious damage to his body, or fear of death does not respond well to joking, indifference, or fearful-tearful attention. Fear and anxiety may take as high a toll on the soldier's strength as does the loss of blood." (The Department of the Army; Washington, DC, 4 December 1991)

Specific Interventions for COSR:

Combat Operation Stress Control (COSC) utilizes the management principles of brevity, immediacy, contact, expectancy, proximity, and simplicity (BICEPS). These principles apply to all COSC interventions or activities throughout the theater, and are followed by COSC personnel in all BH/COSC elements. These principles may be applied differently based on a particular level of care and other factors pertaining to mission, enemy, terrain and weather, troops and support available, time available, and civil considerations (METT-TC).

The actions used for COSC (commonly referred to as the 6 Rs) involve the following actions:

Reassure of normality (normalize the reaction)

Rest (respite from combat or break from work)

Replenish bodily needs (such as thermal comfort, water, food, hygiene, and sleep).

Restore confidence with purposeful activities and talk

Retain contact with fellow soldiers and unit

Remind / **R**ecognize emotion of reaction (specifically potentially life-threatening thoughts and behaviors).

For additional information see COSR protocols for DoD specific services.

E. Person has Trauma-Related Symptoms, Significant Impaired Function, or Diagnosis of ASD

Identify patients who have excessive post-traumatic stress symptoms or significant distress impaired function, or are diagnosed with ASD.

BACKGROUND

Since people who develop ASD are at greater risk of developing PTSD, they should be identified and offered treatment as soon as possible. Although ASD does not occur in all people who later develop PTSD, treatment should be considered for all acutely traumatized people with ASD, those with severe PTSD symptoms but do not meet ASD diagnostic criteria, and those with functional impairment because of acute physiological symptoms (e.g., hyperarousal).

Some patients with an acute stress reaction may benefit from augmentation of the acute intervention and additional follow-up. Because people vary in their reaction and in the rate that they recover from traumatic stress, some individuals may require more time or an adjustment of the treatment prior to improvement. Some want and feel a need to discuss the event, and some have no such need. Respect individual and cultural preferences in the attempt to meet their needs as much as possible. Allow for normal recovery and monitor.

RECOMMENDATIONS

1. Acutely traumatized people, who meet the criteria for diagnosis of ASD, and those with significant levels of post-trauma symptoms after at least two weeks post-trauma, as well as those who are incapacitated by acute psychological or physical symptoms, should receive further assessment and early intervention to prevent PTSD.
2. Trauma survivors, who present with symptoms that do not meet the diagnostic threshold for ASD, or those who have recovered from the trauma and currently show no symptoms, should be monitored and may benefit from follow-up and provision of ongoing counseling or symptomatic treatment.
3. Service members with COSR who do not respond to initial supportive interventions may warrant referral or evacuation.

DISCUSSION

Stress reactions produce biological, psychological, and behavioral changes. Biological alterations include disruptions in neurochemicals, sleep patterns, hyperarousal, and somatic symptoms (e.g., pain, gastrointestinal symptoms). Psychological changes include: mood disturbances (e.g., emotional lability, irritability, blunting, numbing), anxiety (e.g., increased worry, ruminations), and cognitive disturbances (e.g., memory impairment, confusion, and impaired task completion).

Not all individuals who are exposed to trauma or who have a COSR require a mental health referral. However, those service members who are deteriorating or who are not responding to acute supportive interventions need to be identified and evacuated to a more definitive level of care. Also, patients who have a high potential for dangerousness or the development of symptoms suggestive of a stress-related disorder (e.g., ASD) also need to be identified and referred to a facility that can provide appropriate mental healthcare.

Patients who do not respond to first-line supportive interventions may warrant treatment augmentation or a mental health referral. Clear indications for a mental health referral include: a worsening of stress-related symptoms, new onset of

dangerousness or maladaptive coping to stress, exacerbation of co-morbid psychiatric conditions, or deterioration in function. Because patients with new onset stressors, poor social supports, or inadequate coping skills may be at heightened risk to develop PTSD, a mental health referral is also indicated.

Acute Stress Disorder (ASD)

Different types of trauma can lead to ASD, from interpersonal assaultive violence to accidents to combat related trauma. As many as ninety percent of individuals, who experience sexual assault, will have acute stress symptoms but not ASD (Breslau, 1998). Additionally, surveys from the OIF/OEF combat theaters indicate that about 10 to 18 percent of deployed US combat forces experience trauma-related stress symptoms (as measured with PCL cutoff score of 50+).

Prior to DSM-IV (American Psychiatric Association, 1994), severe distress occurring in the month after a traumatic event was not regarded as a diagnosable clinical problem. Although this prevented the pathologizing of transient reactions, it hampered the identification of more severely traumatized individuals who might have benefited from early interventions. To address this issue, DSM-IV introduced the diagnosis of acute stress disorder (ASD) to describe those acute reactions associated with an increased likelihood of developing chronic PTSD (see Table A - 3). A diagnosis of ASD is given when an individual experiences significantly distressing symptoms of re-experiencing, avoidance, and increased arousal within 4 weeks of the trauma. These symptoms must be present for at least two days before the diagnosis of ASD can be made. The DSM-IV diagnosis of ASD requires that the victim report at least three of the following five symptoms labeled as indicators of dissociation: numbing, reduced awareness of surroundings, derealization, depersonalization, and dissociative amnesia. These requirements are based on evidence found in previous studies that dissociative symptoms at the time of (or shortly after) the traumatic event are predictive of the subsequent development of chronic PTSD (Bremner et al., 1992; Marmar et al., 1994; Koopman et al., 1994). Thus, the fundamental differences between PTSD and ASD involve time elapsed since the trauma and the relative emphasis on dissociative symptoms in the ASD diagnosis.

Table A - 3 Diagnostic criteria for 308.3 Acute Stress Disorder (DSM-IV)

A. The person has been exposed to a traumatic event in which both of the following were present:
   - 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
   - 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:
   - a subjective sense of numbing, detachment, or absence of emotional responsiveness
   - a reduction in awareness of his or her surroundings (e.g., "being in a daze")
   - derealization
   - depersonalization
   - dissociative amnesia (i.e., inability to recall an important aspect of the trauma)

C. The traumatic event is persistently re-experienced 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, and 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 pre-existing Axis I or Axis II disorder.

F. Assess Medical and Functional Status

OBJECTIVE

Obtain complete history, physical examination, relevant laboratory tests, and assessment of functioning to determine course of treatment.

BACKGROUND

One of the key goals of ASR supportive care is to address immediate physical health problems and to assist the individual in beginning to return to a normal level of function. In order to do this, the clinician or caregiver must assess the individual's current state of health and functioning.

RECOMMENDATIONS

1. Medical status should be obtained for all persons presenting with symptoms to include:
    a. History, physical examination, and a neurological examination
    b. Use of prescribed medications, mood or mind-altering substances, and possible biological or chemical agent exposure
    c. A mini-mental status examination (MMSE) to assess cognitive function if indicated.
2. The history and physical examination findings should lead the provider to other assessments as clinically indicated. Based on the clinical presentation, assessment may include:
    a. Screen for toxicology if the symptom presentation indicates
    b. Radiological assessment of patients with focal neurological findings or possible head injury
    c. Appropriate laboratory studies to rule out medical disorders that may cause symptoms of acute stress reactions (e.g., complete blood count [CBC], chemistry profile, thyroid studies, HCG, EKG, EEG).
3. A focused psychosocial assessment should be performed to include assessment of active stressors, losses, current social supports, and basic needs (e.g., housing, food, and financial resources).
4. A brief assessment of function should be completed to evaluate: 1) objectively impaired function based on general appearance and behavior; 2) subjectively impaired function; 3) baseline level of function (LOF) vs. current LOF; and 4) family and relationship functioning.

DISCUSSION

Whenever possible, providers should include assessment of any physical injuries, review of targeted H&P and laboratory results (if available), assessment of the individual's level of functioning, and level of family and relationship functioning. Ideally, the current clinical picture should be compared to the individual's pre-trauma state, but often this may not be possible in the immediate aftermath of a traumatic event. Evaluation of the patient's level of function is warranted, because evidence has shown that functional impairment after trauma is a predictor for later development of PTSD (Norris et al., 2002).

G. Assess Pre-Existing Psychiatric and Medical Conditions

OBJECTIVE

Identify patients at risk for complications.

BACKGROUND

Circumstances brought about by a traumatic event may complicate any existing psychiatric conditions or may exacerbate pre-existing pathology.

RECOMMENDATIONS

1. Assess patients for pre-existing psychiatric conditions to identify high-risk individuals and groups.
2. Assure access and adherence to medications that the patient is currently taking.
3. Refer patients with pre-existing psychiatric conditions to mental health specialty when indicated or emergency hospitalization if needed.

DISCUSSION

The NIMH (2002) guideline addresses the need to manage pre-existing psychiatric and medical conditions. The authors point to the "special needs of those who have experienced enduring mental health problems, those who are disabled, and other high-risk groups who may be vulnerable and less able to cope with unfolding situations." They also call for additional attention to be paid to members of these groups in the immediate post-trauma period. However, they also emphasize that "the presumption of clinically significant disorders in the early post-incident phase is inappropriate, except for individuals with preexisting conditions."

H Assess Risk Factors for Developing ASD/PTSD

BACKGROUND

Not all trauma survivors develop permanent stress disorders. Early identification of those at-risk for negative outcomes following trauma can facilitate prevention, referral, and treatment. Screening for those at greatest risk should address past and current psychiatric and substance use problems and treatment, prior trauma exposure, pre-injury psychosocial stressors, and existing social support.

RECOMMENDATIONS

1. Trauma survivors who exhibit symptoms or functional impairment should be screened for the following risk factors for developing ASD/PTSD:

Pre-traumatic factors

1. Ongoing life stress
2. Lack of social support
3. Young age at time of trauma
4. Pre-existing psychiatric disorders, or substance misuse
5. History of traumatic events (e.g., MVA)
6. History of post-traumatic stress disorder (PTSD).
7. Other pre-traumatic factors, including: female gender, low socioeconomic status, lower level of education, lower level of intelligence, race (Hispanic, African-American, American Indian, and Pacific Islander), reported abuse in

childhood, report of other previous traumatization, report of other adverse childhood factors, family history of psychiatric disorders, and poor training or preparation for the traumatic event.

Peri-traumatic or trauma-related factors

1. Severe trauma
2. Physical injury to self or others
3. Type of trauma (combat, interpersonal traumas such as killing another person, torture, rape, or assault convey high risk of PTSD)
4. High perceived threat to life of self or others
5. Community (mass) trauma
6. Other peri-traumatic factors, including: history of peri-traumatic dissociation.

Post-traumatic factors

1. Ongoing life stress
2. Lack of positive social support
3. Bereavement or traumatic grief
4. Major loss of resources
5. Negative social support (shaming or blaming environment)
6. Poor coping skills
7. Other post-traumatic factors, including: children at home and a distressed spouse.

DISCUSSION

Risk Factors for ASD

When evaluating risk factors for ASD, the clinician should keep in mind that ASD is no longer diagnosed later than four weeks after a traumatic event. Thus, not enough time will have passed following the trauma for many post-trauma factors to have had their full impact on the course of symptoms.

Risk Factors for PTSD

When evaluating risk factors for developing PTSD, the clinician should keep in mind that PTSD is defined as occurring only after four weeks have elapsed following a traumatic event. PTSD symptoms, however, may not appear until a considerable time has passed, sometimes surfacing years later.

For further discussion of risk factors for PTSD - See Module B: Annotation F

2. TREATMENT

I. Provide Education and Normalization / Expectancy of Recovery

OBJECTIVE

Help trauma survivors cope with ASR/COSR by providing information that may help them manage their symptoms and benefit from treatment.

BACKGROUND

Education for trauma survivors and their families may help normalize common reactions to trauma, improve coping, enhance self-care, facilitate recognition of significant problems and increase knowledge of, and access to, services. Individuals should be reassured about common reactions to traumatic experiences and be advised regarding positive and problematic forms of coping with them.

Information about social support and stress management is particularly important. Opportunities to discuss emotional concerns in individual, family, or group meetings can enable survivors to reflect on what has happened. Education regarding indicators that initial acute reactions are failing to resolve will be important. Signs and symptoms of PTSD, anxiety, depression, substance use disorders, and other difficulties should be explained. Survivors will need information about financial, mental health, rehabilitation, legal, and other services available to them, as well as education about common obstacles to pursuing needed services.

RECOMMENDATION:

1. All survivors should be given educational information to help normalize common reactions to trauma, improve coping, enhance self-care, facilitate recognition of significant problems, and increase knowledge of and access to services. Such information can be delivered in many ways, including public media, community education activities, and written materials.

DISCUSSION:

Immediate post-trauma distress will remit naturally for many patients (Blanchard et al., 1995), and provision of mental health services may be unnecessary. Hypothetically, it is even possible that too much focus on mental health issues may be iatrogenic for some survivors, centering their attention on symptoms and problems and making attention and caring contingent on needing such help.

J. Initiate Brief Intervention

OBJECTIVE

To lessen the physical, psychological, and behavioral morbidity associated with acute stress reaction (ASR), hasten the return to full function (duty), and reduce the risk for development of ASD or PTSD following a traumatic event.

BACKGROUND

It is likely that not all patients will require intervention immediately following a traumatic occurrence. Depending on the intensity and duration of the trauma, there will be people who will make it through unharmed. Often, if a person appears to be coping well and denies symptoms of ASD or PTSD, specialized care may not be needed.

For people who show symptoms of ASD or PTSD (including symptoms of intrusive recollections, avoidance, numbing, and physiological hyperarousal when confronted with reminders of the trauma), brief acute intervention may be indicated.

Early interventions may need to assist the individuals with anticipating problems in using their support system. This may be particularly important in light of the fact that the psychological aftermath of trauma may significantly disrupt a person's capacity to use others to cope with and manage post-traumatic symptoms and daily demands. Table A-4 summarizes the interventions and their potential benefit in the first month after exposure to the trauma.

Table A-4 Early Interventions after Exposure to Trauma (4 to 30 days after exposure)

	Balance of Benefit and Harm			
SR	**Significant Benefit**	**Some Benefit**	**Unknown Benefit**	**No Benefit**
A	- Brief Cognitive Behavioral Therapy (4-5 sessions)			
B				
C		- Social support		
D				- Individual psychological debriefing ⊘ - Formal psychotherapy for asymptomatic survivors ⊘ - Benzodiazepines ⊘ - Typical Antipsychotics ⊘
I		- Psychoeducation and normalization	- Imipramine - Propranolol - Prazosin - Other Antidepressants - Anticonvulsants - Atypical Antipsychotics - Spiritual support - Psychological First Aid	- Group psychological debriefing

⊘= *Potential harm; SR = Strength of recommendation (see Appendix A)*

RECOMMENDATIONS

The following treatment recommendations should apply for all acutely traumatized people who meet the criteria for diagnosis of ASD, and for those with significant levels of acute stress symptoms that last for more than two weeks post-trauma, as well as those who are incapacitated by acute psychological or physical symptoms.

1. Continue providing psychoeducation and normalization.
2. Treatment should be initiated after education, normalization, and Psychological First Aid has been provided and after basic needs following the trauma have been made available.

3. There is insufficient evidence to recommend for or against the use of Psychological First Aid to address symptoms beyond 4 days following trauma. [I]
4. Survivors who present symptoms that do not meet the diagnostic threshold of ASD or PTSD should be monitored and may benefit from follow-up and provision of ongoing counseling or symptomatic treatment.
5. Recommend monitoring for development of PTSD using validated symptom measures (e.g., PTSD Checklist, other screening tools for ASD/PTSD).

6. Psychotherapy:

a. Consider early brief intervention (4 to 5 sessions) of cognitive-based therapy (CBT) that includes exposure-based therapy, alone or combined with a component of cognitive re-structuring therapy for patients with significant early symptom levels, especially those meeting diagnostic criteria for ASD. [A]

b. Routine formal psychotherapy intervention for asymptomatic individuals is not beneficial and may be harmful. [D]

c. Strongly recommend **against** individual Psychological Debriefing as a viable means of reducing acute stress disorder (ASD) or progression to post-traumatic stress disorder (PTSD). [D]

d. The evidence does not support a single session group Psychological Debriefing as a viable means of reducing acute stress disorder (ASD) or progression to post-traumatic stress disorder, but there is no evidence of harm (Note: this is not a recommendation pertaining to Operational Debriefing). [D]

e. Groups may be effective vehicles for providing trauma-related education, training in coping skills, and increasing social support, especially in the context of multiple group sessions. [I]

f. Group participation should be voluntary.

7. Pharmacotherapy:

a. There is no evidence to support a recommendation for use of a pharmacological agent to prevent the development of ASD or PTSD. [I]

b. Strongly recommend **against** the use of benzodiazepines to prevent the development of ASD or PTSD [D]

For discussion of the supporting evidence and grading of the recommendations, see Module I-1: Early Interventions to Prevention of PTSD

DISCUSSION

ASD Treatment

The relationship between ASD and PTSD was examined in three prospective studies. Classen and colleagues (1998) studied the acute stress reactions of bystanders to a mass shooting in an office building. They assessed 36 employees (bystanders) 8 days after the shooting. Between 7 and 10 months later, they reassessed 32 employees for post-traumatic stress symptoms and found that 33 percent of them met criteria for ASD and that meeting criteria for ASD was a strong predictor of PTSD

(accounting for 19 percent of the variance), as well as intrusion (accounting for 53 percent of the variance) and avoidance (accounting for 45 percent of the variance).

In another prospective study, Harvey and Bryant (1998) examined the relationship between ASD and PTSD in 92 motor vehicle accident survivors. From the twelve participants (13 percent) who met criteria for ASD within 2 to 26 days of the accident, 78 percent met criteria for PTSD 6 months later. Nineteen participants (21 percent) met some but not all of the criteria for ASD; of the 15 individuals available for follow-up, 9 (60 percent) met criteria for PTSD. From the 61 participants who did not meet the criteria for ASD; only 2 met criteria for PTSD. This study provides strong evidence of ASD being a predictor of PTSD. Nevertheless, Harvey and Bryant concluded that the current criteria for ASD might be too stringent for ASD to be used to predict the risk for PTSD. Harvey and Bryant (1998a) also examined the relationship between ASD and PTSD for a subset (n=79) of the motor vehicle accident survivors who suffered mild traumatic brain injury as a result of the accident. They were particularly interested in the utility of ASD as a predictor of PTSD in individuals with post-concussive symptoms that could overlap with ASD symptoms. Their results were similar to previously reported findings: 14 percent met criteria for ASD; six months after the event, 82 percent of those with ASD also met criteria for PTSD.

In another prospective study, Brewin and colleagues (1999) evaluated the use of ASD to predict PTSD in 157 survivors of violent assault. Participants were assessed for several ASD symptoms using items from the Post-Traumatic Stress Disorder Symptoms Scale; additional items were generated to determine whether the event met the ASD criterion. Nineteen percent of participants met criteria for ASD, and 20 percent met criteria for PTSD at 6-month follow-up. They found that meeting full criteria for ASD was a better predictor of PTSD than any of the symptom clusters. Eighty three percent of participants who met criteria for ASD were diagnosed with PTSD six months later.

Research suggests that relatively brief but specialized interventions may effectively prevent PTSD in some subgroups of trauma patients. Several controlled trials have suggested that brief (i.e., 4 to 5 sessions) cognitive-behavioral treatments, comprised of education, breathing training/relaxation, imaginal and *in vivo* exposure, and cognitive restructuring, delivered within weeks of the traumatic event, can often prevent PTSD in survivors of sexual and non-sexual assault (Foa et al., 1995) and MVAs and industrial accidents (Bryant et al., 1998 , 1999). Brief intervention with patients hospitalized for injury has been found to reduce alcohol consumption in those with existing alcohol problems (Gentilello et al., 1999). Controlled trials of brief early intervention services targeted at other important trauma sequelae (e.g., problems returning to work, depression, family problems, trauma recidivism, and bereavement-related problems) remain to be conducted, but it is likely that targeted interventions may be effective in these arenas for at least some survivors.

Two well-designed studies offer evidence that brief treatment interventions utilizing a combination of cognitive behavioral techniques may be effective in preventing PTSD in a significant percentage of subjects. In a study of a brief treatment program for recent sexual and nonsexual assault victims who all met criteria for PTSD, Foa at al. (1995) compared repeated assessments *vs*. a Brief Prevention Program (BPP) (four sessions of trauma education, relaxation training, imaginal exposure, *in vivo* exposure, and cognitive restructuring). Two months posttrauma, only 10 percent of the BPP group met criteria for PTSD, whereas 70 percent of the repeated assessments group met criteria for PTSD. In a study of motor vehicle and industrial accident victims who met criteria for ASD, Bryant et al. (1998) compared five

sessions of nondirective supportive counseling (support, education, and problem-solving skills) *vs*. a brief cognitive-behavioral treatment (trauma education, progressive muscle relaxation, imaginal exposure, cognitive restructuring, and graded *in vivo* exposure to avoided situations). Immediately post-treatment, 8 percent in the CBT group met criteria for PTSD, versus 83 percent in the supportive counseling group. Six months post-trauma, 17 percent in the CBT group met criteria for PTSD versus 67 percent in supportive counseling. One important caveat to these interventions is that the dropout rate was high, and the authors concluded that *those with more severe symptoms might need supportive counseling prior to more intensive cognitive behavioral interventions.*

In addition to targeted brief interventions, some trauma survivors may benefit from follow-up provision of ongoing counseling or treatment. Candidates for such treatment would include survivors with a history of previous traumatization (e.g., survivors of the current trauma who have a history of childhood physical or sexual abuse) or pre-existing mental health problems.

EVIDENCE

	Recommendation	Sources	LE	QE	SR
1	Monitor patient with ASD for development of PTSD (ASD predictor of PTSD).	Brewin et al., 1999 Bryant et al., 1998, 1999	I	Good	A
2	Brief intervention of CBT (4 to 5 sessions).	See Module I-1: Brief early CBT			

LE =Level of Evidence; QE = Quality of Evidence; SR = Strength of Recommendation (see Appendix A)

K. Acute Symptom Management

BACKGROUND

Survivors of trauma may not complain directly of ASD symptoms, such as re-experiencing or avoidance. Instead, they may complain of sleeping problems, pain, or other somatic concerns. After addressing immediate needs and providing education and intervention, alleviating these symptoms will make it easier for survivors to cope and recover from their traumatic experience.

RECOMMENDATIONS

1. Symptom-specific treatment should be provided after education, normalization, and basic needs are met.
2. Consider a short course of medication (less than 6 days), targeted for specific symptoms in patients post-trauma
    a. Sleep disturbance/insomnia
    b. Management of pain
    c. Irritation/excessive arousal/anger.
3. Provide non-pharmacological intervention to address specific symptoms (e.g., relaxation, breathing techniques, avoiding caffeine) to address both general recovery and specific symptoms (sleep disturbance, pain, hyperarousal, or anger).

For discussion of the supporting evidence of the recommendations see Module I-3: Management of Specific Symptoms

L1. Facilitate Spiritual Support

BACKGROUND

Religion and spirituality may provide a framework by which many survivors of trauma construct a meaningful account of their experience and seek solace, and may provide a useful focus for intervention with trauma survivors. The terms "religious" and "spiritual" are both used in the clinical literature to refer to a set of beliefs and practices to which individuals may turn for support following a traumatic event.

RECOMMENDATIONS

1. Ensure patient access to spiritual care when sought.
2. Assess for spiritual needs.
3. Provide opportunities for grieving for losses (providing space and opportunities for prayers, mantras, rites, and rituals and end-of-life care, as determined important by the patient).

DISCUSSION

For discussion of the supporting evidence of the recommendations see Module I-2: D2- Spiritual **Support.**

L2. Facilitate Social Support

BACKGROUND

PTSD is often associated with withdrawal from participation in social activities, limited friendships, and reduced emotional intimacy. Some research also suggests that veterans with PTSD have greater rates of social anxiety disorder. Poor social support predicts development of PTSD and a more chronic course of the disorder. Veterans with PTSD who are more involved in the community are more likely to show remission in PTSD symptoms than those with less community involvement and adjustment to peacekeeping is significantly related to self-disclosure, especially to supportive significant others. Overcoming problems in social functioning and promoting social participation may require active, sustained intervention. When indicated, improvements in social functioning should be established as a formal treatment goal. Social support is critical for helping the individual cope after a trauma has occurred. It may be necessary to identify potential sources of support and facilitate support from others (e.g., partners, family, friends, work colleagues, and work supervisors). Survivors can also be taught a range of social skills to facilitate social participation and support-seeking.

RECOMMENDATIONS

1. Immediately after trauma exposure, preserve an interpersonal safety zone protecting basic personal space (e.g., privacy, quiet, personal effects).
4. As part of Psychological First Aid, reconnect trauma survivors with previously supportive relationships (e.g., family, friends, unit members) and link with additional sources of interpersonal support.
1. Assess for impact of PTSD on social functioning.
5. Facilitate access to social support and provide assistance in improving social functioning, as indicated.

DISCUSSION

Optimizing existing social supports is helpful in settings of acute stress and may decrease risk of suicide in PTSD (Kotler et al., 2001). For example, higher social support for women who have experienced domestic violence may reduce risk of PTSD and other mental disorders (Coker et al., 2002).

3. RE-ASSESSMENT

M. Reassess Symptoms and Function

OBJECTIVE

Identify patients with persistent traumatic stress symptoms, related dysfunction, or additional treatment needs.

BACKGROUND

Clinical reassessment of response to the acute intervention is indicated to determine if there are persistent symptoms and, if necessary, to develop a follow-up plan.

Especially important are acute levels of traumatic stress symptoms, which predict chronic problems; for example, more than three-quarters of MVA patients diagnosed with ASD will have chronic PTSD at 6 months post-trauma.

In follow-up appointments, it will be important to screen for PTSD and other anxiety disorders, depression, alcohol and substance abuse, problems with return to work and other productive roles, adherence to medication regimens and other appointments, and potential for re-traumatization.

RECOMMENDATIONS

1. Assessment of the response to the acute intervention should include an evaluation for the following risk factors:
    a. Persistent or worsening traumatic stress symptoms (e.g., dissociation, panic, autonomic arousal, cognitive impairment)
    b. Significant functional impairments (e.g., role/work, relationships)
    c. Dangerousness (suicidal or violent ideation, plan, and/or intent)
    d. Severe psychiatric co-morbidity (e.g., psychotic spectrum disorder, substance use disorder or abuse)
    e. Maladaptive coping strategies (e.g., pattern of impulsivity, social withdrawal, or other reactions under stress)
    f. New or evolving psychosocial stressors
    g. Poor social supports.
2. Follow-up after acute intervention to determine patient status should include the following:
    a. Patient does not improve or status worsens – continue management of PTSD (See Module B) in consultation or referral to PTSD specialty care or mental health provider. Recommend involvement of the primary care provider in the treatment. Patients with multiple problems may benefit from a multi-disciplinary approach to include

occupational therapy, spiritual counseling, recreation therapy, social work, psychology, and/or psychiatry.

b. Patient demonstrates partial improvement (e.g., less arousal, but no improvement in sleep) – consider augmentation or adjustment of the acute intervention and follow up within 2 weeks.

c. Patient recovers from acute symptoms – provide education about acute stress reaction and contact information with instructions for available follow-up if needed.

DISCUSSION

After initiating an acute intervention, it is crucial for providers to follow-up and assess for treatment response and for any new or additional risk factors. Studies of exposed populations show that poor social supports and severe stress after the trauma may increase the risk of developing PTSD. Persons with stress reactions may respond with maladaptive coping styles or health risk behaviors; so, an assessment of coping styles and health risk behaviors is warranted. Those patients who respond well to acute interventions can then be offered contact information for follow-up should they later become symptomatic.

4. FOLLOW-UP

N. Persistent (>1 Month) or Worsening Symptoms, Significant Functional Impairment, or High Risk for Development of PTSD.

OBJECTIVE

Identify patients with PTSD or high risk for developing PTSD who may benefit from PTSD treatment.

BACKGROUND

A crucial goal of follow-up activities is referral, as necessary, for appropriate mental health services. In fact, referral, and subsequent delivery of more intensive interventions, will depend upon adequate implementation of screening. Screening, whether conducted in formal or informal ways, can best help determine who is in need of referral. But even if those who might benefit from mental health services are adequately identified, factors such as embarrassment, fear of stigmatization, practical barriers (e.g., distance from services), and cultural norms that do not support help-seeking may all limit motivation to seek help or pursue a referral. Those making referrals can directly discuss these attitudes about seeking help and attempt to preempt avoidance of needed services. Motivational interviewing techniques (Rollnick et al., 1992) may help increase rates of referral acceptance.

RECOMMENDATIONS

1. Individuals who fail to respond to early interventions should be referred for PTSD treatment when they have:
    a. Worsening of stress-related symptoms
    b. High potential or new-onset potential for dangerousness
    c. Development of ASD/PTSD
    d. Maladaptive coping with stress (e.g., social withdrawal, alcohol use)
    e. Exacerbation of pre-existing psychiatric conditions
    f. Deterioration in function

g. New onset stressors
h. Poor social supports.

2. Primary Care provider should consider initiating therapy pending referral or if the patient is reluctant or unable to obtain specialty services.
3. Primary Care provider should continue evaluating and treating co-morbid physical illnesses and addressing any other health concerns, as well as educating and validating the patient regarding his/her illness.

DISCUSSION

Not all individuals who are exposed to trauma or who have an Acute Stress Reaction (ASR) following trauma require a mental health referral. However, patients who are deteriorating or not responding to acute supportive interventions need to be identified and referred to mental health. Also, those patients who have a high potential for dangerousness or potential for the development of PTSD also need to be identified and referred to specialty care.

Some patients with an ASR who show partial improvement may benefit from augmentation of the acute intervention and additional follow-up. Because people recover from traumatic stress-related problems at different rates, some individuals may require more time or an adjustment of the treatment prior to improvement. For example, early in treatment, medications may be adjusted to target prominent symptoms.

Patients with partial PTSD exhibit clinically meaningful levels of functional impairment in association with their symptoms (Stein, 1997). Functional impairment, rates of co-morbid disorders, and rates of suicidal ideation were shown to increase linearly with an increasing number of PTSD symptoms, and individuals with sub-threshold PTSD had increased suicidal ideation, even after controlling for the presence of co-morbid major depressive disorder (Marshall, 2001).

Patients who do not respond to first-line interventions may warrant treatment augmentation or a mental health referral. Clear indications for a mental health referral include: a worsening of stress-related symptoms, new onset of dangerousness or maladaptive coping to stress, exacerbation of co-morbid psychiatric conditions, or deterioration in function. Because patients with new-onset stressors, poor social supports, or inadequate coping skills may be at heightened risk to develop PTSD, mental health referral is also indicated.

Primary Care providers who identify patients with possible PTSD should consider referral to a Mental Health or PTSD clinic. This referral should be made in consultation with the patient, and with consideration of the patient's severity of problems and preferences.

Several treatment modalities can be initiated and monitored in the primary care setting (e.g., Pharmacotherapy, Supportive Counseling). Therefore, the Primary Care practitioner should consider initiating therapy pending referral. However, if the patient is reluctant or unable to obtain specialty services (see Module B), the Primary Care provider should continue evaluating and treating co-morbid somatic illnesses and addressing any other health concerns, as well as educating and validating the patient regarding his/her illness. If patients are referred to specialty care, it is vital that the Primary Care team (including the Healthcare Integrator) stay actively involved in coordination with the Specialist in the care of patients with PTSD.

Additional Points:

- Don't suggest or insinuate that physical or cognitive symptoms co-existing with ASD/PTSD are related to a "stress," "emotional," or "psychological" problem. Educate patients about the physiological dysregulation associated with PTSD and how this can impact physical and cognitive functioning
- Encourage referral to behavioral health specialty care via collaborative discussion, if indicated
- Primary Care providers should not hesitate to ask questions about trauma-related symptoms Providers should be aware that narration of traumatic experiences may be associated with increased distress temporarily, and allow time to address it.

O. Monitor and Follow-Up

BACKGROUND

Many trauma survivors experience some symptoms in the immediate aftermath of a traumatic event. In most instances, these symptoms will eventually remit and do not require long-term follow-up. Those exposed to traumatic events and who manifest no or few symptoms after a period of time (approximately two months) do not require routine follow-up, but follow-up should be provided if requested.

RECOMMENDATIONS

1. Follow-up should be offered to individuals who request it or to those at high risk of developing adjustment difficulties following exposure to major incidents and disasters, including individuals who:
    a. Have acute stress disorder or other clinically significant symptoms stemming from the trauma
    b. Are bereaved
    c. Have a pre-existing psychiatric disorder
    d. Require medical or surgical attention
    e. Were exposed to a major incident or disaster that was particularly intense and of long duration.
2. Primary Care providers should follow-up with patients about issues related to trauma in an ongoing way. Patients with initial sub-threshold presentation are at increased risk of developing PTSD and may need symptom-specific management.

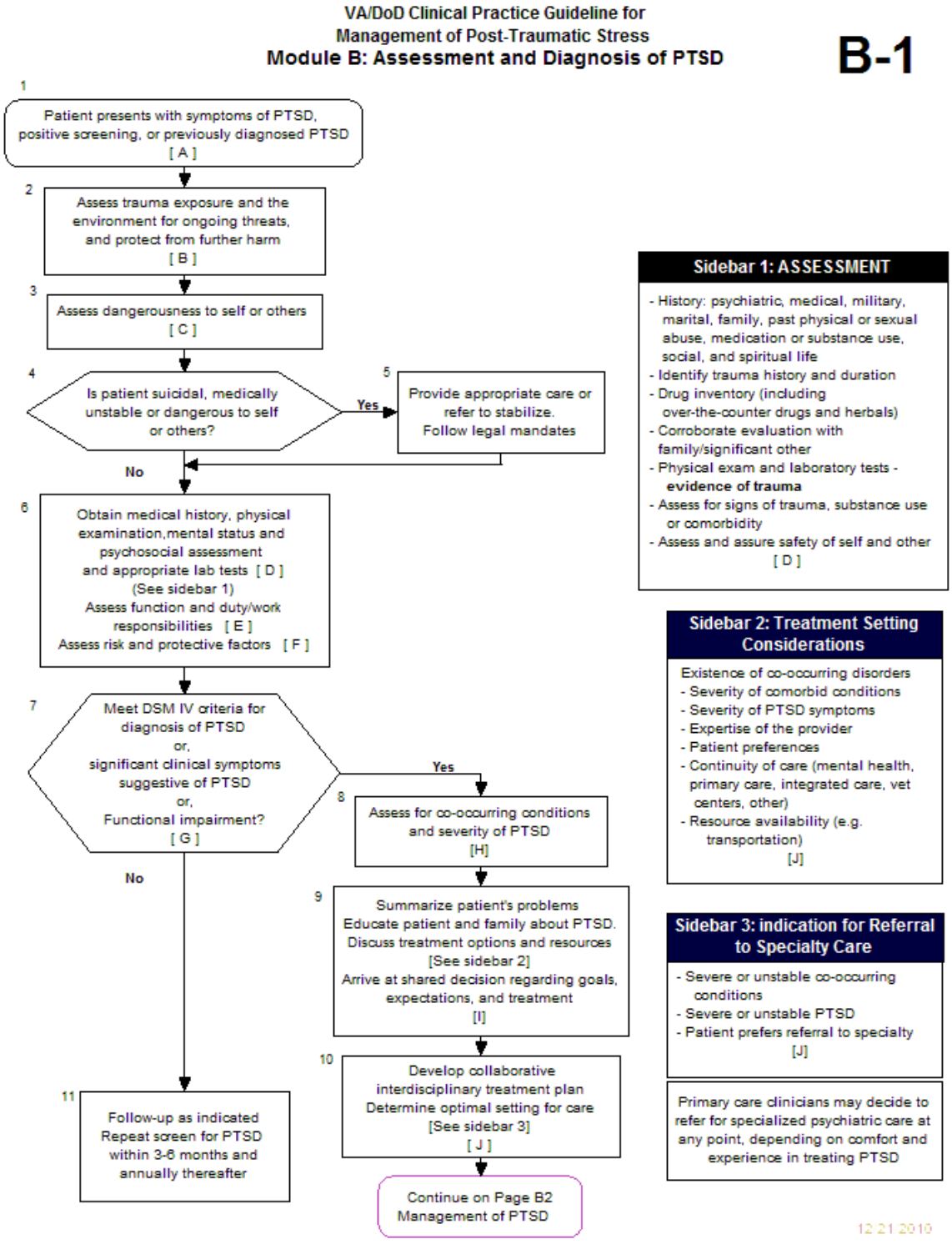
DISCUSSION

For many types of trauma, experience indicates that relatively few survivors make use of available mental health services. This may be due to a lack of awareness of the availability of such services, low perceived need for them, lack of confidence in their utility or negative attitudes toward mental healthcare. Therefore, those planning follow-up and outreach services for survivors must consider how to reach trauma survivors to educate them about sources of help and market their services to the intended recipients (Excerpted from Raphael, 2000).

In the chaos of some kinds of traumatic events (e.g., natural disaster), it is important that workers systematically obtain detailed contact information to facilitate later follow-up and outreach. In addition, it is important that those providing outreach and follow-up efforts be opportunistic in accessing settings where survivors

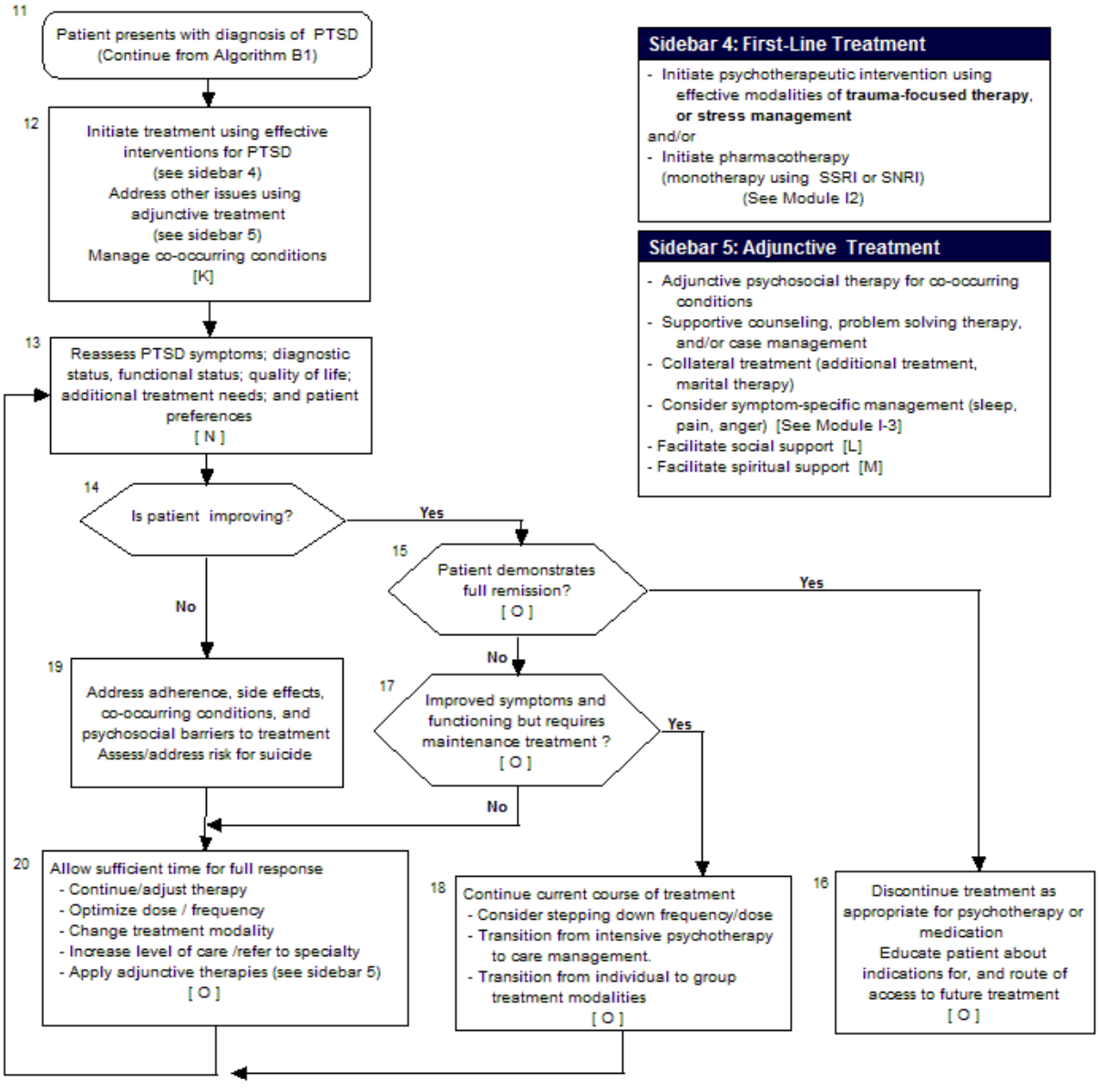
are congregating. Each contact with the system of formal and informal services available to survivors affords an opportunity to screen for risk and impairment and intervene appropriately. Settings providing opportunities for contact with survivors are diverse (e.g., remembrance ceremonies, self-help group activities, settings where legal and financial services are delivered, interactions with insurance companies). For survivors injured or made ill during the traumatic event, follow-up medical appointments represent opportunities for reassessment, referral, and treatment.

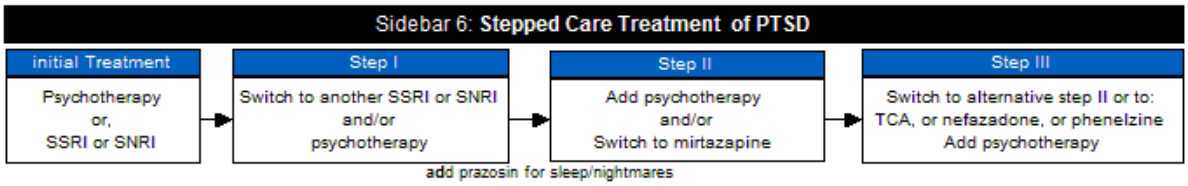
MODULE B: ALGORITHM



VA/DoD Clinical Practice Guideline for Management of Post-Traumatic Stress
Module B: PTSD Treatment

B-2





MODULE B: ANNOTATIONS

1. ASSESSMENT

A. Assessment of Stress Related Symptoms

BACKGROUND

Post-traumatic stress disorder (PTSD) is the development of characteristic and persistent symptoms, along with difficulty functioning after exposure to a life-threatening experience or to an event that either involves a threat to life or serious injury. Symptoms of PTSD may diminish with the passage of time, or they may persist for many years. PTSD often occurs together with, or precedes other psychiatric illnesses. Patients are most likely to present to primary care with unexplained somatic and/or psychological symptoms (e.g., sleep disturbance, night sweats, fatigue, and difficulty with memory or concentration). The common symptoms after exposure to trauma are included in Table B-1.

The symptoms required for the diagnosis of PTSD may be divided into 3 clusters and should be present for at least 1 month.

- **Intrusion or re-experiencing** - memories of the trauma or "flashbacks" that occur unexpectedly; these may include nightmares, intrusive mental images or extreme emotional distress, and/or physiological reactivity on exposure to reminders of the traumatic event.
- **Avoidance** - avoiding people, places, thoughts, or activities that bring back memories of the trauma; this may involve feeling numb or emotionless, withdrawing from family and friends, or "self-medicating" by abusing alcohol or other drugs.
- **Hyperarousal** - feeling "on guard" or irritable, having sleep problems, having difficulty concentrating, feeling overly alert and being easily startled, or having sudden outbursts of anger.

PTSD is frequently under-recognized and therefore often goes untreated. In a general survey in Israel, 9 percent of patients in a primary care setting were found to have PTSD. Only 2 percent of the sample was recognized as having the disorder. Despite this lack of recognition, more than 80 percent of men and 92 percent of women with PTSD in this survey reported significant distress from the disorder. Even individuals with "subthreshold" symptoms who do not meet full diagnostic criteria for the disorder suffer from significant impairments, including increased suicidal ideation.

In the case that this syndrome originates in war experiences, the presumed cause presents itself as an exceptional event overcoming the individual's resources. The notion of war traumatization has been extended to other events, such as catastrophes, physical attacks, rapes, child and wife battering, and sexual abuse. However, the events that cause PTSD are significantly more numerous. For example, it can be seen that medical events, such as giving birth, miscarriage, heart attack, cancer, or hospitalization following resuscitation may give rise to PTSD. Further, people experiencing prolonged periods of distress may equally develop a post-traumatic syndrome without any one particular event having occurred to surpass their defenses.

In some cases, providers may initially consider PTSD and use this guideline first, whereas in others it may be useful to follow the algorithms and recommendation of the DoD/VA guideline for Post Deployment Health, the VA/DoD guideline for medically unexplained symptoms or the VA/DoD guideline for Major Depressive Disorder (MDD). All these guidelines provide a link to this module when appropriate.

RECOMMENDATIONS

1. Patients who are presumed to have symptoms of PTSD or who are positive for PTSD on the initial screening should receive a thorough assessment of their symptoms that includes details such as time of onset, frequency, course, severity, level of distress, functional impairment, and other relevant information to guide accurate diagnosis and appropriate clinical decision-making.
2. Consider use of a validated, self-administered checklist to ensure systematic, standardized, and efficient review of the patient's symptoms and history of trauma exposure. Routine ongoing use of these checklists may allow assessment of treatment response and patient progress (see Appendix C: PCL-C).
3. Diagnosis of PTSD should be obtained based on a comprehensive clinical interview that assesses all the symptoms that characterize PTSD. Structured diagnostic interviews, such as the Clinician-Administered PTSD scale (CAPS), may be considered.

DISCUSSION

Initial screening is discussed in the CORE module (See Core Module Annotation C, and Appendix C: Screening Tools).

Table B - 1 Common Symptoms following Exposure to Trauma

Physical	Cognitive / Mental	Emotional	Behavioral
• Chills • Difficulty breathing • Dizziness • Elevated blood pressure • Fainting • Fatigue • Grinding teeth • Headaches • Muscle tremors • Nausea • Pain • Profuse sweating • Rapid heart rate • Twitches • Weakness	• Blaming someone • Change in alertness • Confusion • Hyper-vigilance • Increased or decreased awareness of surroundings • Intrusive images • Memory problems • Nightmares • Poor abstract thinking • Poor attention • Poor concentration • Poor decision-making • Poor problem solving	• Agitation • Anxiety • Apprehension • Denial • Depression • Emotional shock • Fear • Feeling overwhelmed • Grief • Guilt • Inappropriate emotional response • Irritability • Loss of emotional control	• Increased alcohol consumption • Antisocial acts • Change in activity • Change in communication • Change in sexual functioning • Change in speech pattern • Emotional outbursts • Inability to rest • Change in appetite • Pacing • Startle reflex intensified • Suspiciousness • Social withdrawal

B. Assessment of Trauma Exposure

BACKGROUND

Assessment should include a careful examination of the traumatic experience itself, including the nature of the event and the patient's involvement in it; the patient's emotional, physical, and behavior responses at time of traumatization; and thoughts and feelings about those responses (e.g., what he or she did or did not do).

RECOMMENDATIONS

1. Assessment of the trauma exposure experience should include:
    a. History of exposure to traumatic event(s)
    b. Nature of the trauma
    c. Severity of the trauma
    d. Duration and frequency of the trauma
    e. Age at time of trauma
    f. Patient's reactions during and immediately following trauma exposure (e.g., helplessness, horror, and fear)
    g. Existence of multiple traumas.
2. If trauma exposure is recent (<1 month), particular attention should be given to the following:
    a. Exposure to/Environment of trauma
    b. Ongoing traumatic event exposure
    c. Exposure, perhaps ongoing, to environmental toxins
    d. Ongoing perceived threat.
3. When assessing trauma exposure, the clinician must consider the patient's ability to tolerate the recounting of traumatic material, since it may increase distress and/or exacerbate PTSD symptoms.

DISCUSSION

The history also should include an assessment of prior stressful life events; coping skills; ego resources and self-capacities; environmental and social resources; cognitive functioning; psychiatric history; medical, family, social, and occupational history; and cultural and religious background. This background is necessary to establish an appropriate treatment plan specific to the individual patient. For example, if the individual does not feel safe in his or her current living situation, issues concerning safety need to be addressed first. Or, if the individual has a history of childhood abuse and has learned to use dissociation to protect the self, treatment will need to focus on helping the trauma victim manage his or her tendency to dissociate under stress. Assessment of cognitive ability may be important after trauma exposure because the patient's cognitive status could influence the course of psychotherapy, the specific psychotherapeutic technique recommended to the patient, or the provision of group versus individual psychotherapy. The repeatedly traumatized individual may also need to work through earlier childhood traumas as well as the more recent traumatic event.

C. Assessment of Dangerousness to Self or Others

BACKGROUND

It is crucial to assess for safety and dangerousness in persons with PTSD, including current risk to self or others, as well as historical patterns of risk. Assessment of dangerousness needs to take place in a safe and secure environment and should begin with the building of rapport. In patients with thoughts of self-harm, assessment should include existence of current intent and previous suicidal ideation, intent, or history of a suicide attempt.

RECOMMENDATION

1. All patients with PTSD should be assessed for safety and dangerousness, including current risk to self or others, as well as historical patterns of risk:
    a. Suicidal or homicidal ideation, intent (plan), means (e.g., weapon, excess medications), history (e.g. violence or suicide attempts), behaviors (e.g., aggression, impulsivity), co-morbidities (substance abuse, medical conditions) [B]
    b. Family and social environment – including domestic or family violence, risks to the family [B]
    c. Ongoing health risks or risk-taking behavior [B]
    d. Medical/psychiatric co-morbidities or unstable medical conditions [B]
    e. Potential to jeopardize mission in an operational environment. [I]

DISCUSSION

Any history of suicidal attempts or a family history of a completed or attempted suicide should be taken seriously. Pay careful attention to patients with behaviors that may signal dangerousness (e.g., agitation, threatening, intimidation, paranoia). Access to weapons or other means of harm should also be taken seriously. Assess for domestic or family violence, because these are elevated in those with PTSD. Assessment of medical, psychiatric, and social/environmental risks is also warranted.

Assessment of dangerousness can include questions, such as:

- You sound like you've had a very difficult time recently. Has life ever seemed like it's not worth living?
- Have you ever thought about acting on those feelings? Have you thought of how you would do this?
- Sometimes, when people get really upset or angry, they feel like doing harm to other people. Have you had any thoughts recently about harming others?
- How do you express your feelings?
- Are there times you are afraid to go home?

Dangerousness to Self

Suicidality - Persons with PTSD, including sub-threshold PTSD, are at high risk for suicidal ideation (Marshall et al., 2001) and, for women, suicide attempts (Breslau, 2000; Ferrada-Noli et al., 1998; Kaslow et al., 2000; Prigerson & Slimack, 1999).

Suicidal behavior is best assessed with the following criteria: presence of active depression or psychosis, presence of substance abuse, past history of suicidal acts, formulation of plan, a stated intent to carry out the plan, feeling that the world would

be better off if the patient were dead, availability of means for suicide (e.g., firearms and pills), disruption of an important personal relationship, and failure at an important personal endeavor. The presence of these factors often constitutes a psychiatric emergency and must always be taken seriously. Among young adults, aggressive symptoms may be predictive of suicidality in men and elevated symptoms of PTSD and/or depression may be more predictive in women (Prigerson & Slimack, 1999). Other predictors of completed suicide in general include history of suicide attempts, family history of suicide, access to weapons, male gender, and Caucasian race. Rates of suicidal ideation in treatment-seeking Vietnam veterans have been 70 to 80 percent (Kramer et al., 1994). Additionally, Vietnam veterans with diagnosed PTSD have an increased risk of death due to suicide as compared to those without PTSD (Bullman & Kang, 1994). Among veterans with PTSD, intensive combat-related guilt has been linked to suicidality (Hendin & Haas, 1991). These findings point to the need for greater clinical attention to the role of guilt in the evaluation and treatment of suicidal veterans with PTSD.

Individuals with severe childhood trauma (e.g., sexual abuse) may present with complex PTSD symptoms and parasuicidal behaviors, (e.g., self mutilation, medication overdoses) (Roth et al., 1997). Further, limited cognitive coping styles in PTSD have been linked to a heightened suicide risk (Amir et al., 1999). Fostering competence and social support may reduce this risk (Kotler et al., 2001). Co-morbid substance use disorders may increase the risk of suicidality. Additionally, persons with PTSD may also be at personal risk of danger through ongoing or future victimization in relationships (e.g. domestic violence/battering, or rape).

- Many war veterans suffer from post-traumatic stress disorder (PTSD), depression, or both disorders (Tanielian, 2008 RAND). The majority of US soldiers in Iraq were exposed to some kind of traumatic, combat-related situations, such as being attacked or ambushed (92 percent), seeing dead bodies (94.5 percent), being shot at (95 percent), and/or knowing someone who was seriously injured or killed (86.5 percent) (Hoge, 2004).
- In a nationally representative sample (N = 5877; age 15-54) that compared the relationship between anxiety disorders and suicidal ideation or suicide attempts, PTSD was significantly associated with suicidal ideation (adjusted odds ratio = 2.79; $p < 0.01$) and suicide attempts (adjusted odds ratio = 2.67; $p < 0.01$). None of the other anxiety disorders was significantly associated with suicidal ideation or attempts (Sareen, 2005).
- Older and younger veterans are more prone to suicide than are middle-aged veterans (Zivin, 2007). Veterans with PTSD have been reported to have high levels of suicidal ideation and behaviors (Oquendo, 2005).
- Jakupcak (2009) found PTSD to be a risk factor for suicidal ideation in Iraq and Afghanistan War veteran. Veterans from OEF/OIF who screened positive for PTSD were more than 4 times as likely to endorse suicidal ideation relative to non-PTSD veterans. Among veterans who screened positive for PTSD (n = 202), the risk for suicidal ideation was 5.7 times greater in veterans who screened positive for two or more co-morbid disorders relative to veterans with PTSD only.
- Patients with co-occurring disorders, such as depression and alcohol abuse or depression and posttraumatic stress disorder (PTSD), have been reported to be at much higher risk for suicide than patients with only 1 of these disorders.
- Male veterans with schizophrenia or schizoaffective disorder and co-morbid PTSD were reported to have higher rates of suicidal ideation and suicidal behaviors compared to those without co-morbid PTSD (Strauss, 2006).

- In a large, nationally representative, longitudinal data set of depressed veterans whose causes of death have been definitively identified using linked National Death Index data, veterans who received a PTSD diagnosis had a lower rate of suicide than did veterans without PTSD (68.16 vs 90.66, respectively). The suicide rate was higher in the South than in the Northeast (88.93 vs 73.55, respectively) or central regions (88.93 vs 83.09, respectively) but slightly lower than rates in the West (88.93 vs 90.04, respectively). Veterans with a service-connected disability had a lower rate of suicide than those without a service-connected disability (70.06 vs 92.20) (Zivin, 2007).

Dangerousness to Others

Some individuals with PTSD may be at risk for violence toward others (Swanson et al., 2002). Explosivity, anger problems, and past history of violence are associated with an increased risk for violent behavior. Violence often emerges as a response to a perceived threat or marked frustration by the patient stemming from his or her inability to meet goals by nonviolent means. The specific factors that contribute to violent behavior may include psychiatric, medical, environmental and situational/social engagements. Often, it is a combination of these factors that precipitates and aggravates the potential for violence, which may quickly escalate to agitation or the carrying out of violent impulses. Whatever the cause, the following situations may serve as warning signs pointing toward a very real threat of violence:

- Ideation and/or intent to harm others
- Past history of violent behaviors
- Severely agitated, aggressive, threatening, or hostile behaviors
- Actively psychotic presentation.

Clinicians should keep in mind the possibility that thoughts or plans of violent acts toward others may represent thoughts of suicide, either after committing violence against another person, or by creating a situation where another person will be forced to harm the patient (e.g. 'suicide by cop'). Special attention to the risk of domestic violence is warranted. Careful attention to the home environment and relationships is essential. If there are children, an assessment of parenting skills, anger management, caregiver burden, and discipline style is crucial. Advising high-risk patients and their families on gun removal and safe storage practices has been recommended to decrease the risk of violence (Seng, 2002). PTSD is a predictor of violence in persons with severe mental illness (Swanson et al., 2002). Also, substance use disorders are highly co-morbid in PTSD and can also predict violence. Immediate attention and intervention may be required in order to ward off the potential for escalation of agitation or violent impulses.

Health Risks

Persons with PTSD may have high rates of health risk behaviors, health problems, and medical conditions. Thus, an assessment of health and behavioral risks in individuals with PTSD is warranted. In addition to alcohol and drug use, persons with PTSD are at high risk for cigarette smoking (Acierno et al., 1996) and obesity (Vieweg et al., 2006). PTSD is a predictor of several HIV-risk behaviors as well as a risk factor for related blood-borne infections, such as hepatitis B and C (Hutton et al., 2001). Other potentially dangerous co-morbid medical conditions are intoxication or withdrawal syndromes requiring medical detoxification (e.g., alcohol, benzodiazepine, barbiturates, and possibly opiates). Medical conditions that can present a danger to others include the risk of transmission of blood-borne

pathogens, such as HIV and HCV/HBV; thus, risk assessment and serotesting are warranted.

Medical Conditions

Urgent conditions - Any condition immediately threatening to life, limb, or eyesight or requiring emergency medical care requires immediate attention.

Chronic diseases - PTSD has also been linked to cardiovascular disease, anemia, arthritis, asthma, back pain, diabetes, eczema, kidney disease, lung disease, ulcers, chronic pain, work absenteeism, and other generalized health problems (Weisberg et al., 2002; Hoge et al., 2007). One explanation for these problems may relate to the association of PTSD with dysregulation of the neuroendocrine, autonomic, nervous, and immune system functions (Schnurr and Green, 2003; Gill et al., 2009). Patients who have PTSD and other chronic medical diseases may find that PTSD worsens their medical conditions. Some medical conditions, which can be acutely dangerous in the presence of PTSD, include bronchial asthma, peptic ulcer disease, GI bleed, and malignant hypertension (Davidson et al., 1991).

Psychiatric Conditions

Delirium - (also known as organic brain syndrome, organic psychosis, acute confusional state, acute brain syndrome, and various other names) is a disorder of cognition and consciousness with abrupt onset that is frequently overlooked. This is common in the elderly and medically ill (Farrell & Ganzini, 1995).

Acute or marked psychosis - "Psychosis" in and of itself is not a psychiatric disorder. Rather, psychosis is a symptom, which may present in a variety of conditions. Psychotic patients have an impaired sense of reality, which may manifest in several forms (hallucinations, delusions, mental confusion or disorganization). Acute psychosis represents a medical emergency.

Severe debilitating depression (e.g., catatonia, malnourishment, severe disability) - While many mild to moderate illnesses may not necessarily present situations mandating immediate attention, the presence of severe depressive symptoms may represent a medical emergency, even in the absence of suicidal ideation.

EVIDENCE

	Recommendation	Sources	LE	QE	R
1	Assess for dangerousness including suicidal or homicidal ideation, intent, means, history, behaviors, and co-morbidities	Breslau, 2000 Bullman & Kang, 1994 Ferrada-Noli et al., 1998 Kaslow et al., 2000 Marshall et al., 2001 Prigerson & Slimack, 1999 Swanson et al., 2002 Zivin, 2007	III II-2 III II-2 II II II II-2	Good	B
2	Assess family and social environment – including risks for family	Seng, 2002 Swanson, 2002	III II	Good	B
3	Assess ongoing health risks or risk-taking behaviors	Acierno et al., 1996 Hutton et al., 2001 Vieweg et al., 2006	II-2 II II-2	Good	B

4	Assess medical or psychiatric co-morbidities or unstable medical condition	Davidson et al., 1991 Farrell et al., 1995 Weisberg et al., 2002 Hoge et al., 2007 Gill et al., 2009	II III III III III	Good	B
5	In operational environment, consider the potential to jeopardize the mission	Working Group Consensus	III	Poor	I

LE = Level of Evidence; QE = Quality of Evidence; SR= Recommendation (see Appendix A)

D. Obtain Medical History, Physical Examination, Laboratory Tests and Psychosocial Assessment

OBJECTIVE

Obtain comprehensive patient data in order to reach a working diagnosis.

BACKGROUND

A wide range of medical conditions and treatments may result in abnormal behavior, and many medical disorders may produce or exacerbate psychiatric symptoms in patients with pre-existing mental illness. Multiple studies indicate high rates of medical disease (24 to 50 percent) in patients presenting with psychiatric symptoms (Williams & Shepherd, 2000). Failure to detect and diagnose underlying medical disorders may result in significant and unnecessary morbidity and mortality (Lagomasino et al., 1999). The converse problem is far greater in primary care: patients present with somatic symptoms and have psychiatric disorders that have not been properly diagnosed or treated. In one study, 5 of 6 patients with a psychiatric diagnosis had a somatic presentation, and the primary care physician made the diagnosis only half the time, whereas for the 16 percent with a psychological complaint, the correct diagnosis was made 94 percent of the time (Bridges et al., 1985). A standardized approach to medical evaluation, including a thorough history, physical examination, laboratory evaluation, and occasionally other ancillary testing, prevents the omission of important aspects of the evaluation (Williams & Shepherd, 2000).

RECOMMENDATIONS

1. All patients should have a thorough assessment of medical and psychiatric history, with particular attention paid to the following:
    a. Baseline functional status
    b. Baseline mental status
    c. Medical history: to include any injury (e.g., mild-TBI)
    d. Medications: to include medication allergies and sensitivities; prescription medications; herbal or nutritional supplements; and over-the- counter (OTC) medications (caffeine, energy drinks or use of other substances)
    e. Past psychiatric history: to include prior treatment for mental health and substance use disorder, and past hospitalization for depression or suicidality
    f. Current life stressors.
2. All patients should have a thorough physical examination. On physical examination, particular attention should be paid to the neurological exam and

stigmata of physical/sexual abuse, self-mutilation, or medical illness. Note distress caused by, or avoidance of, diagnostic tests/examination procedures.

3. All patients, particularly the elderly, should have a Mental Status Examination (MSE) to include assessment of the following:
    a. Appearance and behavior
    b. Language/speech
    c. Thought process (loose associations, ruminations, obsessions) and content (delusions, illusions and hallucinations)
    d. Mood (subjective)
    e. Affect (to include intensity, range, and appropriateness to situation and ideation)
    f. Level of Consciousness (LOC)
    g. Cognitive function
    h. All patients should have routine laboratory tests as clinically indicated, such as TSH, Complete Metabolic Panel, Hepatitis, HIV, and HCG (for females). Also consider CBC, UA, Tox/EtoH panel, and other tests
    i. Other assessments may be considered (radiology studies, ECG, and EEG), as clinically indicated
    j. All patients should have a narrative summary of psychosocial assessments to include work/school, family, relationships, housing, legal, financial, unit/community involvement, and recreation, as clinically appropriate.

DISCUSSION

Differential diagnosis is important, given the many co-morbidities associated with PTSD, including dementia, depression, substance abuse/withdrawal, bereavement, psychosis, bipolar disorder, seizure disorder, persistent post-concussion syndrome, thyroid disease, neoplasm, somatoform-spectrum disorders (including irritable bowel, chronic fatigue, headaches, and non-cardiac chest pain), anxiety disorders, toxicosis, rheumatoid-collagen vascular disease, hypoxia, sleep apnea, closed head injury, CHF, and delirium.

Medical and Psychiatric History

The medical history may be obtained from the patient, family, friends, or coworkers or from official accounts of a traumatic event.

- Substance use and misuse can cause, be caused by, and/or exacerbate PTSD. Use of screening tools (such as the AUDIT, MAST, or DAST) can improve detection of substance use disorders (see the VA/DoD Guideline for Substance Use Disorder)
- The active ingredients of OTC/herbal supplements can create pharmacokinetic and pharmacodynamic interactions with prescribed medications or medications that might be prescribed for treatment of PTSD (Shord et al., 2009; Ulbricht et al., 2008). For example, the serotonin syndrome, present with substantial anxiety symptoms, is the result of the interaction between SSRI or SNRI medications and another serotonergic substance such as St John's Wort or

dextromethorphan (common ingredient in cough syrup); or prescription medications such as tramadol (a prescription-only analgesic), or methadone. An additional concern involves energy drinks. These drinks contain caffeine in modest to excessive amounts (Reissig et al., 2009) that may exacerbate anxiety symptoms, or, indicate a deficit in attention that should be pursued further for an underlying co-morbid etiology such as Attention-Deficit Hyperactivity Disorder.

- Risk factors suggesting the need for a higher-than-usual index of suspicion include certain physiological and psychological conditions or life events that may contribute to the development or exacerbation of PTSD symptoms (see Annotation F).

Physical Examination

A brief screening physical examination may uncover endocrine, cardiac, cerebrovascular, or neurologic disease that may be exacerbating or causing symptoms. Particular attention should be given to a neurological examination and stigmata of physical/sexual abuse, self-mutilation, or medical illness. Special note should also be made of distress caused by, or avoidance of, diagnostic tests or examination procedures, since these reactions may be suggestive of prior physical or sexual abuse. Careful attention should also be given to complying with legal mandates for documentation, reporting, and collection of evidence.

Mental Status Examination (MSE)

Particularly in the elderly patient, a full Mental Status Examination (MSE) including a cognitive screening assessment should occur. The assessment may consist of using a standardized instrument, such as the Folstein Mini-Mental State Examination (MMSE) (Crum et al., 1993; Cummings, 1993; Folstein et al., 1975). Typically scores below 24 on the Mini Mental State Exam (MMSE) are suggestive of cognitive impairment however, some older adults do not score well on the MMSE (there some tasks patients have to perform with their hands; thus those who have full or partial paralysis or even bad arthritis have a hard time doing those tasks and lose at least 2-3 points). If screening is suggestive of cognitive impairment and the patient is not delirious, then a laboratory evaluation to assess for reversible causes of dementia is appropriate. However, the PTSD assessment should be continued. If delirium is present, consider it an emergency and stabilize the patient before continuing with the PTSD assessment.

Level of Consciousness (LOC) should be assessed to rule out delirium. Abnormal tics or movements should be noted, as well as dysarthria, dysprosody, aphasia, agraphia, and alexia, which may suggest underlying neurological disease. Sensory illusions may be seen in neurological syndromes and intoxications (Lagomasino et al., 1999).

Consider seeking further evaluation and consultation from neuropsychology specialty in cases of suspected cognitive disorders.

Laboratory Evaluation

The history and physical examination findings should be used to direct a conservative laboratory evaluation. There is no test for PTSD, but PTSD is frequently co-morbid with substance use disorders, depression, and high-risk behaviors. Therefore, testing is directed toward detection of associated medical conditions and ruling out any contraindications to medical therapy. Appropriate laboratory studies include: TSH, Complete Metabolic Panel, Hepatitis, HIV, and HCG (for females). Also consider CBC, UA, Tox/EtoH Panel, and other tests, as clinically indicated.

Other Evaluation

- Diagnostic imaging and neuropsychological testing are not a part of the standard evaluation for PTSD. Proceed with management while awaiting the completion of the laboratory evaluation
- MRI/CT of the head may be indicated to rule out mass lesions, intracranial bleeding, hydrocephalus, or subdural hematomas (Lagomasino et al., 1999)
- ECG may rule out underlying cardiac abnormalities that preclude the use of medications, such as tricyclic antidepressants (Lagomasino et al., 1999)
- Consider EEG or other diagnostic testing, as indicated by history and physical exam.

Psychosocial Assessment

- Past psychiatric illness, treatment, or admission
- Past/ongoing problems with anxiety, impulsivity, mood changes, intense/unstable interpersonal relationships, suicidality, and hallucinations
- Recreational use of drugs/alcohol/tobacco/caffeine
- Social supports (family, friends, homelessness/housing, community, and financial status)
- Losses (bereavement, friend/family member injuries/death, occupation, and moral injury/betrayal)
- Occupational/educational/military history
- Environmental resources
- Coping Skills
- Factors affecting expression and intensity of PTS symptoms
- Legal issues
- Religious/spiritual history.

Consider use of checklists to determine if psychosocial rehabilitation services are indicated in PTSD treatment (see Module I-2: D. Psychosocial Rehabilitation Intervention).

E. Assessment of Function, Duty/Work Responsibilities and Patient's Fitness (In Relation To Military Operations)

BACKGROUND

One of the key goals of care is to assist the individual in beginning to return to a normal level of functioning. The clinician must assess the individual's current level of family, relationship, work/school, and social functioning.

Ideally, service members who become ineffective as a result of PTSD will be returned to duty at the earliest possible time. For most military specialties, the time required to enlist and train the soldier to minimal operational readiness often exceeds a year. Consequently, service members who become ineffective due to stress-related conditions constitute a significant source of trained personnel who potentially have much to offer despite their disability. Assessment of fitness for duty may also have implications for medical boards and vocational rehabilitation.

RECOMMENDATION

1. Assessment of function should be obtained through a comprehensive narrative assessment (see Table B-2), and the use of standardized, targeted, and validated instruments designed to assess family/relationship, work/school, and/or social functioning.
2. The determination of when to return to work/duty should take into consideration the complexity and importance of the patient's job role and functional capabilities.
3. The continuing presence of symptoms of PTSD should not be considered in itself as sufficient justification for preventing a return to work/duty.

DISCUSSION

Global Functional Assessment

Consider using instruments, such as the GAF (American Psychiatric Association, 1994) or the SF-36 (McHorney, 1994), to assess function. Such measures are useful for directing therapeutic interventions and monitoring response to treatment. The GAF score, while readily available and familiar to mental health professionals, is a poor predictor of function among combat veterans with PTSD. The GAF score explained only 17 percent of the variability in the scores among these combat veterans (Miller et al., 2008). No single test (GAF, SF-36, PCL, or many others) can replace a careful and thoughtful clinical assessment when the clinician is tasked with determining level of function.

Narrative Functional Assessment

Functional assessment must be considered from the patient's point of view as well as from the clinician's point of view. A narrative account provides a more complete picture of the patient and his/her response to trauma. It allows for targeted social and behavioral interventions. Components of functional assessment should include: work/school, relationships, housing, legal, financial, unit/community involvement, and recreation.

Duty/Work Responsibilities

Practitioners who are managing patients suffering from stress reactions or PTSD should consider a variety of factors when deciding if, and when, the individual is ready to return to work or military duty, including severity of the condition, level of occupational impairment, nature of the occupation, and the level of social support.

Table B - 2 Components of Functional Assessment

Work	• Is the person unemployed or seeking employment? • If employed, any changes in productivity? • Have co-workers or supervisors commented on any recent changes in appearance, quality of work, or relationships? • Tardiness, loss of motivation, loss of interest? • Been more forgetful, easily distracted?
School	• Changes in grades? • Changes in relationships with friends? • Recent onset or increase in acting out behaviors? • Recent increase in disciplinary actions? • Increased social withdrawal? • Difficulties with concentration and short-term memory?
Marital & Family Relationships	• Negative changes in relationship with significant others? • Irritable or easily angered by family members? • Withdrawal of interest in or time spent with family? • Any violence within the family? • Parenting difficulties? • Sexual function difficulties?
Recreation	• Changes in recreational interests? • Decreased activity level? • Poor motivation to care for self? • Sudden decrease in physical activity? • Anhedonia?
Housing	• Does the person have adequate housing? • Are there appropriate utilities and services (electricity, plumbing, other necessities of daily life)? • Is the housing situation stable?
Legal	• Are there outstanding warrants, restraining orders, or disciplinary actions? • Is the person regularly engaging in or at risk to be involved in illegal activity? • Is patient on probation or parole? • Is there family advocacy/Dept. of Social Services (DSS) involvement?
Financial	• Does the patient have the funds for current necessities, including food, clothing, and shelter? • Is there a stable source of income? • Are there significant outstanding or past-due debts, alimony, child support? • Has the patient filed for bankruptcy? • Does the patient have access to healthcare and/or insurance?
Unit/Community Involvement	• Does the patient need to be put on profile, MEB, or limited duty? • Is patient functional and contributing in the unit environment? • Is there active/satisfying involvement in a community group or organization?

F. Assessment of Risk/Protective Factors

BACKGROUND

Following a traumatic event, a majority of those exposed may experience acute-traumatic stress reaction. Of the population of persons who experience a traumatic event, only a subset will ultimately develop PTSD. After 9 to 12 months, 15 to 25 percent continue to be disturbed by these symptoms. This group with persistent symptoms may have a distinct combination of characteristics that determine the presence of ongoing problems. The presence of, and interplay among, three groups of risk factors—biological factors (including genetics), the nature of the trauma, and the recovery environment (psychological and social support) work together to contribute to an individual's vulnerability or resilience to PTSD.

RECOMMENDATIONS

1. Patients should be assessed for risk factors for developing PTSD. Special attention should be given to post-traumatic factors (i.e., social support, ongoing stressors, and functional incapacity) that may be modified by intervention.
2. When evaluating risk factors for PTSD, the clinician should keep in mind that PTSD is defined as occurring only after four weeks have elapsed following a traumatic event. PTSD symptoms, however, may not appear until a considerable time has passed—sometimes surfacing years later.

DISCUSSION

Risk Factors for PTSD

Two major systematic reviews of predictors of PTSD have been published (Brewin et al., 2000; Ozer et al., 2003). The main outcome measure considered in the reviews was effect size calculated for the different factors. Effect sizes give an indication of the magnitude of the associations found.

The meta-analysis of risk factors for PTSD of assessed studies of trauma-exposed adults reported that 14 different risk factors in the literature have a modest association with PTSD development (Brewin et al., 2000). The review by Ozer et al. (2003) focused on personal characteristics salient for psychological processing and functioning and aspects of the traumatic event or its sequelae. Dissociation during the trauma, perceived support, and perceived life threat were strongly associated with PTSD. Prior trauma and prior (in early childhood or in adult life) adjustment factors were identified among the pre-trauma factors. Prior trauma was more strongly related to PTSD when the traumatic experience involved non-combat interpersonal violence than when the traumatic experience resulted from combat or an accident. Perceived life threat was more associated when assessment was further away from the traumatic event and in non-combat interpersonal violence than in accidents. Perceived social support was also more significant in studies that assessed individuals further away from the time of the traumatic event. Family history of psychiatric disorders was more significant among survivors of non-combat interpersonal violence than when the traumatic experience was combat exposure.

The following characteristics have been reported in studies to be risk factors for the development of PTSD:

Pre-traumatic factors

- Ongoing life stress or demographics
- Lack of social support
- Young age at time of trauma
- Pre-existing psychiatric disorder
- Female gender
- Low socioeconomic status, lower level of education, lower level of intelligence, race (African-American, American Indian, and Pacific Islander)
- Prior trauma exposure (reported abuse in childhood, report of other previous traumatization, report of other adverse childhood factors)
- Family history of psychiatric disorders (genetics).

Peri-traumatic or trauma-related factors

- Severe trauma
- Type of trauma (interpersonal traumas, such as torture, rape, or assault, convey a high risk of PTSD)
- High perceived threat to life
- Community (mass) trauma
- Peri-traumatic dissociation.

Post-traumatic factors

- Ongoing life stress
- Lack of positive social support
- Negative social support (e.g., negative reactions from others)
- Bereavement
- Major loss of resources
- Other post-traumatic factors, including children at home and distressed spouse.

Overall, factors, such as gender, age at trauma, and race, predicted PTSD in some populations but not in others. Further, factors, such as education, prior trauma, and childhood adversity, predicted PTSD more consistently (Harvey & Bryant, 2000; Harvey & Bryant, 1998b). However, this varies with the population and study methods. Prior psychiatric history, childhood abuse, and family psychiatric history have more consistent predictive effects. Factors operating during or after the trauma (e.g., trauma severity, lack of social support, and additional life stress) have somewhat stronger effects than pre-trauma factors. This finding is consistent with other studies that suggest poor social support and ongoing life stress to be predictors of PTSD development. This may have clinical implications, as early interventions that increase social support after trauma exposure may reduce the likelihood of PTSD (Litz et al., 2002).

The development of Acute Stress Disorder (ASD) at the time of the trauma is also a risk for developing PTSD (Classen et al., 1998). Numerous prospective cohort studies with various types of trauma exposure (e.g., violent assault and accidents) support that ASD is a predictor of later PTSD (Brewin et al., 1999; Bryant et al., 2000;

Harvey & Bryant, 1999; Mellman et al., 2001). In these studies, among persons with ASD, 40 to 80 percent did develop PTSD. Finally, most studies suggest an increased risk of PTSD development among individuals with peri-traumatic dissociation (Birmes et al., 2001; Murray et al., 2002). Subsequent research indicates that it is the post-traumatic duration of dissociation, rather than the peri-traumatic occurrence of dissociation (Panasetis & Bryant, 2003), that predicts the development of PTSD.

Pre-Traumatic Factors

Prior exposure to traumatic events is a risk indicator for chronic PTSD (Brewin et al., 2000; Ozer et al., 2003). In particular, a history of exposure to interpersonal violence, in childhood or adulthood, substantially increases the risk for chronic PTSD following exposure to any type of traumatic event (Breslau, 2002; Brewin et al., 2000; Ozer et al., 2003). Green et al. (2000) surveyed 1909 college-aged women and found that those who had experienced interpersonal trauma and those who had experienced multiple traumas exhibited elevated symptoms. Dougall et al. (2000) hypothesized that prior trauma history sensitizes victims to the new stressor, thus potentiating its impact. They argued that evaluating trauma history is essential for improving early intervention efforts.

Epidemiological studies have yielded higher rates of PTSD *in women* than in men in general populations, and there are also a number of gender differences in clinical presentation after trauma. Seedat and Stein (2000) studied a series of patients presenting with physical trauma after interpersonal violence and found that "women were more likely than men to have been previously assaulted or to have sustained injury by a relative or someone known to them, but less likely to have used substances at the time of the assault or to require emergency surgery." Although there is considerable evidence suggesting a gender difference in PTSD prevalence, it is unclear whether this difference may be related to a higher risk of traumas that result in increased risk (e.g., rape) or greater willingness to seek mental healthcare for PTSD among women. One analysis in military personnel suggested that women and men who are working in support units with similar level of combat exposure appear to have an equivalent risk of developing PTSD (Hoge et al., 2007), and further research is needed. Numerous epidemiological studies utilizing representative samples that have examined the prevalence of traumatic exposure and rates of PTSD across the adult lifespan found that younger adults had the highest prevalence of traumatic events and PTSD, followed by middle-aged adults and then older adults (Creamer & Parslow, 2008; De Vries & Olff, 2009; Kessler et al., 2005; Spitzer et al., 2008).

Pre-existing psychiatric problems are associated with more adverse responses to trauma (Norris et al., 2002; Breslau, 2002), as shown in a review of epidemiological studies that found that preexisting psychiatric disorder was one of 3 factors that had a predictable effect on the development of PTSD. Two meta-analyses of risk or predictive factors for PTSD have identified prior psychiatric history as a risk factor for the development of PTSD (Brewin et al., 2000; Ozer et al., 2003). A f*amily history* of psychiatric disorders may also contribute to a person's vulnerability to PTSD. Brewin and colleagues (2000) found that "factors, such as psychiatric history, reported *childhood abuse*, and family psychiatric history … had more uniform predictive effects" than did other risk factors, such as gender or age at trauma.

Genetics – Family history of any psychiatric disorder or possible genetic differences in regulating pre-synaptic uptake of serotonin (or other neurobiological mechanism) can increase risk. Genetic research has shown that of the two variants of the gene regulating pre-synaptic uptake of serotonin, the long form appears to be associated

with resilience and the short form with the vulnerability to stress events. Individuals who inherited the short form and were exposed to four of more stressful life events were much more likely to develop PTSD and depression and to attempt suicide (Koenen et al., 2009). Other genes that may confer vulnerability or resilience are currently under investigation. Twin studies have also indicated that there is a genetic vulnerability to PTSD. Twin research to date suggests that exposure to assaultive trauma is moderately heritable, whereas exposure to non-assaultive trauma is not. PTSD symptoms are moderately heritable, and co-morbidity of PTSD with other disorders may be partly due to shared genetic and environmental influences (Koenen, 2008; Afifi, 2010).

Peri-Traumatic Factors

Foy et al. (1984) published one of the first formal studies to look at risk factors for PTSD and reported characteristics of trauma exposure to be of central importance. Numerous studies have since observed a dose-response relationship between trauma severity and PTSD. The more severe the trauma, the more likely the person experiencing it will develop PTSD. Armenian and colleagues (2000) found this to be true among disaster victims. Feehan et al. (2001) found higher PTSD rates among more severely traumatized members of a general cohort.

With regards to type of trauma, interpersonal violence (rape, torture, physical assault) was found to be more likely to produce PTSD than more impersonal events (such as accidents or group trauma) (Holbrook et al., 2001).

Situations where the trauma is potentially life-threatening also carry a high risk of PTSD; in a meta-analysis of 68 PTSD studies, Ozer et al. (2003) found “perceived life threat” to have a high risk value, and in Woods’ study of abused women, the perceived threat of homicide played a role in the later development of PTSD. Holbrook et al. (2001) diagnosed 261 (32 percent) of 824 individuals as having PTSD 6 months after major physical trauma. Patients who were totally incapacitated, experienced physical injury, or suffered major losses were also at higher risk for developing PTSD. Factors associated with a PTSD diagnosis included perceived threat to life, female gender, younger age, and lower income.

Ozer et al. (2003) also found that dissociation at the time of the trauma is predictive of later development of PTSD. Demographic factors may also be predictive. Finnsdottir & Elklit (2002) found higher rates of PTSD among disaster victims who were young at the time of the trauma. In a general group of psychiatric patients, Neria et al. (2002) found young age at trauma to be a risk factor for PTSD. Finally, biological factors may also be relevant to predicting PTSD. Shalev et al. (1998) measured the heart rate and blood pressure of eighty-six trauma survivors at the time of their presentation at a hospital emergency room and concluded that “elevated heart rate shortly after trauma is associated with the later development of PTSD.” In a meta-analysis, Yehuda et al. (1998) reported that studies “demonstrated increased heart rate and lower cortisol levels at the time of the traumatic event in those who have PTSD at a follow-up time compared to those who do not.”

Post-Traumatic Factors

The post-trauma environment has been shown to be an important predictor of chronicity (Berwin, 2000). The experience of traumatization may have life-altering consequences in terms of social status, employment, and health, and continuing difficulties in these areas may contribute to the likelihood that a person will develop PTSD. Feehan et al. (2001), in interviews with 374 trauma survivors, found

unemployment to be a risk factor. Likewise, in a meta-analysis performed by Norris et al. (2002), "resource loss" was cited as a risk for PTSD.

Impaired social support is a not-infrequent outcome of a traumatic experience. Armenian et al. (2000), Brewin et al. (2000), Gregurek et al. (2001), and Ozer et al. (2003) all reported that the loss of support from significant others can pose a risk for development of PTSD.

Finally, general ongoing life stress may also play a role. Brewin et al. (2000) reported "life stress" to be more predictive of PTSD development than pre-traumatic factors, such as gender or age at trauma. Norris et al. (2002) found that in disaster victims, "secondary stressors" increased the likelihood of adverse outcomes.

Some have suggested that secondary gain related to compensation may predict treatment outcome. Laffaye et al. (2008), in a comprehensive review of the literature, found that initial levels of perceived support and stressors did not predict the course of chronic PTSD symptoms. Furthermore, the literature indicates that veterans who are seeking, or have been awarded, compensation participate in treatment at similar or higher rates than do their non-compensation-seeking counterparts. Veteran treatment outcome studies produced either null or mixed findings, with no consistent evidence that compensation-seeking predicts worse outcomes. Studies of motor vehicle accident survivors found no association between compensation status and course of recovery (Laffaye, 2007).

Risk Factors for PTSD in Military Veterans

Friedman et al. (1994) concluded that "the likelihood of developing chronic PTSD depends on premilitary and postmilitary factors in addition to features of the trauma itself. Premilitary factors include negative environmental factors in childhood, economic deprivation, family psychiatric history, age of entry into the military, premilitary educational attainment, and personality characteristics. Postmilitary factors include social support and the veteran's coping skills. Among military personnel, there are three populations at risk for unique problems that may amplify the psychological impact of war-zone stress. They are women whose war-zone experiences may be complicated by sexual assault and harassment; nonwhite ethnic minority individuals whose premilitary, postmilitary, and military experience is affected by the many manifestations of racism, and those with war-related physical disabilities, whose PTSD and medical problems often exacerbate each other."

Among military service members, combat exposures are reported as the strongest predictors of subsequent PTSD (Berwin, 2000; Clancy 2006; Foy, 1987; Baker 1997; Smith 2008). The frequency and intensity of direct combat appears to be one of the strongest predictors of PTSD. A number of studies have found a strong dose response relationship of combat frequency and intensity to PTSD prevalence (e.g. Hoge, et al., 2004; Dohrenwend et al., 2006; MHAT6 report (2009); and the Smith, Ryan et al., 2008). Wartime exposure includes numerous combat events such as being wounded, losing a team member, near miss of life witnessing, torture, witnessing killing, or killing enemy or civilian in combat (Maguen et al., 2010).

Studies of veterans have reported gender differences in PTSD risks: war zone stressors appear preeminent for PTSD in men, and post-trauma resilience-recovery variables are more important for women (King et al., 1999).

There are good arguments favoring a genetic contribution to the PTSD diagnosis in combat veterans. The Vietnam Twin Registry studies found the effect size of combat exposure was 5 to 12 times smaller than the effect size of zygosity upon PTSD

diagnosis (Gilbertson et al., 2006). Kremen et al. (2007) reported a similar relationship by examining cognitive ability and rates of PTSD diagnosis. These data from the Vietnam Twin Registry involve small N due to the sample nature, but they do offer excellent support for a genetic contribution to PTSD diagnosis.

Clancy et al. (2006) examined the effect of exposure before, during, or after military service. Findings indicated that non-military-related trauma was prevalent among the veterans sample (90 percent). Regression analyses for PTSD symptom severity revealed that age, greater combat exposure, and a history of physical assault after military service were significantly associated with more severe PTSD symptoms. Childhood physical abuse, adult sexual trauma, and a history of being physically assaulted during military service were also significantly associated with PTSD symptom severity.

Injury severity was a significant predictor of any mental health diagnosis and PTSD diagnosis. Gunshot wounds and diastolic blood pressure were significant predictors of any mental health diagnosis but not PTSD. A study of a sample of 1968 men (831 battle injuries and 1137 non-battle injuries) injured during Operation Iraqi Freedom (OIF) found that those with battle injuries compared with non-battle injuries had a greater risk of PTSD and other mental health diagnoses, and there was a positive association with injury severity (MacGregor et al., 2009). Aggressive pain control after injury has shown, in one study, to reduce the incidence of PTSD. The study (Holbrook, 2010) found that the use of morphine during trauma care may reduce the risk of subsequent development of PTSD after serious injury.

One semi-prospective study (Zohar, 2009) examined risk factors for the development of post-traumatic stress disorder following combat trauma by comparing a large sample of war veterans (Israeli Defense Force) who developed PTSD with a matched control group of veterans who did not. Neither behavioral assessment nor training was found to predict PTSD. The predictive factors that were found were essentially non-specific, such as cognitive functioning, education, rank, and position during the trauma, with little effect from training. The author concluded that "… an armed force that uses universal recruitment, carefully structured predrafting psychological assessment of social and individual qualifications (including motivation) failed to identify increased risk factors for PTSD. However, nonspecific factors were found to be associated with an increased risk for PTSD. This study suggests that the focus of future research on risk factors for PTSD should incorporate other domains rather than behavioral assessment alone" (Zohar et al., 2009).

Phillips et al. (2010) identified risk factors for PTSD among military service members as related to their combat exposure. The threat of death and serious injury and the witnessing of injury or death are significant risk factors for screening positive for post-deployment PTSD among male Marines, as well as violence exposures prior to entering the Marine Corps, which are independent of future combat exposures. Prior assault was also found to increase vulnerability, rather than resilience against, PTSD symptoms among military professionals in the Millennium Cohort Study of US military cohort deployed in the wars in Iraq and Afghanistan (Smith, 2008). Higher frequency and intensity of combat has been strongly associated with increased rates of PTSD (Hoge, 2004; Dohrenwend, 2006).

PTSD symptoms among service members deployed to Iraq or Afghanistan have been associated with lower rank, being unmarried, less formal education, and a history of childhood adversity (Smith, 2008; Iversen, 2008).

The intrapersonal characteristic of hardiness as well as post-war social support may be protective against developing PTSD. In contrast, negative life events in the postwar or trauma period are linked to PTSD (King et al., 1998).

There is evidence that a strong social support network, indicated by unit cohesion, is protective. A large social support network may diminish the association between stressful life events and PTSD symptoms (Schnurr et al., 2004; Benotsch et al., 2000; Brailey et al., 2007).

EVIDENCE

	Evidence	Sources	LE	QE	SR
1	Assessment of persons exposed to trauma for risk factors for developing PTSD (pre-trauma and post-trauma risks)	Brewin et al., 2000 Ozer et al., 2003	II	Good	B
2	Assessment of trauma type, nature, and severity	Brewin et al., 1999 Bryant et al., 2000 Harvey & Bryant, 2000 Mellman et al., 2001	II	Good	B
3	Assessment of existing social supports and ongoing stressors.	Litz et al., 2002	II	Good	B
4	Patients with dissociative symptoms or ASD warrant careful clinical attention due to a high risk for developing PTSD	Birmes et al., 2001 Brewin et al., 1999 Bryant et al., 2000 Harvey & Bryant, 2000 Mellman et al., 2001 Murray et al., 2002	II	Good	B

LE = Level of Evidence; QE = Quality of Evidence; SR = Strength of Recommendation (see Appendix A)

2. TRIAGE

G. Diagnosis of PTSD or Clinical Significant Symptoms Suggestive of PTSD?

BACKGROUND

In the primary care setting, providers often do not have the time or resources to accomplish a detailed mental health intake evaluation; so it is important for them to be comfortable with the initial evaluation and management of stress-related disorders without having to be concerned with the fine details of DSM-IV and making a definite diagnosis. Providers who perform the initial evaluation of a patient with suspected PTSD should recognize that a detailed recounting of the traumatic experience may cause further distress to the patient.

Please refer to Annotation A for a discussion of post-traumatic symptoms.

RECOMMENDATION

1. A diagnosis of stress-related disorder consistent with the DSM IV criteria for PTSD should be formulated before initiating treatment.
2. Diagnosis of PTSD should be obtained based on a comprehensive clinical interview that assesses all the symptoms that characterize PTSD. Structured diagnostic interviews, such as the Clinician-Administered PTSD scale (CAPS), may be considered.
3. When a diagnostic work out cannot be completed, primary care providers should consider initiating treatment or referral based on a working diagnosis of stress-related disorder.
4. Patients with difficult or complicated presentation of the psychiatric component should be referred to PTSD specialty care for diagnosis and treatment.
5. Patients with partial or sub-threshold PTSD should be carefully monitored for deterioration of symptoms.

DISCUSSION

Approximately 90 percent of patients with a mental health diagnosis are seen in primary care (Gebhart, 1996).

Many options are available to primary care providers to treat stress-related disorders and to relieve the burden of suffering for PTSD patients including pharmacotherapy, supportive counseling, and referral. Because these interventions can be helpful in a variety of psychiatric disorders, it is not essential that a detailed diagnostic assessment be completed prior to initiating treatment for PTSD.

In addition, a detailed recounting of the traumatic experience may cause further distress to the patient and is not advisable unless a provider has been trained and is able to support the patient through this experience.

Table B - 3 Diagnostic criteria for Post-Traumatic Stress Disorder (DSM-IV)

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 re-experienced 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

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

DSM-IV & DSM-IV-TR Cautionary Statement

- The specified diagnostic criteria for each mental disorder are offered as guidelines for making diagnoses, because it has been demonstrated that the use of such criteria enhances agreement among clinicians and investigators. The proper use of these criteria requires specialized clinical training that provides both a body of knowledge and clinical skills.
- These diagnostic criteria and the DSM-IV Classification of mental disorders reflect a consensus of current formulations of evolving knowledge in our field. They do not encompass, however, all the conditions for which people may be treated or that may be appropriate topics for research efforts.
- The purpose of DSM-IV is to provide clear descriptions of diagnostic categories in order to enable clinicians and investigators to diagnose, communicate about, study, and treat people with various mental disorders. It is to be understood that inclusion here, for clinical and research purposes, of a diagnostic category, such as Pathological Gambling or Pedophilia, does not imply that the condition meets legal or other non-medical criteria for what constitutes mental disease, mental disorder, or mental disability. The clinical and scientific considerations involved in categorization of these conditions as mental disorders may not be wholly relevant to legal judgments, for example, that take into account such issues as individual responsibility, disability determination, and competency.
- Dissociative symptoms are not considered an essential feature of PTSD, as they are for ASD. Dissociative symptoms included among the diagnostic criteria for PTSD are categorized as reexperiencing (e.g., dissociative flashbacks) or avoidance /numbing (e.g., dissociative amnesia and psychic numbing). For example, the dissociative symptom of psychic numbing which is an expression of a restricted range of affect among the avoidance/numbing symptoms of PTSD. Similarly, the inability to remember an important aspect of the trauma describes the dissociative symptom of amnesia. Thus, while dissociation has not been identified as a central feature of PTSD, dissociative symptoms can contribute to a diagnosis of PTSD, making the comparison of ASD and PTSD less inconsistent than it might seem.

Partial or Sub-threshold PTSD

Studies in which the prevalence of partial or sub-threshold PTSD was examined found it to be substantial. In one study of infantry soldiers returning from Iraq, the prevalence of PTSD was estimated to be 12 percent when a stringent PCL definition of PTSD was utilized but rose to 18-20 percent when a more liberal DSM symptom-based definition was applied (Hoge, 2004). A large Canadian epidemiological study assessing for current PTSD found the incidence to be 5.0 percent (women) and 1.7 percent (men), but the incidence of partial PTSD was even higher at 5.7 percent and 2.2 percent for women and men, respectively. Individuals with sub-threshold PTSD showed similar levels of social and occupational impairment as those meeting full criteria (Stein, 1997; Marshall, 2001).

H. Assess for Co-Occurring Disorders

OBJECTIVE

Improve management of PTSD symptoms when they are complicated by the presence of a medical or psychiatric co-morbidity.

BACKGROUND

Co-morbid medical and psychiatric conditions are important to recognize, because they can modify clinical determinations of prognosis, patient or provider treatment priorities, selection of interventions, and the setting where PTSD care will be provided. Patients with PTSD have been found to frequently report physical symptoms, cognitive health concerns, and utilize high levels of medical care services. Providers should also expect that 50 to 80 percent of patients with PTSD will have one or more coexisting mental health disorders. PTSD is strongly associated, among veterans from recent deployment (OEF/OIF), with generalized physical and cognitive health symptoms attributed to concussion/mild traumatic brain injury (mTBI).

Because of the many potential etiologies of these symptoms, it is generally best to develop a collaborative care treatment strategy based in primary care and address these health concerns simultaneously with PTSD symptoms (see VA/DoD Clinical Practice Guideline for Post-Deployment Health). Management should focus on identifying and treating the symptoms that are causing the most impairment, regardless of the cause or diagnosis.

Some co-morbid medical or psychiatric conditions may require early specialist consultation in order to assist in determining treatment priorities. In some cases, these disorders may require stabilization before (or in concert with) initiation of PTSD treatment.

RECOMMENDATIONS

1. Providers should recognize that medical disorders/symptoms, mental health disorders, and psychosocial problems commonly coexist with PTSD and should screen for them during the evaluation and treatment of PTSD.
2. Because of the high prevalence of psychiatric co-morbidities in the PTSD population, screening for depression and other psychiatric disorders is warranted (see also VA/DoD Clinical Practice Guidelines for the Management of Major Depressive Disorder [MDD] and for Bipolar Disorder).
3. Patterns of current and past use of substance by persons with trauma histories or PTSD should be routinely assessed to identify substance misuse or dependency (alcohol, nicotine, prescribed drugs, and illicit drugs) (see also VA/DoD Clinical Practice Guideline for Substance Use Disorders).
4. Pain (acute and chronic) and sleep disturbances should be assessed in all patients with PTSD.
5. Generalized physical and cognitive health symptoms - also attributed to concussion/mild traumatic brain injury (mTBI) and many other causes - should be assessed and managed in patients with PTSD and co-occurring diagnosis of mTBI (see also VA/DoD CPG for Concussion/mild-TBI, and the CPG for Post-Deployment Health).
6. Associated high-risk behaviors (e.g., smoking, alcohol/drug abuse, unsafe weapon storage, dangerous driving, HIV and hepatitis risks) should be assessed in patients with PTSD.

7. Providers should consider the existence of co-morbid conditions when deciding whether to treat patients in the primary care setting or refer them for specialty mental healthcare (See Annotation J).
8. Patients with complicated co-morbidity may be referred to mental health or PTSD specialty care for evaluation and diagnosis (see Annotation J).

DISCUSSION

Co-morbid conditions and psychosocial problems of significant importance to treatment planning include:

***Medical Conditions**:* PTSD is associated with elevated rates of generalized physical and cognitive health concerns, which are thought to be mediated in part by neuroendocrine dysregulation and autonomic nervous system reactivity (Hoge et al., 2007; Schnurr & Green, 2004). These health conditions can include chronic headaches, chronic musculoskeletal pain, memory and attention problems, fatigue, dizziness, gastrointestinal symptoms, sleep dysfunction, hypertension, rapid heart rate (sometimes in association with panic symptoms), cardiovascular disease, impulsivity, anger, sexual problems, and a variety of other health complaints. The trauma-focused techniques may be undesirable and counter-productive for older adults as they can lead to increased autonomic arousal and decreased cognitive performance. In patients with serious cardiac problems, consultation from the primary care physicians can be sought. If in consultation with other health professionals, and the patient, it is decided that trauma-focused treatments is feasible, then mental health treatment providers can proceed with caution and closely monitor patients at greater risk from high arousal. These health concerns can sometimes cluster together and may present as multisystem problems in the same manner as somatoform-spectrum or medically unexplained physical symptom (MUPS) conditions. These symptoms have been commonly described after all wars, overlap with numerous conditions, and often have more than one potential etiology (see DoD/VA Post-Deployment Health CPG). For example, service members or veterans who present to primary care with headaches, cognitive problems, fatigue, dizziness, and/or irritability may be experiencing these symptoms as a result of chronic sleep deprivation, neuroendocrine/autonomic nervous system dysregulation associated with PTSD, residual effects of injuries during deployment (including concussions/mTBIs), chronic pain, medication side effects, depression, substance misuse, or other causes. For veterans of combat, their experiences may have involved the extremes of physiological stress, contributing to long-term dysregulation of neuroendocrine and autonomic nervous systems.

It is important for clinicians to be aware of the high medical co-morbidity of PTSD and the fact that physical health concerns (e.g., chronic pain, headaches) may make it more difficult to treat PTSD symptoms. Because of the many potential etiologies of these symptoms, it is generally best to develop a collaborative care treatment strategy based in primary care and address these health concerns simultaneously with PTSD symptoms, or the VA post-deployment care clinic model, an integrated primary care – mental health clinical setting centered on the combat veteran (see also the VA/ DoD Guideline for Post-Deployment Health).

Some medical disorders may restrict PTSD treatment options (e.g., dementia limits psychotherapeutic options; cardiac conduction problems may limit some pharmacotherapeutic options; and disorders that restrict mobility may limit ability to attend weekly treatment sessions). It is generally best to maximize medical management of these conditions first and then focus on PTSD treatment.

Substance Use Disorders*:* Patients with PTSD frequently use alcohol, nicotine, and other substances in maladaptive ways to cope with their symptoms. Approximately 40 to 50 percent of PTSD patients treated in the VA have current substance use problems. Effective PTSD treatment is extremely difficult in the face of active substance use problems, unless substance use disorders are also treated. Most often, attempts to address substance problems should proceed concurrently with the direct management of PTSD. However, in cases when the substance use is severe, substance use may require initial treatment and stabilization before progressing to PTSD care (e.g., patient requires detoxification from opiates) (see Annotation J2 - Concurrent PTSD and Substance Abuse). Ongoing heavy alcohol use will interfere with prolonged exposure therapy by chemically enhancing the extinction of anxiety, thus not allowing the patient an opportunity to fully engage in therapy.

Mild-Traumatic Brain Injury (mTBI): Providers should have specific awareness of traumatic brain injury (TBI), particularly concussion/mTBI, in the post-deployment population because of the high incidence of concussion/mTBI during deployment (10-20 percent of combat veterans), and high co-morbidity of post-concussive symptoms (PCS) with PTSD. Concussion/mTBI is common in sports injuries, motor vehicle accidents, military training (e.g., hand-to-hand combat), and combat. It is associated with a variety of symptoms that will manifest immediately following the event, and may resolve quickly, within minutes to hours after the injury event. In certain individuals the symptoms persist longer leading to a persistent post-concussion symptoms (PPCS). Although there has been controversy of the relative contribution of concussion/mTBI and PTSD to post-deployment health outcomes, what is not controversial, is that PPCS include many of the same symptoms that veterans report after combat service, and overlap with the physical and cognitive health problems associated with PTSD, depression, and other causes (Bryant, 2008; Hoge, 2009; Stein & Mcallister, 2009). Several studies in OIF/OEF veterans have shown that PTSD is associated with post-deployment cognitive impairment, headaches, and other post-concussive symptoms. A history of concussion/mTBI with loss of consciousness has also been shown to have a small independent association with some post-deployment outcomes (Schneiderman et al., 2008; Hoge et al., 2008; Marx et al., 2009; Pietrzak et al., 2009). These studies highlight the complex interrelationship of causal factors responsible for post-deployment symptoms, and supports collaborative care approaches to treatment.

It is often difficult to precisely attribute symptoms to concussive events that occurred months or years earlier. Combat-related concussions (particularly those involving loss of consciousness) are associated with an increased risk of PTSD, presumably because of the life-threatening context of the concussion (distinct from concussions occurring in non-life threatening situations, such as sports accidents, which are not associated with PTSD), but possibly because of other factors (e.g., physiological, neurocognitive) inherent to the TBI, as well.

Psychiatric Disorders: In addition to substance use disorders, other commonly occurring mental disorders that co-exist with PTSD include: major depression, dysthymia, panic disorder, obsessive-compulsive disorder, and generalized anxiety disorder. Treatment of these disorders often occurs concurrently with therapy for PTSD, but on occasion they will take precedence. These disorders have evidence-based therapies that may pose additional effective treatment options. Co-morbid disorders that are less common with PTSD, but not rare, include psychotic disorders and bipolar disorder. Practitioners should be alert to co-morbid eating disorders, such as bulimia, particularly in women.

Personality Disorders: Personality disorders are long-term problems of coping that begin in childhood or adolescence and are often associated with past abuse or neglect and recurrent relationship problems. These patterns often result in poor adherence to prescribed PTSD management, and the primary care provider may require early assistance and advice from the mental healthcare provider.

The primary care practitioner should remain cautious of making a personality disorder diagnosis when PTSD is a known or suspected diagnosis. In some instances, PTSD could explain behavior attributed to a personality disorder. A mistaken personality disorder diagnosis can lead to delays in treatment for PTSD. For example, poor adherence to treatment may indicate a personality disorder, but it also may indicate a patient who was sexually assaulted on active military duty and is angry with authority figures because the assault was not appropriate investigated by the military chain of command.

Psychosocial Problems: Associated behavior problems and psychosocial deficits commonly present in patients with chronic PTSD include:

- Homelessness
- Suicidality
- Domestic violence or abuse
- Aggression, rage.

I. Educate Patient and Family

OBJECTIVE

Help trauma survivors cope with ASD/PTSD by providing information that may help them manage their symptoms and benefit from treatment.

BACKGROUND

Education of the trauma survivor is a core component of all PTSD treatment. Survivors need to better understand what they are experiencing, how to cope with reactions or symptoms, and what happens in treatment. It is also helpful to provide this information to family members or to the patients' significant others so that they can more effectively support the patient's recovery.

Education may be helpful in encouraging patients to self-refer to treatment or for family members encouraging a patient to attend treatment. Chaplains, particularly in the active duty military population, can be highly effective educational liaisons. Military culture does not attach any stigma to speaking with a chaplain although some military members may be reluctant to seek mental health assistance. Education from military chaplains may reduce barriers to care.

Caregivers (informal and formal) are often integral to treatment with older adults who are physically and mentally vulnerable/compromised. When conducting therapy with those with cognitive or physical impairments, providers may want to engage caregivers for additional support, reinforcement of materials presented in therapy, and assistance with transportation in getting to treatment.

RECOMMENDATIONS

1. Trauma survivors and their families should be educated about PTSD symptoms, other potential consequences of exposure to traumatic stress, practical ways of coping with traumatic stress symptoms, co-morbidity with other medical health concerns, processes of recovery from PTSD, and the nature of treatments. [C]

2. Providers should explain to all patients with PTSD the range of available and effective options for PTSD treatment.
3. Patient preferences along with provider recommendations should drive the selection of treatment interventions in a shared and informed decision-making process.

DISCUSSION

PTSD education involves teaching the survivor to label, recognize, and understand PTSD symptoms (and other trauma-related problems) that he or she is experiencing. Education should include discussion of the adaptive nature of many of the symptoms, which have to do with survival and the body's normal responses to threat. This is particularly important if PTSD occurred after exposure encountered in an occupational context, where the person was trained to respond to critical incidents (e.g., military, firefighter, police, and other first responders). Education should also provide simple advice regarding coping (such as sleep hygiene instruction), explain what can be done to facilitate recovery, and describe treatment options**.** Education can help make symptoms more understandable and predictable, decrease fear of symptoms, increase social support and lessen feelings of isolation, increase awareness of coping options and reduce maladaptive coping, and help survivors decide whether to seek treatment or learn how to better participate in treatment.

Education should be one of the first steps of PTSD treatment. It can help establish the credibility of the treatment provider, make treatment seem immediately helpful to the patient, and help prepare the patient for next steps in treatment. In fact, education should continue throughout PTSD treatment, sometimes in brief discussions when the patient has questions and sometimes more systematically as a formal activity. It can be delivered to individuals or to groups. Because patients with PTSD often have difficulties with concentration and memory, repetition of educational information and provision of written information are important.

The content of PTSD-related education can include the following topics:

Nature of PTSD symptoms: It is useful to help the survivor identify and label the reactions that he or she may be experiencing, recognize that emotional and physical reactions are expected after trauma, understand how the body's response to trauma includes many of the symptoms of PTSD, and understand that anxiety and distress are often "triggered" by reminders of the traumatic experience that can include sights, sounds, or smells associated with the trauma, physical sensations (e.g., heart pounding), or behaviors of other people.

Practical steps to cope with trauma-related problems: Survivors can also be educated about ways of coping with their PTSD symptoms in order to minimize their impact on functioning and quality of life. While education about coping is not a substitute for more systematic coping skills training, information on specific topics can be useful. Survivors can be helped to distinguish between positive and negative coping actions. Positive coping includes actions that help to reduce anxiety, lessen other distressing reactions and improve the situation; they include relaxation methods, physical exercise in moderation, talking to another person for support, positive distracting activities, and active participation in treatment. Negative coping methods may help to perpetuate problems and can include continual avoidance of thinking about the trauma, use of alcohol or drugs, social isolation, and aggressive or violent actions.

Nature of the recovery process and PTSD treatment: Survivors will sometimes have unrealistic or inaccurate expectations of recovery and may benefit from understanding that recovery is an ongoing daily gradual process (i.e., it doesn't happen through sudden insight or "cure") and that healing doesn't mean forgetting about the trauma or having no emotional pain when thinking about it. Education about what happens in treatment is also important. Treatment providers should explain and encourage discussion of treatment options, including evidence-based treatments. This can help build motivation to participate or persist in treatment.

Despite the fact that education is a component in all PTSD treatment and the strong clinical consensus that exists as to the importance of education, there is little empirical evidence that it reduces PTSD symptoms. Education is a component of empirically supported treatments but has not been evaluated as a "stand-alone" treatment (nor is it intended to be delivered in the absence of other treatment elements).

EVIDENCE

	Recommendation	Sources	QE	QE	R
1	Educate patients and family members regarding the trauma, its effects, ways of coping, and the treatment process.	Working Group Consensus	III	Poor	C

LE = Level of Evidence; QE = Quality of Evidence; SR= Recommendation (see Appendix A)

J. Determine Optimal Setting for Management of PTSD and Co-Occurring Disorders

J1. Management of PTSD with Co-morbidity

BACKGROUND

When PTSD has been determined to be the primary target of intervention because it is significantly impairing a patient's functioning or causing a high level of distress, the patient's preferences and motivation, the co-occurrence of other conditions, and the capacity to provide the necessary services should be considered in determining the optimal setting for treatment and long-term management. The referral to specialty care may be considered in this context.

When there are co-occurring medical or psychiatric conditions, the clinician will need to determine the best strategy for prioritizing and treating multiple disorders. In general, these disorders should be treated concurrently with PTSD treatment, although there are exceptions, such as severe substance dependence, that require medical detoxification prior to other forms of treatment. One important decision point is whether PTSD and its psychiatric co-morbidities should be treated in the primary care setting or referred to specialty mental healthcare.

REOMMENDATIONS

Consultation / Referral

1. PTSD and co-morbid mental health conditions should be treated concurrently for all conditions through an integrated treatment approach, which considers patient preferences, provider experience, severity of the conditions, and the availability of resources.

2. Patients with PTSD and severe co-morbid mental health conditions should be treated either through referral or in consultation with a provider that is experienced in treating the co-morbid conditions.
3. Because of the profound social impairment of PTSD (caused, for example, by the patient's anger and avoidance symptoms), close friends and family members in the patient's immediate daily environment (e.g., parents, spouse, or children) should be provided with education and advised to consider assistance from specialty care, both for individual treatment and couples/family treatment.
4. Factors to consider when determining the optimal setting for treatment include:
    a. Severity of the PTSD or co-occurring disorders
    b. Local availability of service options (specialized PTSD programs, evidence-based treatments, behavioral health specialty care, primary care, integrated care for co-occurring disorders, Vet Centers, other)
    c. Level of provider comfort and experience in treating psychiatric co-morbidities
    d. Patient preferences
    e. The need to maintain a coordinated continuum of care for chronic co-morbidities
    f. Availability of resources and time to offer treatment.
5. Considerations related to possible referral:

    Complicated severe PTSD: Some patients with PTSD have complicated, challenging presentations. These patients warrant referral to specialty PTSD care that includes access to cognitive-behavioral evidence-based treatments (see Module I-2: Treatment for PTSD).

    Co-occurring major depressive disorder (MDD) in the absence of significant suicidality, panic, or generalized anxiety often shows reduction in intensity when the PTSD is treated. Depression of mild severity may not require referral to specialty care or additional treatments outside those targeting PTSD. Patients should be carefully monitored for changes in symptoms. A reduction of PTSD symptoms that is not accompanied by reduction of symptoms in depression or anxiety would justify a more formally targeted treatment (refer to the VA/DoD guideline for MDD).

    Co-occurring mild to moderate disorders, such as substance use, pain disorders, and sleep problems, can frequently be effectively treated in the context of PTSD treatment and do not require a referral to specialty care. Consultation, to integrate adjunctive interventions, may be considered (see the respective VA/DoD CPGs).

    Co-occurring severe psychiatric disorders, while not precluding concurrent PTSD treatment, typically justify referral to specialty care for evaluation and treatment. These disorders may include: *Severe Major Depression or Major Depression with suicidality, Unstable Bipolar Disorder, Severe Personality Disorders, Psychotic Disorders, Significant TBI, and Severe Substance Use Disorder (SUD) or substance abuse* of such intensity that PTSD treatment components are likely to be difficult to implement.

    Persistent Post-Concussion Symptoms in patients who present with PTSD and a history of concussion/mTBI may be best managed within either primary care or polytrauma rehab settings that utilize a multidisciplinary

team approach. Providers should recognize that mTBI/concussion is one of numerous possible etiologies of co-morbid post-deployment symptoms occurring in veterans and service members with PTSD, and it is often difficult to precisely attribute symptoms to concussive events that occurred months or years earlier. From a treatment standpoint, physical or cognitive symptoms, such as headaches or memory problems, or other persistent post-concussive symptoms should be treated symptomatically whether or not concussion/mTBI is thought to be one of the causal factors. Clinicians should not get caught up in debating causation but maintain focus on identifying and treating the symptoms that are contributing to the most impairment. There is no evidence to support withholding PTSD treatments while addressing post-concussive symptoms.

DISCUSSION

A number of guiding principles should be considered in making treatment decisions with these patients:

- Integrated care models, in which the physical and mental health needs of patients are addressed in a single setting by a multidisciplinary provider team, have potential to reduce perceived stigma associated with help-seeking
- In systems where integrated care models do not exist, consultation and comprehensive assessment by a mental health provider are recommended
- In general, referral to specialty mental health is indicated if a patient with PTSD has co-morbid mental disorders that are severe or unstable. Examples include: patients whose depression is accompanied by suicidality, patients with substance dependence, and patients with concurrent psychotic or bipolar disorder. If the patient is referred to mental health for treatment of PTSD, then it is usually best for the mental health provider to provide comprehensive treatment for all mental disorders.
- For patients referred to specialty mental healthcare, it is important to preserve the continuity of care by ensuring ongoing communication with the primary care provider and to ensure coordination of care when multiple providers are involved.

J2. Management of Concurrent PTSD and Substance Use Disorder

OBJECTIVE

Improve management of PTSD symptoms when they are complicated by a concurrent substance abuse problem.

BACKGROUND

Research has documented a strong relationship between co-occurring PTSD and substance use problems in civilian and military populations of both genders (e.g., Jacobsen et al., 2001; Kessler et al., 1995). In FY 2008 almost 22% of VA patients diagnosed with PTSD also received a SUD diagnosis with rates of 70% seen in patients hospitalized for PTSD. Similarly, an extensive literature has documented high rates of PTSD among male veterans seeking SUD treatment. Patients diagnosed with both disorders tend to have poorer long-term prognoses for each condition than do those with one diagnosis without the other.

A similar relationship exists between PTSD and nicotine dependence (Fu et al., 2007; McFall et al., 2006). Smoking rates were high among clinical samples with PTSD

(40%-86%) as well as nonclinical populations with PTSD (34%-61%). Most studies showed a positive relationship between PTSD and smoking and nicotine dependence, with odds ratios ranging between 2.04 and 4.52.

In addition to the recommended PTSD services, programs should address substance use conditions that exist in association with the PTSD. Treatment services directed toward these additional SUD problems, when they exist, are associated with SUD improvement. Given improvement in PTSD symptoms, co-occurring SUD problems may show some spontaneous improvement if services are not provided, but in many instances the SUD must be addressed directly.

There is no evidence to support a preferred sequencing of treatments for diagnoses. In general, treatments for patients with both PTSD and SUD can be effectively delivered concurrently. Providers should consult the relevant CPG for SUD and PTSD individually. Clinical judgment based on systematic symptom monitoring will continue to be needed in deciding which specific treatments to implement, for which patients, and under which treatment conditions.

RECOMMENDATIONS

1. All patients diagnosed with PTSD should receive comprehensive assessment for SUD, including nicotine dependence (as recommended by the separate Clinical Practice Guideline).
2. Recommend and offer cessation treatment to patients with nicotine dependence. [A]
3. Patients with SUD and PTSD should be educated about the relationships between PTSD and substance abuse. The patient's prior treatment experience and preference should be considered since no single intervention approach for the co-morbidity has yet emerged as the treatment of choice
4. Treat other concurrent Substance Use Disorders consistent with VA/DoD clinical practice guidelines including concurrent pharmacotherapy:
    a. Addiction-focused pharmacotherapy should be discussed, considered, available and offered, if indicated, for all patients with alcohol dependence and/or opioid dependence.
    b. Once initiated, addiction-focused pharmacotherapy should be monitored for adherence and treatment response.
5. Provide multiple services in the most accessible setting to promote engagement and coordination of care for both conditions. [I]
6. Reassess response to treatment for SUD periodically and systematically, using standardized and valid self-report instrument(s) and laboratory tests. Indicators of SUD treatment response include ongoing substance use, craving, side effects of medication, emerging symptoms, etc.
7. There is insufficient evidence to recommend for or against any specific psychosocial approach to addressing PTSD that is co-morbid with SUD. [I]

DISCUSSION

Co-occurring PTSD and SUD is associated with: more severe PTSD symptoms; the higher the rates of other co-occurring Axis I and II Disorders, the higher the rates of medical problems and the greater the likelihood of relapse (Najavits, 1997: Ouimette and Brown, 2002; Brady, 2001). Rates of co-occurrence are high: Men with PTSD are

5 times more likely to have a SUD compared to the general population. Women with PTSD are 1.4x more likely (Helzer et al., 1987). Lifetime prevalence of PTSD among individuals seeking SUD treatment has been reported as high as 50%. Population based data are lower.

The literature, in general, provides support for improved SUD and PTSD symptoms when individuals are provided treatment. No systematic findings indicate harm to patients provided integrated treatment for co-occurring SUD and PTSD and there is recognition that both conditions ought to be addressed. There are findings that support provision of integrated treatment for SUD and PTSD both as an adjunct to existing SUD treatment services or as stand-alone treatments (Ouimette et al., 1998a). However, the data are limited making it difficult to clearly identify one specific treatment as the "gold standard." Studies examining both patient characteristics and clinician concerns indicate that one central feature may be the high rate of other co-occurring Axis I and Axis II psychiatric disorders among this cohort and not just SUD and PTSD alone (Cacciola et al., 2008). A key component of this seems to be the likelihood of more severe symptom presentation (e.g., history of suicide attempts, inpatient psychiatric hospitalizations).

Addiction-focused pharmacotherapy should be provided in addition to any indicated pharmacotherapy for co-existing PTSD and directly coordinated with specialty psychosocial treatment and adjunctive services for psychosocial problems as well as with the patient's primary care and/or general mental health providers.

Because withdrawal symptoms experienced during early abstinence may be associated with a resurgence of traumatic memories, worsening PTSD symptoms, and, possibly, increased risk for suicidal thoughts or attempts (Kosten & Krystal, 1988), the client should be supported closely through this period, prepared for possible short-term worsening of PTSD symptoms, and helped to develop strategies for managing symptoms and urges to drink or use.

Because patients with SUD and PTSD may be at higher risk for relapse and their relapses may be "triggered" in part by trauma reminders and cues, clinicians should adapt relapse prevention methods to help substance abuse patients identify their trauma-related relapse cues and prepare them to cope with those triggers without drinking or using.

EVIDENCE

	Recommendation	Sources	QE	QE	R
1	Routinely assess substance use patterns of clients with trauma histories or PTSD	Working Group Consensus	III	Poor	I
2	Offer addiction-focused pharmacotherapy when appropriate	Brady et al., 2005	I	Good	B
4	Educate substance-abusing patients with PTSD about the relationships between PTSD and substance abuse	Working Group Consensus	III	Poor	I
5	Consider concurrent PTSD treatment or provision of integrated PTSD/substance abuse treatment	Najavits, 2002 Ouimette et al., 1998	II-2	Mod	C
6	Follow-up care for SUD-PTSD should include a continued focus on both and monitoring	Ouimette et al., 2000	II-3	Fair	I

LE = Level of Evidence; QE = Quality of Evidence; SR= Recommendation (see Appendix A)

J3. The Role of the Primary Care Practitioner

BACKGROUND

Primary care clinicians may decide to refer for specialized psychiatric care at any point, depending on their level of comfort, experience, experience in treating PTSD, the particular needs and preferences of the patient, and the availability of other services.

RECOMMENDATIONS

1. Primary care providers should routinely provide the following services for all patients with trauma-related disorders, especially those who are reluctant to seek specialty mental healthcare:
   - Education about the disorder and importance of not letting stigma and barriers to care interfere with specialty treatment if needed
   - Provision of evidence-based treatment within the primary Care or through referral
   - Regular follow-up and monitoring of symptoms
   - Regular follow-up and monitoring of co-morbid health concerns.
2. Primary care providers should consider consultation with mental health providers for patients with PTSD who warrant a mental health referral but refuse it or seem reluctant to talk to a mental health provider.
3. Primary care providers should take leadership in providing a collaborative multi-disciplinary treatment approach. Team members may include the primary care providers, mental health specialists, other medical specialists (e.g., neurology, pain management), chaplains, pastors, social workers, occupational or recreational therapists, Vet Center staff members, staff of family support centers, exceptional family member programs, VA benefits counselors, vocational rehabilitation specialists, peer counselors, and others.
4. When an integrated behavioral health clinician is available (e.g., collaborative care model, or Post-Deployment Care clinics) evidence-based treatment should be provided.
5. Primary care providers should continue to be involved in the treatment of patients with acute or chronic stress disorders. All patients with PTSD should have a specific primary care provider assigned to coordinate their overall healthcare.

3. TREATMENT

K. Initiate Treatment Using Effective Interventions for PTSD

BACKGROUND

Many treatment strategies are available to treat stress-related disorders and to relieve the burden of suffering for PTSD patients. Options include pharmacotherapy, psychotherapy, and somatic and alternative medicine interventions. Treatment may be provided by primary care providers, specialty mental health providers, or some combination of these.

Primary care is an ideal setting in which to educate patients and their families about treatment options for post-traumatic stress. Patient education is recommended as an element of treatment of PTSD for all PTSD patients and their family members. Such educational efforts must include informing patients that even if they respond to medication therapy, treatment for a longer period may be needed. The patient's preferences along with provider recommendations should drive the selection of treatment interventions in a shared decision-making process.

Discussion of the evidence supporting the recommendations for treatment intervention is included in Module I-2: Treatment for PTSD.

RECOMMENDATIONS

1. A supportive and collaborative treatment relationship or therapeutic alliance should be developed and maintained with patients with PTSD.
2. Evidence-based psychotherapy and/or evidence-based pharmacotherapy are recommended as first-line treatment options.
3. Specialized PTSD psychotherapies may be augmented by additional problem-specific methods/services and pharmacotherapy.
4. Consider referral for alternative care modalities (Complementary Alternative Medicine) for patient symptoms, consistent with available resources and resonant with patient belief systems. [See Module I-2]
5. Patients with PTSD who are experiencing clinically significant symptoms, including chronic pain, insomnia, anxiety, should receive symptom-specific management interventions. [See Module I-3]
6. Management of PTSD or related symptoms may be initiated based on a presumptive diagnosis of PTSD. Long-term pharmacotherapy will be coordinated with other intervention.

For Specific Treatment Modalities: See Module I-2 Treatment Interventions for PTSD:

Psychotherapy

Pharmacotherapy

Adjunctive Treatments

Somatic Therapy

Complimentary Alternative Therapy (CAM)

DISCUSSION

Establishing Therapeutic Alliance

Many people with PTSD find that their relationships with others have changed as a result of exposure to trauma. They often report that they have difficulty trusting others, are suspicious of authority, dislike even minor annoyances, and generally want to be left alone. Since the clinician-patient relationship draws heavily on trust, respect, and openness, and since the relationship often has to be formed in a busy clinical or bureaucratic setting, the provider may find the PTSD patient to seem to be withholding, negativistic, or even hostile at the initial meeting. The patient may seem to have "an attitude," or "Axis II" co-morbidity. As a result, many combat veterans feel misunderstood or misdiagnosed by otherwise competent professionals, and

ultimately the patient suffers through feeling betrayed and misunderstood by the mental health professional. If a therapeutic relationship is to have any opportunity to develop, the treatment provider must adopt a stance of caring and concerned involvement that takes what the patient says at face value, doesn't judge or label this type of behavior, and doesn't withdraw into an "objective" "professional" role. In short, the clinician who can relate honestly and openly is more likely to have a patient who is willing to relate to him/her as a fellow human being and an effective partner in treatment.

A general understanding of what has happened to the veteran is critical in this process of developing a therapeutic relationship. Every provider working with combat veterans should be advised to read some basic material on the experience of combat and watch documentaries of the same. The provider must develop an understanding that wartime and military service involves some of the most intense human experiences and that those feelings of profound rage, fear, and grief can be an expected part of these experiences. These feelings will be present in the interview setting and must be met with respect and compassion. It is also helpful for the professional to be careful not to assume that they have any understanding of the military experience if they have not themselves served in the military and should not be afraid to ask questions when they don't understand something about the military that the patient is referring to.

Family, religious organizations and community leaders can be helpful when dealing with an unfamiliar culture and/or religion. It may also be appropriate to consult a local cultural adviser. But particular attention should be paid to the individual's own beliefs and values, and confidentiality always must be maintained when getting input from other sources. Patient's beliefs should be seen in the context of their social, religious, and cultural environment, and if need be, a trusted member of the person's faith or cultural group should be consulted.

PTSD Treatment

Refer to the evidence-based treatment strategies for PTSD, summarized in the section on Pharmacotherapy and Psychotherapy Intervention of this guideline. The section also includes medication tables that summarize indications/benefits, contraindications/adverse effects, and usual dosages (see Module I-2).

Supportive counseling for PTSD has received little study to date and cannot be endorsed as an evidence-based psychotherapeutic strategy. However, it has been shown to be effective compared with no treatment and may be the sole psychotherapeutic option available for the patient with PTSD who is reluctant to seek specialty mental healthcare. It may be a useful engagement strategy to provide temporary support, with the ultimate goal to convince patient to accept evidence-based treatment.

L. Facilitate Spiritual Support [*See Module I-2: D2- Spiritual Support]*

M. Facilitate Social Support [*See Module A: Annotation L2*]

4. RE-ASSESSMENT & FOLLOW-UP

N. Assess Response to Treatment

OBJECTIVE

Re-assess patient status following therapeutic intervention to determine response to treatment, inform treatment decisions, and identify need for additional services. Re-assessment should address PTSD symptoms, diagnostic status, functional status, quality of life, additional treatment needs, and patient preferences.

RECOMMENDATIONS

1. At a minimum, providers should perform a brief PTSD symptom assessment at each treatment visit. The use of a validated PTSD symptom measure, such as the PTSD Checklist, should be considered (see Appendix C).
2. Comprehensive re-assessment and evaluation of treatment progress should be conducted at least every 90 days, perhaps with greater frequency for those in active treatment, and should include a measure of PTSD symptomatology (e.g., PCL) and strongly consider a measure of Depression symptomatology (e.g., PHQ9).
3. Other specific areas of treatment focus (e.g., substance abuse) should also be reevaluated and measured by standardized measures of outcome.
4. Assessment of functional impairment should also be made, at a minimum, by asking patients to rate to what extent their symptoms make it difficult to engage in vocational, parental, spousal, familial, or other roles.
5. Consider continued assessment of:
    - Patient preferences
    - Treatment adherence
    - Adverse treatment effects.

DISCUSSION

Patients should be assessed at least every three months after initiating treatment for PTSD, in order to monitor changes in clinical status and revise the intervention plan accordingly. The interval of three months is suggested because many controlled trials of first-line therapies for PTSD demonstrate clinically significant changes during this time frame. Assessment of the following domains is advised: (a) symptom severity and diagnostic status of PTSD, co-morbid mental disorders, and co-morbid medical conditions; (b) functional status and quality of life in major areas of adjustment (e.g., occupation, social and family relations, activities of daily living and capacity for self-care, physical health needs, and spiritual fulfillment); (c) psychosocial needs (e.g., financial and housing deficits); (d) patient satisfaction with treatment received and preferences for type and amount of continued treatment; (e) compliance or adherence with treatments provided; and (e) adverse side effects of pharmacological or psychosocial treatments administered.

A number of interview and questionnaire methods are recommended for assessing the diagnostic status and clinical severity of patients (see Annotation E). These measures may be used to identify the presence/absence of major mental disorders, including PTSD, as well as the degree of symptom severity. Much of this information

is important to share with patients in assessing progress of treatment and making collaborative decisions about future directions of care. The DSM-IV criteria for PTSD domains (b – e) can be routinely measured using standard clinical interview methods.

Regular Follow-Up and Monitoring

The use of pencil-and-paper measures of PTSD symptom severity, such as the PTSD Checklist (PCL; see appendix C), should be considered. Scores on the PCL may be plotted serially over time to create a longitudinal record of symptom severity and may be helpful for recognizing environmental (e.g., renewed proximity to a previously abusive parent) or seasonal (e.g., anniversary of a traumatic war event) precipitants of PTSD symptoms.

Early Recognition of a Psychosocial Crisis and Referral to Specialists

Primary care providers may be the first to recognize that a patient with PTSD is entering a psychosocial crisis. Depending on the severity and disability associated with the crisis and the potential for harm to the patient or others, the primary care provider may be obliged to obtain specialty mental health services, even if that patient is reluctant to seek those services.

Coordination of General Healthcare

The traditional role of the primary care provider as the coordinator of various disciplines and consultants involved in the treatment of any single patient is especially relevant for the patient with PTSD. Particularly in patients with chronic PTSD, medically unexplained symptoms or problems with substance use (including smoking) may lead to the need for a wide range of specialists. Coordination of these services is important to avoid confusion and unnecessary healthcare use.

O. Follow-Up

BACKGROUND

Because of risk of relapse following discontinuation of treatment in patients with chronic PTSD, long-term treatment is often needed. Most patients with chronic PTSD (defined by the DSM-IV as full-criterion symptoms lasting 3 months or more) should be monitored for at least 1 year, with regularly scheduled follow-up in order to prevent relapse.

The continued importance of psychoeducation and reinforcement of health-promoting behaviors by the primary care physician is an important but generally neglected area of public health.

RECOMMENDATIONS

1. If patient does not improve or status worsens, consider one of the following treatment modification options:
    a. Continue application of the same modality at intensified dose and/or frequency
    b. Change to a different treatment modality
    c. Apply adjunctive therapies

d. Consider a referral to adjunctive services for treatment of co-morbid disorders or behavioral abnormalities (e.g., homelessness, domestic violence, or aggressive behavior).

e. For patient with severe symptoms or coexisting psychiatric problems consider referrals to:

- Specialized PTSD programs
- Specialized programs for coexisting problems and conditions
- Partial psychiatric hospitalization or "day treatment" programs
- Inpatient psychiatric hospitalization.

2. If patient demonstrates partial (insufficient) remission, consider one of the following treatment modification options:

a. Before making any therapeutic change, ensure that "treatment non-response" is not due to one or more of the following: not keeping psychotherapy appointments, not doing prescribed homework, not taking prescribed medications, still using alcohol or illicit substances, still suffering from ongoing insomnia or chronic pain, not experiencing any new psychosocial stressors, the original assessment did not overlook a co-morbid medical or psychiatric condition

b. Continue the present treatment modality to allow sufficient time for full response

c. Continue application of the same modality at intensified dose and/or frequency

d. Change to a different treatment modality

e. Apply adjunctive therapies

f. Increase level of care (e.g., referral facility, partial hospitalization, inpatient hospitalization, residential care)

g. Consider a referral to adjunctive services for treatment of co-morbid disorders or behavioral abnormalities (e.g., homelessness or domestic violence).

3. If patient demonstrates improved symptoms and functioning but requires maintenance treatment:

a. Continue current course of treatment

b. Consider stepping down the type, frequency, or dose of therapy

c. Consider:

- Transition from intensive psychotherapy to case management contacts
- Transition from individual to group treatment modalities
- Transition to as-needed treatment

d. Discuss patient status and need for monitoring with the primary care provider

e. Consider a referral to adjunctive services for treatment of co-morbid disorders or behavioral abnormalities (e.g., homelessness or domestic violence).

4. If patient demonstrates remission from symptoms and there are no indications for further therapy:
   a. Discontinue treatment
   b. Educate the patient about indications for and route of future care access
   c. Monitor by primary care for relapse/exacerbation.
5. Evaluate psychosocial function and refer for psychosocial rehabilitation, as indicated. Available resources include, but are not limited to: chaplains, pastors, Family Support Centers, Exceptional Family Member Programs, VA benefits counselors, occupational or recreational therapists, Vet Centers, and peer-support groups (see Module I-2 D: Psychosocial Rehabilitation).
6. Provide case management, as indicated, to address high utilization of medical resources.

Table B-4 Treatment Response and Follow-Up

Step	Patient Condition	Options	Reassess at:*
1	Initial Treatment	• Psychotherapy and/or • SSRI/SNRI	2 weeks ** / 4 weeks
2	Non response to initial dose	• Assess adherence • Increase dose • Consider longer duration • Switch to another SSRI or SNRI • Add psychotherapy • Consider referral to specialty care	4 to 6 weeks
3	Failed second trial of antidepressant	• Switch to another SSRI/SNRI or mirtazapine • Add psychotherapy • Augment with prazosin (sleep/nightmare)	8-12 weeks
4	Failed three trials including augmentation	• Re- evaluate diagnosis and treatment • Switch to TCA • If no response consider nefazodone (monitoring side effects), or phenalzine (with careful consideration of risks) • Consider referral to specialty care	> 12 weeks

** Times are general guidelines and may vary considerably*
***If treatment is not tolerable, switch to another antidepressant.*

DISCUSSION

Patient Does Not Improve or Status Worsens:

Re-assessment of patient's clinical status may occasionally show that symptoms and/or functional status are failing to improve or are deteriorating in a sustained way. It is important to determine if this static or deteriorated state is not simply the result of a major life crisis unrelated to the therapy being administered.

The clinician must next determine if a patient's unimproved clinical status reflects a temporary exacerbation of symptoms expected to occur in the course of treatment that will ultimately prove to be effective. For example, it is common for patients in a range of trauma-focused therapies to experience some brief distress or symptom exacerbation during initial phases of treatment where they focus on emotions associated with traumatic memories. In this case, it is important to reassure the patient about the natural course of recovery through treatment, assist him/her in coping with symptoms, and enlist him/her in the decision to continue with the current method of treatment. Increasing session contacts and or increasing the dose of medications may provide needed support.

If the clinician and patient agree that the current treatment regimen is ineffective, then a collaborative decision can be made to switch to a different modality. Another approach is to hold the course of a current therapy, which may appear ineffective, but apply adjunctive treatments (see Module I-2: PTSD Interventions). There is no empirical evidence that supports the effectiveness of combination treatments for PTSD. However, there is clinical consensus that some treatments can act synergistically (e.g., combining coping skills and symptom management approaches with exposure-based treatments).

Clinicians should consider changing the treatment plan by increasing the level of care offered to patients. Levels of care for PTSD vary in intensity, including infrequent visits administered in outpatient clinics, partial hospital programs, specialized inpatient PTSD programs, PTSD residential care programs and domiciliaries, and acute inpatient hospitalization. Patients who fail to progress in outpatient treatment may benefit from a temporary transition to a higher level of care, followed by a return to outpatient management after greater stabilization of symptoms has been achieved.

Often, progress in PTSD treatment may be compromised by a concurrent behavioral disorder (e.g., domestic violence), life crisis (e.g., homelessness), or uncontrolled substance use disorder. Referral to ancillary clinical services should be considered for patients for whom these problems emerge during the course of treatment, as identified upon re-assessment.

Patient Demonstrates Improved Symptoms and Functioning but Requires Maintenance Treatment:

Treatment may also lead to slight or moderate improvement that nonetheless leaves the patient with significant distress and impairment in functioning. If the patient demonstrates partial (insufficient) remission, consider one of the following treatment modification options:

- Continue the present treatment approach to allow sufficient time for full response. This option might be worth considering when a treatment involves acquisition of skills (e.g., cognitive restructuring or anxiety management). In such a case, it is possible that the patient may be in the process of learning the skill, with the full impact of therapy dependent on increased practice and

skill mastery. Or, treatment may not have yet yielded its maximum potential effect because of limited patient compliance; steps taken to increase adherence to treatment prescriptions may accelerate responsivity to the intervention.

- If the moderate level of improvement obtained is less than would be expected, given what is known about the patient and the treatment modality, a change to a different treatment approach may be indicated.
- In certain circumstances, a move to an increased level of care may be warranted. For example, if current functioning remains poor despite some symptom improvement or the patient stands to experience major consequences for failure to improve more rapidly (e.g., marital separation), it may be desirable to move from outpatient care to a higher level of care (e.g., residential care).
- Improvement in PTSD symptoms may be inhibited by the presence of untreated additional problems, such as substance abuse or exposure to domestic violence. In such situations, it is important to initiate services for these problems in order to improve the capacity of the PTSD treatment to effect change.
- Patients with partial PTSD may exhibit clinically meaningful levels of functional impairment in association with their symptoms. Functional impairment, rates of co-morbid disorders, and rates of suicidal ideation were shown to increase linearly with increasing number of PTSD symptoms in one study, and individuals with sub-threshold PTSD had increased suicidal ideation, even after controlling for the presence of co-morbid major depressive disorder (Marshall, 2001).

When Symptoms and Other Trauma-Related Problems Show Significant Improvement, the Options Include the Following:

- Discontinue treatment
- Continue the course of treatment as is
- “Step down” to a treatment requiring less intensive resources.

Clinician judgment, based on discussion with the patient, will be the basis of such a decision.

When therapy has resulted in clinically significant improvement but the improvement in functioning is recent and of limited duration, a continuation of the existing type and intensity of treatment may be indicated if the clinician judges that time is required for the patient to continue practicing new skills or to otherwise consolidate treatment gains. This will be especially true if the clinician judges that a reduction in level of therapeutic support would threaten treatment gains.

If treatment has produced clear benefit but the patient is continuing to show treatment gains week-by-week, it may also be helpful to maintain the treatment as is, in hopes of continued improvement. For many patients, some level of continuing care may be indicated after more intensive help has produced improvements. A step-down to less resource-intensive help can often be accomplished by changing treatment type (e.g., from individual psychotherapy to periodic group support), reducing frequency of contacts (e.g., from once per week to twice per month contact), or reducing treatment dose (e.g., medication).

If treatment has resulted in significant reductions in PTSD but related problems (e.g., anger, social isolation, guilt) have shown little change, it will be important to consider adding treatment components to address those problems or refer the patient for additional services.

Patient Demonstrates Remission from Symptoms:

When the patient demonstrates remission from symptoms and there are no indications for further therapy, it is time to discontinue treatment. Discontinuation of treatment may be anxiety-provoking for some patients who have come to depend on the therapist. If this is the case, it may be helpful to discontinue treatment by using the step-down approach noted above and gradually moving toward termination. Whether treatment is ended gradually or more quickly, it is important to educate the patient about expected levels of continuing symptoms, indicators of relapse or need for future care, and ways of accessing care should the need arise. The patient can be encouraged to talk with his or her primary care provider about the treatment experience and enlist help in monitoring improvement.

Psychosocial Rehabilitation for All Patients with PTSD

Patients with persistent mental health symptoms and needs may benefit from a range of assistance strategies provided by a range of disciplines. In addition to the usual general health and mental health specialists, available resources include, but are not limited to, case management, chaplains, pastors, Family Support Centers, Exceptional Family Member Programs, VA Benefits Counselors, vocational counselors, occupational or recreational therapy, Vet Centers, and peer support groups.

In the primary care setting, appropriate encouragement of patients to obtain a mental health referral is important, even if patients are initially hesitant or reluctant to seek it. Mental health referral options include outpatient psychology, social work, or psychiatry clinics, depending on local resources and policies.

In specialty mental health settings, patients may be referred to specialized PTSD programs or programs that focus treatment on important coexisting problems, such as substance use disorder programs or programs for domestic violence or sexual assault/abuse. Depending on the level of associated disability, complexity of medication regimen, and level of threat to self or others, patients with persistent PTSD symptoms and needs may require inpatient or partial psychiatric hospitalization.

Providers referring from either the primary or specialty mental health setting should consider the need for case management to ensure that the range of patient needs is addressed and that follow-up contact is maintained.

See Module I-2: Interventions for PTSD D: Adjunctive Psychosocial Rehabilitation

MODULE I: TREATMENT INTERVENTIONS

Module I-1. EARLY INTERVENTION TO PREVENT PTSD 102

A. PSYCHOTHERAPY 104

A1. Psychological Debriefing 104
A2. Brief Early Cognitive-Behavioral Intervention 108
A3. Other Early Interventions 109
B. Early Pharmacotherapy Interventions to Prevent Development of PTSD 110

Module I-2. TREATMENT FOR PTSD 114

A. Selection of Therapy for PTSD 114
B. PSYCHOTHERAPY INTERVENTIONS FOR PTSD 115
B1. Therapies that More Strongly Emphasize Cognitive Techniques (CT) 119
B2. Exposure Therapy (ET) 123
B3. Stress Inoculation Training (SIT) 126
B5. Imagery Rehearsal Therapy (IRT) 130
B6. Psychodynamic Therapy 132
B7. Patient Education 133
B9. Dialectical Behavior Therapy 140
B10. Hypnosis 142
B11. Behavioral Couples Therapy 143
B12. Telemedicine and Web-based Interventions 144
C. PHARMACOTHERAPY FOR PTSD 149
D. ADJUNCTIVE SERVICES 167
D1. Psychosocial Rehabilitation 167
D2. Spiritual Support 172
E. SOMATIC TREATMENT 173
E1. Biomedical Somatic Therapies 173
E2. Acupuncture 175
F. COMPLEMENTARY AND ALTERNATIVE MEDICINE 176
F1. Natural Products (Biologically Based Practices) 178
F2. Mind-Body Medicine 179
F3. Manipulation and Body-Based Practices (Exercise and Movement) 180
F4. Energy medicine 180
F5. Whole Medical Systems 181
F6. Other Approaches 182

I-3. MANAGEMENT OF SPECIFIC SYMPTOMS 183

A. Sleep Disturbances 183
B. Pain 189
C. Irritability, Severe Agitation, or Anger 194

Module I-1. EARLY INTERVENTION TO PREVENT PTSD

BACKGROUND

Several studies have examined the effectiveness of treatment interventions and acute symptom management early (within 1 month) following a traumatic event in preventing PTSD. This includes the use of various medications for the prevention of PTSD and brief multiple sessions of psychotherapy.

This section summarizes the evidence supporting the recommendations for early intervention discussed in Module A, Annotation J: Brief Intervention. Table I-1 summarizes the recommendations for interventions, and their potential benefit and harm.

Table I - 1 Early Intervention after Exposure to Trauma (4 to 30 days after exposure)

	Balance of Benefit and Harm			
SR	Significant Benefit	Some Benefit	Unknown Benefit	No Benefit
A	- Brief Cognitive Behavioral Therapy (4-5 sessions)			
B				
C		- Social support		
D				- Individual psychological debriefing ⊘ - Formal psychotherapy for asymptomatic survivors ⊘ - Benzodiazepines ⊘ - Typical Antipsychotics ⊘
I		- Psychoeducation and normalization	- Imipramine - Propranolol - Prazosin - Other Antidepressants - Anticonvulsants - Atypical Antipsychotics - Spiritual support - Psychological First Aid	- Group psychological debriefing

⊘= *Potential harm; SR = Strength of recommendation (see Introduction)*

RECOMMENDATIONS

The following treatment recommendations should apply for all acutely traumatized people who meet the criteria for diagnosis of ASD, and for those with significant levels of acute stress symptoms that last for more than two weeks post-trauma, as well as those who are incapacitated by acute psychological or physical symptoms.

1. Continue providing psychoeducation and normalization.
2. Treatment should be initiated after education, normalization, and Psychological First Aid has been provided and after basic needs following the trauma have been made available.
3. There is insufficient evidence to recommend for or against the use of Psychological First Aid to address symptoms beyond 4 days following trauma. [I]
4. Survivors who present with symptoms that do not meet the diagnostic threshold of ASD or PTSD should be monitored and may benefit from follow-up and provision of ongoing counseling or symptomatic treatment.
5. Recommend monitoring for development of PTSD using validated symptom measures (e.g., PTSD Checklist, other screening tools for ASD/PTSD).
6. **Psychotherapy:**
    a. Consider early brief intervention (4 to 5 sessions) of cognitive-based therapy (CBT) that includes exposure-based therapy, alone or combined with a component of cognitive re-structuring therapy for patients with significant early symptom levels, especially those meeting diagnostic criteria for ASD. [A]
    b. Routine formal psychotherapy intervention for *asymptomatic* individuals is not beneficial and may be harmful. [D]
    c. Strongly recommend **against** individual Psychological Debriefing as a viable means of reducing acute stress disorder (ASD) or progression to post-traumatic stress disorder (PTSD). [D]
    d. The evidence does not support a single session group Psychological Debriefing as a viable means of reducing acute stress disorder (ASD) or progression to post-traumatic stress disorder, but there is no evidence of harm (Note: this is not a recommendation pertaining to Operational Debriefing). [D]
    e. Groups may be effective vehicles for providing trauma-related education, training in coping skills, and increasing social support, especially in the context of multiple group sessions. [I]
    f. Group participation should be voluntary.
7. **Pharmacotherapy:**
    a. There is no evidence to support a recommendation for use of a pharmacological agent to prevent the development of ASDor PTSD. [I]
    b. Strongly recommend **against** the use of benzodiazepines to prevent the development of ASD or PTSD [D]

For discussion of use of medication for specific symptom management during the early phase after trauma, see Module I-3: Symptom Management

DISCUSSION

A. PSYCHOTHERAPY

A1. Psychological Debriefing

Psychological debriefing grew out of practices and experiences involving the military of the United States and other Western nations. For soldiers exhibiting signs of acute stress reaction (ASR) following combat-related traumatic events, the practice of conducting early debriefings as part of a larger restoration approach appeared to have significant impact on reducing more permanent disability.

The use of debriefings soon after exposure to traumatic events became part of military doctrine in the United States and elsewhere, as well as part of standards for early response to catastrophes for organizations, such as the Red Cross. Unfortunately, there are very limited randomized control trial data involving professional work groups (e.g. military units, first responders) trained to respond to traumatic events, for which debriefing procedures were originally intended; these procedures appear to be of little help, and are potentially harmful if used for individual victims of trauma as prophylaxis for PTSD.

In considering the use of debriefing procedures as part of early interventions following trauma exposure, a distinction between the general approaches of psychological versus operational debriefings is in order, as well as debriefing of individual victims of traumatic events and professional work groups trained to respond to these events. Moreover, distinction should be made between debriefing procedures that are targeted at all exposed individuals, irrespective of symptom level, and, by contrast, briefer versions of empirically supported brief psychotherapy interventions that are targeted at symptomatic individuals over a few sessions (see Annotation A2: Brief Early CBT).

DEFINITIONS

Psychological Debriefing is a broad umbrella term used to describe a variety of one-time individual and/or group procedures that involve review of a traumatic event, by survivors or other impacted persons, for the purpose of actively encouraging individuals to: (a) talk about their experiences during the event; (b) recognize and verbalize their thoughts, emotions, and physical reactions during and since the event; and (c) learn about coping methods. Specially trained debriefers lead psychological debriefings following several protocols. Protocols generally emphasize normalization of symptoms, group support, and provide some psychoeducation and information about resources.

The term “Psychological Debriefing” does not include purely informational briefings or debriefings used in professional military or other workgroups (e.g., psychological education lectures or stress management briefings, such as Battlemind Training, Battlemind Debriefing, or operational debriefings) discussed below.

Operational Debriefing is a routine individual or team review of the details of an event from a factual perspective for the purpose of: (a) learning what actually happened for the historical record or planning purposes; (b) improving results in similar future situations or missions; and (c) increasing the readiness of those being debriefed for further action. Operational debriefings are conducted by leaders or specialized debriefers according to the organization’s standing operational procedure. They are often referred to as “after action” reviews.

Operational debriefings achieve important objectives of the organization, and there is no reason that they should have any effect on reducing subsequent PTSD or other long-term negative outcomes (nor is there evidence for this). Organizations that use operational debriefings should train their debriefers to avoid causing unintentional psychological harm (such as by encouraging personal disclosure), and to identify individuals who need behavioral health follow-up.

Critical Incident Stress Debriefing (CISD) is a formalized structured review method in a group format of the stressful experience of a disaster that includes psychological debriefing. In fact, CISD was developed to assist first responders, such as fire and police personnel, not the victims/survivors of a disaster or their relatives. CISD was never intended as a substitute for therapy, was designed to be delivered in a group format with professional work groups, and is meant to be incorporated into a larger, multi-component crisis intervention system, labeled Critical Incident Stress Management (CISM).

Critical incident Stress Management (CISM) incorporates several components, including pre-crisis intervention, disaster or large-scale incident demobilization and informational briefings, "town meetings," staff advisement, defusing, CISD, one-on-one crisis counseling or support, family crisis intervention, organizational consultation, and follow-up and referral mechanisms for assessment and treatment, if necessary.

Battlemind Debriefing is a recently developed intervention, aimed specifically at professional military teams/workgroups (like CISD) and designed to reduce any potential iatrogenic effects of psychological debriefing noted in some studies; specifically, less emphasis is given to personal disclosure and review of index events (there is no requirement for individual disclosure; the focus of the debriefing is more broadly on the transition from the entire deployment, rather than a single critical incident), and more emphasis is given to enhancement of peer support. Battlemind debriefing can be delivered in either small group or large group lecture formats. Although Battlemind debriefing has been designed to be used by military units immediately after critical incidents, it has never been tested in this setting. Two published studies of Battlemind debriefing have focused on the post-deployment timeframe in which the entire deployment and facilitating transition home from deployment has been the focus of the intervention.

Individual Debriefing

Reviews and meta-analyses of studies of psychological debriefing as an early intervention to reduce or prevent PTSD symptoms in individuals have concluded that this technique is ineffective or potentially harmful (Rose et al., 2002). Of note, two well-controlled studies with longer-term follow-up of individual patients have suggested that this intervention may be related to a poorer outcome compared to controls (Bisson, 1997; Mayou et al., 2000 which is a follow-up on Hobbs, 1996). Bisson et al. (2008), in a summary of the evidence in the ISTSS guideline (2009), also found no evidence to support the preventive value of individual debriefing delivered in a single session. Of the 10 studies that compared psychological debriefing with no interventions, 2 were positive, 5 were neutral, and 3 had negative results. A meta-analysis conducted by Van Emmerick et al. (2002) included seven studies and found that psychological debriefing interventions (non-CISD) and no intervention improved symptoms of post-traumatic stress disorder, but psychological debriefing did not improve symptoms. Cuijpers et al. (2005) assessed the results of studies examining the effect of prevention and found that the risk of post-traumatic stress disorder was somewhat increased after debriefing but not significantly

(RR=1.33), indicating a possible adverse effect. The RCTs to date cover only a limited variety of traumatic stressors, subject populations, and debriefing protocols. Most controlled studies have been of individually administered, one-time individual debriefings of victims of motor vehicle accidents or crimes, such as rape. However, findings have been consistent across trials.

Group Debriefing

The recommendations regarding group debriefing, either of victims of trauma or professional work groups, is based primarily on the lack of effectiveness in studies; there does not appear to be any evidence of harm. In a partially randomized trial, Deahl et al. (2000) found no benefit of debriefing over assessment only in terms of PTSD symptoms; however, the group receiving debriefing evidenced lower alcohol misuse scores. The non-random assignment to groups weakens conclusions of this study (Commanders blind to condition separated approximately 100 soldiers into two groups based on schedules and responsibilities; the groups were then randomly designated 'debriefing' or 'control.' Thus, outcomes are confounded by whatever factors were used for separating soldiers into groups by commanders.) In another study by Campfield and Hills (2001), robbery victims were randomly assigned to immediate (less than 10 hours) or delayed (greater than 48 hours) CISD groups. Immediate CISD produced more pronounced reduction in symptoms, but no control group was employed, and thus no conclusions regarding efficacy relative to no treatment can be made. This is particularly necessary with this intervention, given that most people will recover spontaneously without any intervention and because of the potentially iatrogenic effects found in some studies of CISD with individuals. Other studies of group debriefing that have been conducted were of poor design in terms of low sample size and/or non-random assignment to group and preclude conclusions regarding efficacy (Eid et al., 2001; Richards, 2001). In an analogue study with students, Devilly et al. (2008) found no advantage of debriefing following a distressing video relative to a post-video snack.

Two more RCT's are relevant to the discussion of group debriefing in combat units, although not specifically at the time of the critical incident events. Adler et al. (2008) conducted a randomized trial of Critical Incident Stress Debriefing (CISD) of groups of soldiers deployed to a Kosovo peacekeeping mission. This trial randomised 1,050 soldiers from 19 platoons into 62 groups receiving one of three conditions: Debriefing (23 groups), Stress Education (20 groups) and No Intervention (19 groups), and focused on the entire deployment period. The authors reported no differences between groups on all behavioural outcomes, though the deployment had resulted in relatively few critical events. In a second RCT by Adler et al. (2009) with U.S. soldiers returning from Iraq who had been exposed to direct combat throughout their deployment, results indicated that compared to a Stress Education control condition, the Battlemind Debriefing had no overall effect on PTSD; within the subgroup with highest levels of combat exposure, Battlemind Debriefing was no more effective than the Battlemind Training lecture (given in both small group and large group formats), with both treatments producing small improvements in PTSD Check List (PCL) scores. A small but significant reduction in PTSD symptoms, depression symptoms, and sleep problems was observed in Soldiers with the highest levels of combat exposure for Battlemind Debriefing compared with standard stress education, although similar benefits were observed for the two other Battlemind training classroom interventions. Thus, given the similar efficacy of the Battlemind Training lecture program, and the very small effect sizes observed, there is no reason to recommend Battlemind Debriefing over the Battlemind lecture program.

It remains possible that group interventions with pre-existing work groups (teams, units, EMTs, co-workers) immediately after traumatic events may assist with prevention of PTSD symptoms or with non-PTSD areas of improvement, such as group cohesion, morale, and other important variables, but the empirical evidence for this is insufficient due to poorly designed studies. Similarly, group interventions may be useful for screening, education, and support. Trained personnel should lead these group interventions and if group approaches are used, group participation should be voluntary. Operational debriefings after traumatic events during on-going military operations also share these considerations, but they have other objectives that may override individual mental health protection. All operational debriefings should select protocols and train the debriefers to minimize psychological harm to the participants.

In conclusion, routine use of individual debriefing or the use of group psychological debriefing for victims of trauma cannot be recommended. There is insufficient evidence for the use of psychological debriefing for professional work groups immediately after critical incidents, though no evidence of harm. The use of post-deployment psychological debriefing in the military is not recommended due to the fact that other forms of psychological training were found to be generally equivalent; there is no evidence of harm. Of importance is the fact that other early treatment interventions have been found to prevent PTSD in symptomatic individuals (see Annotation A2: Brief Early Cognitive-Behavioral Intervention). It appears appropriate to continue to focus resources on identifying and treating those with symptoms arising after trauma. The emphasis should be placed on the early detection of those at risk of developing psychopatholgy and those early interventions that have been found effective should be aimed at this group.

EVIDENCE TABLE

	Evidence	Sources	LE	QE	NET	SR
1	Individual or group psychological debriefing of victims of traumatic events is ineffective and may have adverse effects	Bisson et al., 2009 (ISTSS) Campfield and Hill, 2001 Cuijpers et al., 2005 Devilly et al., 2008 Hobbs et al., 1996 Mayou et al., 2000 Bisson, 1997 Rose, 2002 (Cochrane SR) Sijbrandji et al., 2006 Van Emmerick et al., 2002	I	Good	Zero Small	D
2	There is insufficient evidence for or against psychological debriefing of professional workgroups (e.g. military, first responders) in the immediate aftermath of critical incidents	Carlier et Al., 2000 Deale, et al., 1994 Dolan et al., 1999 Eid et al., 2001 Richards, 2001	I, II-1	Fair	Zero Small	I
3	Psychological debriefing of professional work groups weeks or months after critical incidents is not recommended	Adler et al., 2008 Adler et al., 2009 Deahl et al., 2000	I	Good	Zero, Small	D

LE =Level of Evidence; QE = Quality of Evidence; NET=Net benefit; SR = Strength of Recommendation (see Appendix A)

A2. Brief Early Cognitive-Behavioral Intervention

Research suggests that relatively brief but specialized interventions may effectively prevent PTSD in some subgroups of trauma patients. Several controlled trials have suggested that brief (i.e., 4 to 5 sessions) cognitive-behavioral treatments, comprised of education, breathing training/relaxation, imaginal and in vivo exposure, and cognitive restructuring, delivered within weeks of the traumatic event, can often prevent PTSD in survivors of sexual and non-sexual assault (Foa et al., 1995), MVAs, and industrial accidents (Bryant et al., 1998 , 1999). Brief intervention with patients hospitalized for injury has been found to reduce alcohol consumption in those with existing alcohol problems (Gentilello et al., 1999). Controlled trials of brief early intervention services targeted at other important trauma sequelae (e.g., problems returning to work, depression, family problems, trauma recidivism, and bereavement-related problems) remain to be conducted, but it is likely that targeted interventions may be effective in these arenas for at least some survivors.

At present, it is unknown how much time should elapse after a traumatic experience before cognitive-behavioral intervention is initiated (Litz & Bryant: in Foa 2009 [ISTSS]). If provided too early, individuals who may not need therapy will consume helping resources. For this reason, trials have not commenced before 2 weeks after the trauma (Bryant, 1998, 1999, 2003).

Target Population for Brief CBT

Studies that have targeted all trauma survivors, regardless of levels of stress reactions, have been ineffective in preventing PTSD (Roberts et al., 2009b). Trauma-focused CBT has been found to be effective in reducing and preventing post-traumatic stress symptoms in individuals who were symptomatic, especially those meeting criteria for ASD (Roberts et al., 2009a; Stapleton, 2006). These interventions have focused on the traumatic experience via exposure to memories and trauma reminders, sometimes combined with cognitive therapy or other behavioral interventions. One study has indicated that combined imaginal and in vivo exposure is significantly more effective than pure cognitive restructuring in reducing subsequent PTSD among individuals diagnosed with ASD (Bryant, et al., 2008a). This is an important finding that requires replication.

Cognitive behavioral therapy was more effective in reducing symptoms than a self-help booklet or repeated assessment. The combination of an elevated initial symptom score and failure to improve with self-monitoring was effective in identifying a group of patients with early PTSD symptoms who were unlikely to recover without intervention. (Ehlers, 2003)

Evidence Table

	Evidence	Sources	LE	QE	SR
1	Brief cognitive-behavioral intervention (4 to 5 sessions) may prevent PTSD in those reporting clinically significant symptoms of acute post-traumatic stress	Roberts, 2009a (§) Kornor, 2008 Bryant et al., 1998, 1999 Bryant et al., 2003, 2008a	I	Good	A
2	Multisession early psychological interventions for asymptomatic trauma survivors are not effective and may be harmful	Roberts, 2009b (§)	I	Good	D

LE =Level of Evidence; QE = Quality of Evidence; SR = Strength of Recommendation; §-Systematic Review (see Appendix A)

A3. Other Early Interventions

Efficacious early interventions have largely been structured as brief versions of effective PTSD treatments. This suggests that other interventions may be effective in preventing PTSD, but more research is needed to investigate other intervention methods. Some non-CBT interventions have received research attention. For example, brief structured writing has been found ineffective in preventing PTSD in two studies (van Emmerik, et al., 2008; Bugg, et al., 2009). A memory restructuring intervention failed to show preventive impact relative to a control condition (Gidron et al., 2007). Likewise, providing self-help information as a preventive psychoeducation strategy to prevent PTSD has not been found to be efficacious (Scholes et al., 2007; Turpin et al., 2005)

Table I - 2 Brief Psychotherapy Studies to Prevent the Development of PTSD

Author, Year	Results	n	Trauma	LE	QE	NB
Cognitive Behavioral Therapy (CBT)						
Bryant, 1998a	Brief (5 sessions) CBT within 2 weeks > Supportive counseling in preventing PTSD	24	Civilian	I	Fair	Mod
Bryant, 1999	Brief (5 sessions) PE or PE + Anxiety mgmt > Supportive counseling in preventing PTSD	45	Civilian with ASD	I	Good	Sub
Bryant, 2003	Brief (5 sessions) CBT > Supportive Counseling in preventing PTSD	24	ASD after mTBI	I	Good	Sub
Bryant, 2008a	Brief (5 sessions) ET > CT > No Tx in preventing PTSD	90	ASD civilians	I	Good	Sub
Resnick, 2007	Video intervention reduces PTSD vs. standard care	140	Sexual assault	I	Good	Mod
Self-Help (SH)						
Scholes, 2007	no group differences between SH and no Tx	227	Emergency Room	I	Good	Zero
Turpin, 2005	no group differences in PTSD between SH and no Tx	141	N/R	I	Fair	Zero
Structured Writing Therapy (SWT)						
van Emmerik, 2008	Efficacy of SWT was comparable to CBT	125	ASD and PTSD pts	I	Good	Mod
Bugg, 2009	No differences between writing and self help (information only) groups	67	Emergency room	I	Mod	Small
Memory Structured Intervention (MSI)						
Gidron 2007	No differences between MSI and supportive listening.	34	traffic accident victims	I	Fair	Zero

ET = Exposure therapy; CT= Cognitive Therapy; Tx=Treatment
LE =Level of Evidence; QE = Quality of Evidence;
NB=Net benefit: Sub=Substantial; Mod=Moderate; Zero=–None or small

B. Early Pharmacotherapy Interventions to Prevent PTSD

Prevention of PTSD

Few studies have examined the effectiveness of pharmacological treatment for acute symptom management and PTSD prevention immediately following a traumatic event. This includes the use of various agents for the prevention of PTSD (propranolol, hydrocortisone, and gabapentin). Although of interest, none of these approaches is yet advocated in standard treatment guidelines for PTSD (Stein 2009 [SR]). There is insufficient evidence to draw concrete conclusions or make specific recommendations regarding the use of pharmacological agents for prevention of PTSD. While prevention of ASD is ideal, there are currently no evidence-based pharmacological treatment modalities to arrest symptom formation and prevent progression to ASD during the first days and weeks following the traumatic exposure.

Once potential medical causes of neuropsychiatric impairment are ruled out and other immediate needs are met (e.g., physical needs, practical needs for assistance, normalization, and psychoeducation), then both medications and non-pharmacological interventions may be considered. The selection and effectiveness of specific interventions administered acutely are not well supported in the literature. Although there are no evidence-based pharmacological treatments for ASD, there may be a role for pharmacotherapy to aid in the management of specific symptoms (e.g., insomnia, pain, hyperarousal).

Use of Benzodiazepines

Historically, benzodiazepines were the primary agent in PTSD treatment, particularly alprazolam and clonazepam. However, based on the limited data that are available, benzodiazepine administration should be used with caution (or discouraged) both in acute stress disorder (ASD) and post-traumatic stress disorder (PTSD), due to lack of evidence for effectiveness and risks that may outweigh potential benefits. Although benzodiazepines have been frequently used “as needed” and continuously for anxiety disorders, including to augment evidence-based treatment modalities in PTSD, there is theoretical, animal, and human evidence to suggest that benzodiazepines may actually interfere with the extinction of fear conditioning or *potentiate* the acquisition of fear responses and worsen recovery from trauma. Benzodiazepine should be used especially cautiously in combat veterans with PTSD because of the very high co-morbidity of combat-related PTSD with alcohol misuse and substance use disorders (upwards of 50 percent of co-morbidity) and potential problems with tolerance and dependence. Once initiated, benzodiazepines can be very difficult, if not impossible, to discontinue due to significant withdrawal symptoms compounded by the underlying PTSD symptoms.

Gelpin et al. (1996), in an open-labeled study, treated patients who had recently experienced trauma (within the past 18 days) and were experiencing excessive distress (panic, agitation, or persistent insomnia) for up to 6 months with alprazolam or clonazepam. These 13 patients were compared with a control group of recently traumatized individuals matched for demographics and symptoms (using the Impact of Events Scale). On follow-up, PTSD occurred at a significantly higher rate in the benzodiazepine-treated group (9/13, 69 percent) than in the control group (2/13, 15 percent). Although the strength of the evidence is low (open-labeled study), the study suggested that benzodiazepines may worsen outcomes in the acute period following trauma, and the authors referenced animal data consistent with the hypothesis that benzodiazepines may potentiate the acquisition of fear responses.

Mellman, Bustamante et al. (2002) conducted a double-blind randomized controlled study, during the acute period after trauma (mean 2 weeks after trauma). A short-term (7 day) evening use of temazepam in patients with significant ASD/PTSD symptoms was compared with placebo (11 patients in each group). The study showed no benefits in preventing PTSD, and the trend was similar to the Gelpin study, with 6 of 11 (55 percent) patients who received tamazepam developing PTSD, compared with 3/11 (27 percent) who received placebo.

Davydow, (2008) in a literature review of the risk factors for developing PTSD after serious trauma (requiring ICU treatment), found that greater ICU benzodiazepine administration was one of the consistent predictors of PTSD.

Benzodiazepines can be effective against anxiety and insomnia, but they should be used with caution in patients with ASD and PTSD because of the high frequency of co-occurring substance abuse and dependence in patients with PTSD. The balance between benefit and potential risks, including the risks of dependency and of withdrawal after discontinuation, should be evaluated when considering benzodiazepines in patients with acute stress reaction.

Sleep Disturbance

One of the most difficult symptoms to address in the immediate aftermath of exposure to a traumatic event is sleep disturbance. Theoretically, the more sleep impairment and trauma-related nightmares an individual continues to experience, the more likely he or she is to continue to experience the symptoms of ASD and/or subsequently develop PTSD. There is little evidence for the effectiveness of any sleep aids in the immediate aftermath of trauma.

For Recommendations and discussion of the evidence for sleep disturbance, see Module I-3: A. Sleep Disturbances

Ineffectiveness of Propranolol

Several studies have examined the use of propranolol, hydrocortisone, and gabapentin for the prevention of PTSD (Pittman, 2002; Stein, 2007).

Four small and brief clinical trials were identified in the peer-reviewed medical literature that evaluated the use of pharmacological treatments to prevent the development of post-traumatic stress disorder (PTSD) symptoms in traumatized subjects (Pitman, 2002; Stein, 2007; Reist, 2001; Vaiva, 2003). All studies involved immediate post-traumatic administration of propranolol, and one study also included a trial of gabapentin. Two of the studies (Reist, 2001; Vaiva, 2003) were excluded due to poor quality. Pitman (2002) reported a pilot study of 41 patients who were randomized to begin, within 6 hours of the event, a 10-day course of double-blind propranolol (n = 18) versus placebo (n = 23), 40 mg four times daily. Significant improvement of symptoms was noted in the treatment group. Stein (2007) conducted a double-blind, randomized controlled trial of 14 days of the beta-blocker propranolol (n = 17), the anxiolytic anticonvulsant gabapentin (n = 14), or placebo (n = 17), administered within 48 hours of injury to patients admitted to a surgical trauma center. Of 569 accessible, potentially eligible subjects, 48 (8 percent) participated. Although well tolerated, neither study drug showed a significant benefit over placebo on depressive or post-traumatic stress symptoms.

McGhee et al. (2008) examined the relationship between PTSD prevalence and propranolol administration in 603 soldiers injured in OIF/OEF, of whom 226 completed the PTSD Checklist-Military. Thirty-one soldiers received propranolol, and 34 matched soldiers did not. In propranolol patients, the prevalence of PTSD was

32.3 percent vs 26.5 percent in those not receiving propranolol (P = .785). These data suggest propranolol does not decrease PTSD development in burned soldiers.

Although some positive results were noted, the size and weak study designs of the investigations do not allow for definitive conclusions regarding the value of these medications in preventing the development of PTSD symptoms after traumatic events.

Early Pain Intervention to Prevent PTSD

Acute pain caused by physical injury may by itself be a precursor for PTSD. Injury that is also associated with traumatic exposure increases the risk for PTSD. When pain is treated early and aggressively, patients may have the best chance of getting better. Though many fear addiction from opioids, they can be an important part of halting the pain cycle. Few studies have investigated the effect of pain reduction in the early stages after injury and the development of PTSD.

Bryant et al. (2008c) examined the influence of acute administration of morphine as protective against the development of PTSD in a consecutive sample of patients admitted to hospital after traumatic injury (n = 155) . The patients who met criteria for PTSD at 3 months (14 percent) received significantly less morphine than those who did not develop PTSD; there was no difference in morphine levels in those who did and did not develop a major depressive episode or another anxiety disorder. The authors suggested that administration of morphine in the acute post-traumatic stage may limit fear conditioning in the aftermath of traumatic injury and may serve as a secondary prevention strategy to reduce PTSD development.

Holbrook et al. (2010) analyzed data for 696 military personnel (mostly male, mean age about 24) who were hurt during OIF but who did not have serious traumatic brain injury. About one-third (35 percent) of the injured personnel developed PTSD. The finding was that those who had been administered morphine shortly after their injury (60 percent versus 76 percent) were less likely to develop PTSD (ORs ranging from 0.48 to 0.66, $P<0.05$ for all). Several factors, including severity and mechanism of injury, need for amputation, resuscitation, and the presence of mild traumatic brain injury, were adjusted for. Although causality could not be established, the authors concluded that a reduction in perceived pain levels through the use of morphine or other opioids, as part of trauma care, may lower the rate of PTSD onset after major trauma.

Other Medications

One study that involved administration of cortisol at the time of cardiac bypass surgery (Schelling, 2004) suggested that patients who received stress doses of cortisol had lower PTSD symptom scores than a comparison group (that did not receive cortisol) when questioned six months after surgery.

A crossover trial of 1 month of low-dose cortisol therapy evaluated 3 patients diagnosed with PTSD (Aerni, 2004). The authors reported that each patient demonstrated improvement on at least 1 self-reported PTSD measure. The study was excluded from analysis for this guideline due to small numbers.

Conclusions:

There is a small amount of evidence that suggests that administration of cortisol at the time of, or immediately after, a traumatic event may have a preventive effect on the subsequent development of PTSD symptoms. Little evidence exists suggesting

that gabapentin or propanolol are of value in preventing the development of PTSD after trauma.

Due to the limited support of evidence, the use of medications in the early period post-trauma to prevent PTSD cannot be recommended. Pharmacotherapy may be considered to aid in the management of specific symptoms (e.g., addressing sleep disturbance, irritability, or control of pain).

EVIDENCE TABLE

	Evidence	Sources	LE	QE	Net Effect	SR
1	Pharmacotherapy prophylaxis for PTSD	Stein, 2006 [§]	I	Poor	-	I
2	Propranolol to reduce hyperarousal, excessive arousal, or panic attacks	Pittman et al., 2002 Stein et al., 2007 Reist et al., 2001 Vaiva et al., 2003 McGhee et al.,2008	I	Fair	Small	C
3	Benzodiazepines for hyperarousal, excessive arousal, or panic attacks	Gelpin et al., 1996 Melman et al., 2002 Davydow, 2008	II-2 I	Fair	Small/Neg	D

*LE =Level of Evidence; QE = Quality of Evidence; SR = Strength of Recommendation §=*Systematic Review *(see Appendix A)*

Table I - 3 Pharmacological Studies to Prevent the Development of PTSD

Author, Year	Results	n	Trauma	LE	QE	NB
Propranolol						
Pitman, 2002	Significant improvement post-acute stress	41	Any	I	Good	Small
Stein, 2007	No difference from placebo (gabapentin or propanolol)	48	Severe physical injury	I	Good	Zero
Reist, 2001	Recall of arousing story was reduced	38	N/R	I	Poor	EXC
Vaiva, 2003	PTSD rate and symptoms lower in the propanolol group	19	MVA, assault	I	Poor	EXC
Cortisol						
Schelling, 2004	Hydrocortisone administered during cardiac surgery reduced chronic stress symptom scores	91	Bypass surgery		Fair	Mod
Aerni, 2004	Low-dose cortisol for 1 month reduces the cardinal symptoms of PTSD	3			Poor	EXC
Morphine						
Bryant et al. (2008c)	Patients with PTSD received sig. less morphine than those who did not develop PTSD	155	Injury	II	Fair	Mod
Holbrook et al. (2010)	Wounded morphine shortly after their injury reduced development of PTSD		Combat injury	II-2	Fair	Mod

LE =Level of Evidence; QE = Quality of Evidence;
NB=Net benefit: Sub=Substantial; Mod=Moderate; Zero –None or small; N/R=no reported; EXC= Excluded;

Module I-2. TREATMENT FOR POST-TRAUMATIC STRESS DISORDER (PTSD)

A. Selection of Therapy for PTSD

In clinical practice, providers and patients alike are often faced with important decisions relating to type, number, frequency, and dose of various psychotherapies and pharmacological interventions. Therapies may be broadly divided into (1) evidence-based psychotherapies (e.g., trauma-focused therapies or stress inoculation training), (2) evidence-based pharmacotherapies (particularly SSRIs and SNRIs), and (3) key adjunctive or supplemental treatment modalities.

Providers should explain to all patients with PTSD the range of therapeutic options that are available and effective for PTSD. This discussion should include general advantages and disadvantages associated with each therapeutic option (including side-effects/risks, and time commitment required to complete the therapy). In general, PTSD therapy research has provided sufficient evidence to recommend medication or evidence-based psychotherapy as a first-line treatment. Among the A-level evidence-based psychotherapy treatments, the research suggests that they are much more equivalent in their effectiveness than many clinicians may realize. There is insufficient evidence to suggest for or against combined medication and psychotherapy over only one of the two approaches. Patient preferences and the particular evidence-based treatments that the provider has the most training/expertise in will often drive the initial therapeutic approach.

The level or intensity of care is guided by illness trajectory (degree of chronicity and illness severity), observed outcomes, and previous therapies. Active follow-up is used to determine the level of care each patient requires over time. The provider along with the patient may determine that the first-line therapy will be psychotherapy. If, after a period of treatment, the patient is not responding adequately, the patient may be “stepped up” in therapeutic intensity by adding a medication, such as a selective serotonin reuptake inhibitor (SSRI) to the regimen of ongoing psychotherapy, and reassessing whether additional measures need to be taken to address co-morbid conditions. It may be helpful to coordinate care using a collaborative care approach based in primary care that includes care management. Although supporting evidence is lacking for collaborative care approaches, these approaches have been shown to be useful in the management of depression, chronic pain, chronic fatigue, and other conditions, and are now being tested for PTSD in some military and VA treatment facilities.

RECOMMENDATIONS

1. Providers should explain to all patients with PTSD the range of available and effective therapeutic options for PTSD.
2. Patient education is recommended as an element of treatment of PTSD for all patients and the family members. [C]
3. Patient and provider preferences should drive the selection of evidence-based psychotherapy and/or evidence-based pharmacotherapy as the first line treatment.
4. Psychotherapies should be provided by practitioners who have been trained in the particular method of treatment.
5. A collaborative care approach to therapy administration, with care management, may be considered, although supportive evidence is lacking specifically for PTSD.

B. PSYCHOTHERAPY INTERVENTIONS FOR PTSD

Psychotherapy interventions are aimed at reduction of symptoms severity, improvement of global functioning, and improvement in quality of life and functioning in social and occupational areas. Psychotherapy for PTSD may also have benefits in improving co-morbid physical health conditions, but this is not specifically the focus of treatment.

Table I-4 Psychotherapy Interventions for Treatment of PTSD

	Balance of Benefit and Harm			
SR	**Significant Benefit**	**Some Benefit**	**Unknown Benefit**	**None**
A	**Trauma-focused** psychotherapy that includes components of exposure and/or cognitive restructuring; or, **Stress inoculation** training			
C		Patient Education Imagery Rehearsal Therapy Psychodynamic Therapy Hypnosis Relaxation Techniques Group Therapy		
I		Family Therapy	WEB-Based CBT Acceptance and Commitment Therapy Dialectical Behavioral Therapy	

SR_= Strength of Recommendation (see Appendix A)

Effective Psychotherapies for PTSD

There are significant difficulties in categorizing the different evidence-based psychotherapies that have been found to be most effective for PTSD. There are a number of reasons for this difficulty, including the diversity of treatments available, a lack of a common terminology to describe the same treatment components, the specific ways in which similar components are manualized or packaged, and lack of consensus between proponents for specific treatments.

The evidence-based psychotherapeutic interventions for PTSD that are most strongly supported by RCTs can be considered broadly within in the **trauma-focused psychotherapy** category or **stress inoculation training**. Trauma-focused psychotherapies for PTSD refer to a broad range of psychological interventions based on learning theory, cognitive theory, emotional processing theory, fear-conditioning models, and other theories. They include a variety of techniques most commonly involving exposure and/or cognitive restructuring (e.g. Prolonged Exposure, Cognitive Processing Therapy and Eye movement Desensitization and Reprocessing). They are often combined with anxiety management/stress reduction skills focused specifically on alleviating the symptoms of PTSD. Psychoeducation is another important component of all interventions. Other CBT interventions that are not trauma-focused are less effective.

Stress inoculation training (SIT) does not necessarily focus as explicitly on the exploration of traumatic memories, it is included as a first-line alternative to trauma-focused psychotherapies for treating PTSD. SIT, which was developed originally for anxiety disorders and then modified for rape victims and later for PTSD, has been extensively studied in the treatment of PTSD. It has also been compared head-to-head with trauma-focused psychotherapies, and has been shown to be effective in assisting individuals with reducing trauma-related avoidance, anxiety, and cognitions, and there is good evidence that it is equivalent in efficacy to the trauma-focused psychotherapies.

In formulating the specific recommendations for psychotherpy, the working group evaluated the empirical evidence, considering randomized trials as the highest level of the evidence-based hierarchy. It should be noted that therapy provided in clinical trial settings differs from therapy that is practiced in day-to-day care, and the recommendations represent the techniques and protocols as they were studied and reported in the RCTs.

Packaging of Manualized Approaches of Therapy

The working group recognized that despite various perspectives on how to categorize the most effective PTSD psychotherapies, all of the modalities supported by a level-A evidence likely have overlapping mechanisms of action. Trauma-focused psychotherapies include exposure techniques that involve repetitive review of traumatic memories and trauma-related situations, cognitive techniques that focus on identification and modification of trauma-related beliefs and meanings, and/or stress reduction techniques designed to alleviate PTSD symptoms and assist patients in gaining control and mastery over the physiological reactivity.

SIT protocols that have been tested in clinical trials often include components of cognitive restructuring or in-vivo exposure, and some SIT techniques (e.g. breathing retraining, relaxation) are incorporated into virtually every other trauma-focused psychotherapy that has been studied in RCTs. Consequently, it is difficult to disentangle the relative contribution of SIT techniques in the efficacy of the other trauma-focused psychotherapy treatments.

Components of efficacious interventions for PTSD, studied in clinical trials, have been packaged in various ways. Most RCTs have manualized the techniques to ensure the fidelity of treatment for use by the investigators. Some manualized approaches have gained wide popularity, but there is no evidence that they are any more effective than less accepted protocols that package the core components of trauma-focused therapies in different ways. The core components used in the vast majority of A-level interventions have involved combinations of exposure (particularly in-vivo and imaginal/oral narrative), cognitive restructuring, relaxation/stress modulation techniques, and psychoeducation. Very few studies have dismantled these individual components to assess the relative efficacy of each technique independently. The approaches that have been most extensively studied can be generally grouped into four main categories based on the therapeutic components given the most emphasis, or the specific way in which these components were packaged, although there is overlap between these groups:

- *Exposure-based therapies (ET)* emphasize in-vivo, imaginal, and narrative (oral and/or written) exposure, but also generally include elements of cognitive restructuring (e.g. evaluating the accuracy of beliefs about danger) as well as relaxation techniques and self-monitoring of anxiety. Examples of therapies that include a focus on exposure include Prolonged Exposure Therapy, Brief Eclectic Psychotherapy, Narrative Therapy, written exposure therapies, and many of the

cognitive therapy packages that also incorporate in-vivo and imaginal/narrative exposure.

- *Cognitive-based therapies* (*CT)* emphasize cognitive restructuring (challenging automatic or acquired beliefs connected to the traumatic event, such as beliefs about safety or trust) but also include relaxation techniques and discussion/narration of the traumatic event either orally and/or through writing. Examples include Cognitive Processing Therapy and various cognitive therapy packages tested in RCTs
- *Stress Inoculation Training (SIT)* (the specific anxiety management package most extensively studied in the PTSD literature), places more emphasize on breathing retraining and muscle relaxation, but also includes cognitive elements (self-dialogue, thought stopping, role playing) and, often, exposure techniques (in-vivo exposure, narration of traumatic event).
- *Eye Movement Desensitization and Reprocessing (EMDR) (extensively studied in* a large number of RCTs) closely resembles other CBT modalities in that there is an exposure component (e.g. talking about the traumatic event and/or holding distressing traumatic memories in mind without verbalizing them) combined with a cognitive component (e.g., identifying a negative cognition, an alternative positive cognition, and assessing the validity of the cognition), and relaxation/self-monitoring techniques (e.g., breathing, "body scan"). Alternating eye-movements are part of the classic EMDR technique (and the name of this type of treatment); however, comparable effect sizes have been achieved with or without eye movements or other forms of distraction or kinesthetic stimulation. Although the mechanisms of effectiveness in EMDR have yet to be determined, it is likely that they are similar to other trauma-focused exposure and cognitive-based therapies.

A brief description and summary of the supporting evidence for each of the above, and other therapy approaches is included in the following Sections B1 to B12 of the Discussion.

RECOMMENDATIONS

Treatment Options:

1. Strongly recommend that patients who are diagnosed with PTSD should be offered one of the evidence-based trauma-focused psychotherapeutic interventions that include components of exposure and/or cognitive restructuring; or stress inoculation training. [A]

   The choice of a specific approach should be based on the severity of the symptoms, clinician expertise in one or more of these treatment methods and patient preference, and may include an exposure-based therapy (e.g., Prolonged Exposure), a cognitive-based therapy (e.g., Cognitive Processing Therapy), Stress management therapy (e.g., SIT) or Eye Movement Desensitization and Reprocessing (EMDR).

2. Relaxation techniques should be considered as a component of treatment approaches for ASD or PTSD in alleviating symptoms associated with physiological hyper-reactivity. [C]
3. Imagery Rehearsal Therapy [IRT] can be considered for treatment of nightmares and sleep disruption. [C]
4. Brief Psychodynamic Therapy can be considered for patients with PTSD. [C]

5. Hypnotic Techniques can be considered, especially for symptoms associated with PTSD, such as pain, anxiety, dissociation, and nightmares, for which hypnosis has been successfully used. [C]
6. There is insufficient evidence to recommend for or against Dialectical Behavioral Therapy (DBT) as first-line treatment for PTSD [I]
   - Dialectical Behavioral Therapy can be considered for patients with a borderline personality disorder typified by parasuicidal behaviors. [B]
7. There is insufficient evidence to recommend for or against Family or Couples Therapy as first-line treatment for PTSD; Family or Couples therapy may be considered in managing PTSD-related family disruption or conflict, increasing support, or improving communication. [I]
8. Group Therapy may be considered for treatment of PTSD [C]
   - There is insufficient evidence to favor any particular type of group therapy over other types
   - Patients being considered for group therapy should exhibit acceptance for the rationale for trauma work, and willingness to self-disclose in a group.
9. Consider augmenting with other effective evidence-based interventions for patients who do not respond to a single approach.
10. Supportive psychotherapy is not considered to be effective for the treatment of PTSD. However, multiple studies have shown that supportive interventions are significantly more helpful than no treatment, and they may be helpful in preventing relapse in patients who have reasonable control over their symptoms and are not in severe and acute distress.

Note:

Approaches may also be beneficial as parts of an effectively integrated approach. Most experienced therapists integrate diverse therapies, which are not mutually exclusive, in a fashion that is designed to be especially beneficial to a given patient.

Delivery of care:

1. Telemedicine interventions that involve person-to-person individual treatment sessions appear to have similar efficacy and satisfaction clinically as a direct face-to-face interaction, though data are much more limited than for face-to-face encounters. [C]
   a. Telemedicine interventions are recommended when face-to-face interventions are not feasible due to geographic distance between patient and provider or other barriers to patient access (e.g., agoraphobia, physical disability); when the patient would benefit from more frequent contact than is feasible with face-to-face sessions; or when the patient declines more traditional mental health interventions.
   b. Providers using telemedicine interventions should endeavor to maintain and strengthen the therapeutic relationship, build patient rapport, stress practice and assignment completion, and ensure adequacy of safety protocols using similar techniques as they do in a face-to-face session.

c. Providers using technology-assisted interventions should take steps to ensure that their work complies with the regulations and procedures of the organization in which they are employed, legal standards, and the ethical standards of their professions. Patient confidentiality and safety should be monitored closely.

2. There is insufficient evidence to recommend for or against Web-based interventions as a stand-alone intervention or as an alternative to standard mental health treatment for PTSD. [I]

   If used:

   a. Clinicians should carefully review the content of any web-based materials to ensure their accuracy and ethical application before recommending use to patients.

   b. Web-based approach may be used where face-to-face interventions are not feasible (e.g., geography limits access to other forms of treatment) or when patients decline more traditional mental health interventions. It has also been suggested that web-based interventions may provide more confidentiality than more traditional approaches.

   c. Providers should regularly encourage patients to complete the intervention and endeavor to maintain and strengthen the therapeutic relationship, build patient rapport, stress practice and assignment completion, and ensure adequacy of safety protocols. Availability of telephone contact for initial assessment or other reasons (e.g. emergencies, suicidality/homicidality, or follow-up of specific problems) should be considered.

   d. Providers using technology-assisted interventions should take steps to ensure that their work complies with the regulations and procedures of the organization in which they are employed, legal standards, and the ethical standards of their professions. Patient confidentiality and safety should be monitored closely.

DISCUSSION

B1. Therapies that More Strongly Emphasize Cognitive Techniques (CT)

Cognitive therapy (CT) techniques emerged principally from the work of Albert Ellis (1962) and Aaron Beck (1964). Initially manualized for the treatment of depression, CT techniques have been successfully adapted to the treatment of a diverse set of psychiatric disorders, including PTSD (Freeman & Datillo, 1992; Freeman et al., 1989; Scott et al., 1989) and have been manualized or packaged in various ways.

Several randomized controlled trials (RCTs) demonstrate the efficacy of CT techniques for a wide range of patients with PTSD, demonstrating its use in treating veterans with combat-related trauma, motor vehicle accident (MVA) survivors, sexual or physical assault victims, and victims of natural disasters. Most RCTs have examined CT as delivered in an individual therapy format, though some studies have investigated group-delivered CT.

For purposes of this guideline, the primary goal of CT techniques is to improve mood and behavior through a deliberate and explicit focus on modifying dysfunctional thoughts, beliefs, and expectations. In theory, while behavioral change is a desirable outcome of CT, the treatment components themselves do not explicitly or directly

target behavioral patterns per se (however, it appears that even cognitive interventions may involve exposure or behavioral components, for example, discussing the meaning of a traumatic event inevitably involves exposing oneself to the memories of that event). Likewise, while exposure-based interventions may result in altered cognitions, exposure therapies, per se, do not involve an explicit focus on cognitive restructuring procedures seen in CT. Nonetheless, in practice it is virtually impossible to conduct cognitive trauma-focused therapy without also involving behavioral or exposure-based components, as it is similarly virtually impossible to conduct behavioral or exposure-based therapy without involving cognitive therapy components.

CT is accomplished through a systematic and prescriptive process of (a) identifying dysfunctional beliefs, (b) challenging and disputing these beliefs by examining the evidence for or against them, and (c) restructuring or replacing these beliefs with those that are more functional, logical, and reality-based. According to theories on which CT is based, traumatic events may lead to distorted beliefs regarding personal safety, self-efficacy, relative danger, future consequences of actions, and availability of support. Over time, these maladaptive beliefs lead to or maintain symptoms of PTSD and impair global functioning. The goal of CT for PTSD is to correct these beliefs, which causes a decrease in symptoms and improves functioning.

The CT treatment protocol for PTSD typically begins with an introduction of how thoughts affect emotions and behavior. The cognitive model of change is introduced and the patient is given a detailed rationale and expectations for participation in therapy are established. Treatment interventions are focused on identifying and clarifying patterns of thinking. Several active techniques are used, such as capturing and recording thoughts about significant events, weighing the evidence in support of these thoughts, challenging distressing trauma-related thoughts, and replacing dysfunctional thoughts with more adaptive ones. Through systematic assignments both during and between therapy sessions, dysfunctional thoughts are examined, challenged, and replaced. As thoughts become more logical and reality-based, symptoms decrease and global functioning improves. CT also emphasizes the identification and modification of distorted core beliefs about self, others, and the larger world. CT teaches that improved accuracy of thoughts and beliefs about self, others, and the world leads to improved mood and functioning.

DISCUSSION

Randomized controlled trials (RCTs) have shown that CT alone is an effective intervention for patients with PTSD (Marks et al., 1998; Cottraux, 2008; Resick, 2008). CT is useful for identifying and modifying the many negative beliefs related to a traumatic experience and can be used effectively to reduce distressing trauma-related thoughts (e.g., about survival guilt, self-blame for causing the trauma, feelings of personal inadequacy, or worries about the future). Modifying thoughts about these and other trauma-related issues can reduce PTSD symptoms and improve mood and functioning. Numerous other trials support CT as a key component of combination treatments.

CT techniques are often delivered as part of treatment “packages” that usually include elements of exposure therapy, trauma-related education, and anxiety management. For example, Cognitive Processing Therapy, which has been manualized and validated for use with female sexual assault–related PTSD in women (Resick et al., 2002) and in veterans (Monson, 2006), combines aspects of CT and exposure therapy. CT can also be delivered in conjunction with a range of other psychological therapies (e.g., EMDR and psychodynamic therapy). CT techniques

may be an especially helpful treatment component when co-morbid depressive and/or anxiety disorders are present.

Contraindications for CT have not been empirically established, but may include psychosis, severe brain damage, or severe intellectual impairment.

Summary of Studies:

Twenty-one relevant clinical trials that evaluated the use of CT for PTSD were analyzed. The trials investigated the effect of CT compared with no-treatment conditions, such as placement on a waiting list, and compared with other therapies. Both single-session therapy and long-term therapy were studied, with the longest therapy lasting 30 weeks, plus additional sessions after the end of formal treatment. Although therapists trained in standardized CT methods provided treatment, the actual content of the therapy was often variable, as was the terminology used to describe it. In these studies, although the patients in the control groups and study groups generally improved over time, there was significantly greater improvement in most treated groups, compared with control groups. The studies that enrolled participants from the general population of PTSD patients examined a primarily female population. There was one clinical trial that enrolled male disaster workers, and one involving veterans.

Follow-up intervals ranged from immediate posttreatment to up to 2 years after completion of therapy. Patient retention rates were generally similar to those observed in studies of other types of therapy, but ranged from 52 percent to 100 percent; few studies were blinded, and most relied on self-reported symptom questionnaires to provide data for analysis.

Nine relevant randomized clinical trials compared the effect of CT with that of a nonactive treatment, such as waitlist control group, treatment as usual (TAU), or repeated assessment (Beck, et al., 2009; Classen, Koopman, Nevill-Manning, & Spiegel, 2001; Difede, et al., 2007; Duffy, Gillespie, & Clark, 2007; Ehlers, et al., 2005; Foa, Zoellner, & Feeny, 2006; Monson, et al., 2006; Sijbrandij, et al., 2007; Smyth, Hockemeyer, & Tulloch, 2008). Both group and individual CT appeared to be effective in reducing PTSD symptoms. This was seen for brief, limited treatment models, and for treatment programs taking several months to complete. Four studies compared the effect of CT with that of therapies described as support, supportive care, or Rogerian support therapy (Blanchard, et al., 2003; McDonagh et al., 2005; Foa, Zoellner, & Feeny, 2006; Cottraux, et al., 2008). In these trials, CT was reported to be superior to supportive care in reducing PTSD or in retaining patients in therapy. Notably, in the Cottraux study, there were more drop-outs from the Rogerian group due to worsening symptoms. Additionally, the CT group patients in this study demonstrated sustained improvements in PTSD symptoms at two years follow-up. Trauma-focused group CT and present-focused group therapy were compared in a single study of Vietnam veterans (Schnurr, et al., 2003). Approximately 40 percent of all participants showed significant change in PTSD symptoms, but neither treatment was superior to the other.

The Trauma-Adaptive Recovery Group Education and Therapy (TARGET) model was studied in a trial that compared it with CT in the treatment of substance abuse patients (Frisman et al., 2008). Some improvement in PTSD symptoms was noted in both groups, but TARGET therapy was reported to produce greater improvement in sobriety self-efficacy. One clinical trial (van Emmerik, et al., 2008) compared CT with a structured writing therapy that included three components: (a) writing in the first person, (b) cognitive self-reappraisal of the writing, and (c) farewell and sharing the

writing. The authors reported improvement in both study groups compared with a wait list group, but detected no differences in efficacy between them.

Recently, researchers have attempted to dismantle treatments to examine their efficacious components. Bryant et al. (2003b) reported that patients who received both CT and ET demonstrated less avoidance, depression, and catastrophic cognitions relative to patients who received ET only, while there was no difference in PTSD symptoms between the groups. In a later four arm study to try to determine what specific components of CBT were more effective, Bryant et al. (2008b) compared pure in-vivo exposure alone; pure imaginal exposure alone; pure in-vivo combined with imaginal exposure; and the combination of CR, in-vivo exposure, and imaginal exposure. The combined treatment was most effective; supporting the notion that effective therapy needs to include some element of cognitive restructuring; unfortunately, there was no CR only group. In addition, none of these treatment groups were reported to include relaxation, breathing retraining, or other stress modulation techniques that are a standard part of virtually all of the ET and CT packages.

Resick et al. (2008) found no difference between patients assigned to receive Cognitive Processing Therapy (CPT) and patients assigned to receive only the cognitive component of CPT. Interestingly, a third group that received only written narrative exposure without any of the other CPT techniques performed nearly as well ,with no significant difference compared with full CPT or the cognitive component by the time of the 6-month follow-up. In an attempt to isolate the active ingredients, McDonagh et al. (2005) compared a treatment combining exposure and cognitive therapy elements to both a waitlist control group and a group given Present-Centered Therapy (PCT), a form of problem-solving therapy designed to eliminate the active ingredients found in CBT. Both treatment groups demonstrated improved symptoms over the waitlist control group but did not differ between themselves.

Bisson (2007) performed a systematic review of the randomized trials of all psychological treatments (Cochrane Collaboration Report). Treatments were categorized as trauma-focused cognitive behavioral therapy/exposure therapy (TFCBT); stress management (SM); other therapies (supportive therapy, non-directive counselling, psychodynamic therapy and hypnotherapy); group cognitive behavioural therapy (group CBT); and eye movement desensitization and reprocessing (EMDR). The results showed that TFCBT did significantly better than waitlist/usual care, and other therapies. Stress management did significantly better than waitlist/usual care. There were no significant differences between TFCBT and SM, and there was no significant difference between other therapies and waitlist/usual care control. Group TFCBT was significantly better than waitlist/usual care. EMDR did significantly better than waitlist/usual care and other therapies. There was no significant difference between EMDR and TFCBT or SM.

Conclusions:

There is good evidence that individual CT is effective in reducing PTSD symptoms, and limited evidence that treatment gains persists for up to 2 years. Additional research is needed to demonstrate the efficacy of CT delivered in a group format. Given the contrasting outcomes of available studies comparing combinations and dismantling components, there are insufficient data to conclude that CT is superior to ET at this time.

EVIDENCE TABLE

	Evidence	Sources	LE	QE	SR
1	CT is effective with civilian men and women exposed to combat and non-combat trauma	Bryant et al., 2003b Bryant et al., 2008b Cottraux et al., 2008 Difede et al., 2007 Duffey et al., 2007 Ehlers et al., 2005 Foa et al., 2005 Lovell, et al., 2001 Marks et al., 1998 Sijbrandij et al., 2007 Smyth, Hockemeyer, & Tulloch, 2008 vanEmmerik, Kamphuis, & Emmelkamp, 2008	I	Good	A
3	CT is effective in treating co-morbid substance abuse and PTSD	Frisman et al., 2001	I	Poor	C
4	CT is effective in treating PTSD in motor vehicle accident survivors	Blanchard et al., 2003	I	Mod	B
5	CT is effective in treating PTSD in a group format	Beck et al., 2009	III	Poor	I
2	CT is effective with military and veterans with combat- and non-combat-related PTSD.	Monson et al., 2006	I	Good	B
3	CT is effective for women with PTSD associated with sexual assault.	Chard, 2005 Foa et al., 2004 Foa, Zoellner, & Feeny, 2006 McDonagh et al., 2005 Resick et al., 2002 Resick et al., 2008	I	Good	A

QE = Quality of Evidence;SR =Strength of Recommendation (see Appendix A)

B2. Exposure Therapy (ET)

Exposure therapy protocols have a high level of evidence for treatment of PTSD, and generally include the components of psychoeducation, imaginal or narrative exposure, in-vivo exposure, and processing of thoughts and emotions. The most commonly used protocol is Prolonged Exposure (PE), although various other exposure protocols have been used. Protocols that provide only a portion of these components (e.g. in-vivo exposure or imaginal exposure in isolation) show less robust effect sizes (e.g., Bryant, 2008b). Imaginal exposure involves encouraging the patient to revisit the experience in imagination, and recalling the experience through verbally describing the physical and emotional details of the trauma. In vivo exposure involves asking the patient to physically confront realistically safe but still feared stimuli (e.g. driving a car after having been in a serious motor vehicle accident). In vivo exposure is typically arranged in a hierarchical order based on the perceived difficulty of confronting each stimulus. In addition, each item on the hierarchy may be titrated to make it more or less difficult depending on the patient's progress in treatment. In the preceding example the patient might first sit in a car in the passenger seat, and then in the driver's seat, and then start the car, etc. The patient repeats each situation until a reduction in the intensity of emotional and

physiological response is achieved, at which point they move on to the next item in their hierarchy.

DISCUSSION

RCTs have shown that Exposure Therapy (ET) helps men and women with PTSD symptoms. RCTs of ET have demonstrated its efficacy in female victims of sexual and non-sexual assault, motor vehicle accidents, male combat-related trauma, war refugees, and mixed trauma populations. Several studies indicate that results are highly comparable between exposure therapy and other forms of trauma focused cognitive behavioral therapy (e.g., cognitive therapy, EMDR, stress inoculation training). Findings regarding efficacy in (mostly Vietnam) combat veterans in VA clinical settings are less consistent and the degree of improvement in PTSD symptoms may be less pronounced, although the number of studies are very limited; preliminary data suggest it is efficacious (Rauch et al., 2009).

The mechanism of ET is thought to be related to a reduction in negative emotions (fear, anxiety, sadness, guilt) associated with their experience through repetitive, therapist-guided confrontation of feared places, situations, memories, thoughts, and feelings. ET usually lasts from 8 to 15 sessions depending on the trauma and treatment protocol. In the most common form of ET, Prolonged Exposure therapy patients are repeatedly exposed to their own individualized trauma stimuli, until their arousal and emotional responses are consistently diminished. However, there are various ways in which ET is packaged. ET providers can vary the pacing and intensity of exposing patients to the most difficult details of their trauma based on the patient's emotional response to the trauma and to the therapy itself.

Several studies indicate that results are comparable between exposure with other forms of cognitive behavioral therapy (e.g., cognitive therapy, EMDR, stress inoculation training, or combinations). Variations on exposure therapy that have promising results include written exposure and exposure in the context of a broader narration of the patient's life. For example, in a three arm dismantling RCT by Resick et al. (2008), written exposure was compared directly with CPT (without the written exposure component), and the full CPT program (including written exposure). Treatment sessions for the written exposure only group consisted of two one-hour sessions to provide overview of treatment and education, followed by five two-hour sessions where the patient was asked to write for approximately 60 minutes alone about their worst traumatic event, followed by reading this to the therapist who provided supportive feedback without any of the cognitive restructuring techniques. The written exposure group did nearly as well as both of the CPT treatment arms (which consisted of 12 one-hour sessions), and on the six month follow-up there was no significant difference between the three groups. This finding was replicated using very different methods in a study by van Emmerick, et al. (2008) who found that a structured writing therapy was equivalently efficacious in the treatment of PTSD as cognitive therapy when both were compared with a no treatment control group. These data strongly support the notion that a systematic writing narrative process with therapist involvement may be just as effective in alleviating symptoms as any of the more widely used cognitive therapy techniques.

Oral narrative therapy has also been shown to be highly effective in treating PTSD in war-ravaged refugee populations. In one study of Rwandan refugees with PTSD and severe war-related trauma (Neuner, 2008), lay counselors had patients construct a narration of their life from birth to the present while focusing on detailed exploration of specific traumatic experiences. This resulted in significant improvement in PTSD symptoms, with effects comparable to any of the most cited CPT or PE studies in

U.S. or European clinical samples. Increasingly, virtual (computer based) exposure techniques and strategies are being utilized to accomplish exposure therapy. However, to date, there are no randomized studies of virtual reality compared with either wait list or standard exposure techniques that confirm its efficacy.

Another mode of delivery of exposure therapy that has been found to be effective in two RCTs compared with wait-list control group is Brief Eclectic Psychotherapy developed by researchers in the Netherlands. This treatment includes imaginal exposure combined with relaxation, writing assignments, use of mementos from the traumatic experience, exploration of meaning, a farewell ritual, and psychoeducation (Gersons, 2000; Lindauer, 2005).

There have, as yet, been no randomized trials comparing ET with pharmacotherapy, either alone or in conjunction with one another. However, two trials have examined augmentation strategies. In one trial, the addition of ET following 10 weeks of sertraline resulted in reduction in relapse and additional symptom reduction in those patients who either failed to initially respond or partially responded to sertraline (Rothbaum et al., 2006). In a second study, augmentation with paroxetine for patients who partially responded to 6 sessions of ET did not result in additional benefit (Simon et al., 2008).

As with any treatment, patients need to be screened for their suitability prior to undergoing ET as it may temporarily increase their level of distress. Patients living with the threat of domestic violence should not be considered for ET until their security can be assured. ET has not been studied in patients with health problems that preclude exposure to intense physiological arousal. Therefore, providers should use caution when considering ET for patients with current significant suicide risk, substance dependence, or current psychosis and especially in the elderly. Providers should be aware of the possibility of increased distress as patients confront trauma memories and reminders. As in all PTSD treatments, providers must take concrete steps to prepare patients for the treatment (e.g., present clear rationale, explore patient concerns, encourage realistic expectations, and build commitment to the therapy) in order to reduce the risk of dropout.

EVIDENCE TABLE

	Evidence	Sources	LE	QE	SR
1	ET is effective in the treatment of PTSD (compared to wait list, present centered therapy, and other control comparisons)	Basoglu ,2005, 2007 Cloitre, 2002 Cooper et al., 1989 Feske, 2008 Foa et al., 1991 & 1999a Ironson et al., 2002 Keane et al., 1989 Marks et al., 1998 McDonah, 2005 Neuner, 2004, 2008 (life narration), Schnurr,2007 Tarrier et al., 1999 Gersons et al., 2000 Lindauer et al., 2005	I	Good	A

2	ET compared to other forms of therapy shows equivalent results	Bryant, 2003b Bryant, 2008b Foa et al., 1991 & 1999a Foa, 2005 Marks et al., 1998 Paunovic & Ost, 2001 Power, 2002 Resick ,2002 Resick & Nishith, 2001 Resick, 2008 (written exposure) vanEmmerik, 2008 (written exposure) Rothbaum, 2005 Schnurr, 2001 Tarrier et al., 1999	I	Good	A

QE = Quality of Evidence; R = Recommendation (see Appendix A)

B3. Stress Inoculation Training (SIT)

Several therapy protocols have been developed that focus on anxiety management and coping skills training, including Stress Inoculation Training and Relaxation Training. Stress inoculation training (SIT), is presented as a tool box or set of skills for managing anxiety and stress (Hembree & Foa, 2000). This treatment was originally developed for the management of anxiety symptoms and adapted for treating women rape trauma survivors. SIT typically consists of education and training of coping skills, including deep muscle relaxation training, breathing control, assertiveness, role playing, covert modeling, thought stopping, positive thinking and self-talk, and in-vivo exposure. The rationale for this treatment is that trauma related anxiety can be generalized to many situations (Rothbaum et al., 2000). The Expert Consensus Guideline Series: Treatment of Post-traumatic Stress Disorder notes that anxiety management is among the most useful psychotherapeutic treatments for patients with PTSD (Foa, Davidson et al., 1999a). A Cochrane meta-analysis found that stress management protocols were as effective as other TF-CBT interventions and EMDR. Relaxation protocols that do not include all of the SIT components have also demonstrated very encouraging results in several studies (Marks et al., 1998; Taylor et al., 2003; Vaughn et al., 1994).

SIT is designed to “inoculate” people with PTSD from heightened stress responses through teaching anxiety management skills which can include:

- Relaxation training: teaching patients to control fear and anxiety through the systematic relaxation of the major muscle groups
- Breathing retraining: teaching slow, abdominal breathing to help the patient relax and/or avoid hyperventilation with its unpleasant and often frightening physical sensations
- Positive thinking and self-talk: teaching the person how to replace negative thoughts (e.g., ‘I’m going to lose control’) with positive thoughts (e.g., ‘I did it before and I can do it again’) when anticipating or confronting stressors. This is often combined with in-vivo exposure
- Assertiveness training: teaching the person how to express wishes, opinions, and emotions appropriately and without alienating others

- Thought stopping: distraction techniques to overcome distressing thoughts by inwardly shouting 'stop' (Foa et al., 1999b)

Many SIT protocols also include cognitive restructuring and other elements of exposure therapy.

DISCUSSION

There have been two RCTs that have evaluated SIT and both found SIT to be effective with women who have survived sexual assault. A study by Foa and colleagues (1991) with 45 female sexual assault victims compared SIT, Prolonged Exposure (PE) (see Annotation B2), Supportive Counseling (SC) and wait list control. SIT was found to be the most effective treatment for short-term symptom improvement and both SIT and PE were effective for long term improvement with PE superior to SIT. Rothbaum (2001) reports that the "results suggested that all conditions produced improvement on all measures immediately post-treatment and at follow-up. At follow-up, clients who received PE continued to improve after treatment termination, whereas clients in the SIT and SC conditions evidenced no change between post-treatment and follow-up." Another study with 96 female sexual assault victims compared SIT, PE, combined SIT and PE, and wait list controls (Foa et al., 1999a). The study found that all treatments were better than wait list control for ameliorating PTSD severity at post-treatment and at the 6-month follow-up. Interestingly, although all three treatments were effective, the combined treatment was not superior to either SIT or PE alone. Although this may be related to the fact that clients in the combined treatment group received less PE-or SIT-specific techniques than participants in the individual treatments, the most likely explanation presented in the paper was an uncharacteristically low drop-out rate that happened to occur in the PE-only group.

A study of 15 women by Kilpatrick et al. (1982) found SIT to be effective in reducing rape-related fear and anxiety.

Motor vehicle accident survivors (Hickling & Blanchard, 1997) had a 68 percent reduction of PTSD symptoms after involvement in a modified version of Foa et al.'s SIT/PE combination program.

A controlled study comparing three different forms of relaxation (relaxation, relaxation plus deep breathing, and relaxation plus deep breathing plus biofeedback) for 90 Vietnam veterans found that all treatments were equally effective in leading to improvement (Watson et al., 1997).

Vaughn, et al. (1994) found that relaxation training was superior to waitlist. Taylor et al (2003) also found support for reduction of PTSD with a relaxation protocol though the effects were less than for ET. In a head-to-head comparison study by Marx (1998), relaxation training produced was nearly equivalent to PE.

EVIDENCE TABLE

	Evidence	Sources	LE	OE	SR
1	SIT is effective in the treatment for PTSD.	Foa et al., 1999a Foa et al., 1991 Kilpatrick et al., 1982 Rothbaum, 2001	I	Good	A

LE=Level of Evidence QE=Quality of Evidence; SR= Strength of Recommendation (see Appendix A)

B4. Eye Movement Desensitization and Reprocessing (EMDR)

Eye Movement Desensitization and Reprocessing (EMDR) is a psychological treatment designed to alleviate the distress associated with traumatic memories (Shapiro, 1989a, 1989b). The objective of EMDR is to assist patients to access and process traumatic memories while bringing them to an adaptive resolution (Shapiro, 2001).

In EMDR, the therapist collaborates with patients to: (1) access a disturbing image associated with the traumatic event; (2) solicit the experience of body sensations associated with the disturbing image; (3) identify an aversive self-referring cognition (in concise words) that expresses what the patient "learned" from the trauma, and (4) identify an alternative positive self-referring cognition that the patient wishes could replace the negative cognition. The patient is then asked to hold the disturbing image, sensations, and the negative cognition in mind while tracking the clinician's moving finger back and forth in front of his or her visual field for about 20 seconds. In successive tracking episodes, the patient concentrates on whatever changes or new associations have occurred. Eye movement episodes are repeated until there are no new associations. Subsequent tracking episodes attempt to replace the negative cognitive self-statement with the alternate positive cognition.

Between sessions, the patient is directed to keep a journal of any situations that provoke PTSD symptoms and of any new insights or dreams about the trauma. The number of sessions is dependent upon observed improvements and the number of traumatic events experienced.

Within a session, standard self-rating scales document changes in the intensity of the symptoms and the negative cognition, and the patient's acceptance of the alternative positive cognition. The patient reports following each set of eye movement episode to inform the therapist of the strength of both negative and positive cognitions; changes in cognitions, the images, emotions, or body sensations.

EMDR protocols allow for substitution of left-right alternating tone or touch as modifications to the use of the eye movements, suggesting that it is not the eye movements per se, but rather side to side alternating stimulation that is sought. Studies attempting to ascertain the relative contribution of the eye-movement component suggest that comparable outcomes are attained with or without eye movements. These findings are seen as indicating that this aspect (i.e., eye-movements or alternating stimulation of any type) of the treatment protocol may not be critical components.

Given the success of EMDR and the lack of support for the alternating stimulation components, many theorists are considering the active ingredients for the observed treatment gains. Specifically, EMDR is gaining acceptance as a treatment that shares components with other existing, successful treatments. Derived from desensitization strategies, EMDR counters avoidance of the traumatic memories and related cues by repeatedly accessing the aversive traumatic images themselves, promotes emotional processing by soliciting the emotional responses attendant to the aversive memories; identifies a novel and alternative view of the traumatic experience in conjunction with the patient, and then challenges the patient to consider the validity or accuracy of the alternative perspective. With the focus upon physiological arousal and reactivity, EMDR as a desensitization treatment also provides a component of arousal management that is inherent in the treatment. Thus, EMDR at its most basic level incorporates components of a) exposure to trauma related cues; and b) processing of emotional responses. Each of these EMDR components involves efforts

to mitigate strategic avoidance reactions theoretically viewed as maintaining current symptomatology. EMDR also includes: c) elements of corrective and rational restructuring of the patient's views of the traumatic event; and d) self monitoring of cognitive and emotional responses that are often viewed as key homework components of cognitive behavior therapy in general, and e) a focus on heightened physiological arousal and reactivity.

DISCUSSION

EMDR possesses efficacy for treating patients with PTSD: this conclusion is based upon a thorough review of the literature in the treatment guidelines generated by a task force for the International Society for Traumatic Stress Studies (Spates et al., 2009) as well as by Division 12 of the American Psychological Association (APA) and a Cochrane review (Bisson 2007). The United Kingdom's NICE Guidelines for PTSD (2005) also recommend EMDR as a treatment, supported by multiple efficacy studies. While the results of numerous controlled published studies found medium to large effect sizes for EMDR, there is no evidence that EMDR more efficient or rapid than other forms of cognitive behavioral treatment. Similarly, suggestions that EMDR is more easily tolerated than other psychological treatments remain unsupported empirically.

Results of clinical trials, meta-analytic studies, review articles, and extant practice guidelines suggest that EMDR successfully treats symptoms of PTSD when compared to no treatment or delayed treatment conditions. When compared to other treatment modalities, most studies reviewed indicated that EMDR possessed comparable efficacy to other well-accepted cognitive behavioral treatments to include stress inoculation training (SIT) and exposure therapies.

Maxfield and Hyer (2002) conducted a meta-analysis involving comparisons of EMDR against wait list controls, cognitive behavior therapy involving exposure, and treatment modalities described as other than CBT. Results indicated superiority of EMDR to the wait list control condition. Also, the authors found an overall superiority of EMDR compared to the other active treatment conditions, though they noted sufficient variability that they judged the summed results to indicate comparable vs. superior effectiveness of EMDR over other treatments.

Four studies specifically compared EMDR with Exposure Therapy (Lee et al., 2002; Power et al., 2002; Rothbaum, et al., 2005; and Taylor et al., 2003). Lee et al. (2002) and Power et al. (2002) found that EMDR had equivalent or better results than CBT and was more efficient in that it worked faster. Taylor et al. (2003) did not observe differential efficiency in their trial, but they also used therapist-assisted *in vivo* work plus imaginal work. Rothbaum, et al. (2005) found symptom improvement at post-test to be equivalent between EMDR and prolonged exposure. She writes in the abstract, "PE and EMDR did not differ significantly for change from baseline to either posttreatment or 6-month follow-up measurement on any quantitative scale." Although a measure termed "end-state functioning" was described as favoring PE, this was a composite variable derived from three of the individual clinical scales that were listed as primary outcomes, and it is likely that this variable had the effect of magnifying the small non-significant differences on these individual scales when they were combined.

Criticisms of EMDR stem from its theoretical premises to the necessity of its components to achieve the desired outcome. There is limited support provided by a set of seven studies that the inclusion of eye movement is beneficial, but most of these are studies with analog populations, or in clinical populations exposed to a traumatic event, but who didn't necessarily develop full clinical PTSD (Andrade et al.,

1997; Barrowcliff et al., 2004; Christman and Garvey, 2000; Kavanaugh et al., 2001; Kuiken et al., 2001-2002; Sharpley et al., 1996; Wilson, Silver, Covi, & Foster, 1996; and van den Hout et al., 2001). In aggregate, the data do not suggest that eye movements or other form of kinesthetic stimulation are necessary. Spates et al. (2009) reviews aptly the literature on dismantling studies in EMDR and concludes that "the best provisional conclusion so far is that the bilateral stimulation component of EMDR does not incrementally influence treatment outcome". Notwithstanding the lack of necessity for eye movements, when viewed within the framework of all other trauma-focused CBTs, EMDR is equivalent.

There may be some basis for or against recommending this treatment depending upon the type of trauma leading to PTSD. Specifically, studies of EMDR efficacy with combat veterans have demonstrated variability, with several authors suggesting that the treatment may be less than optimal for this condition (Boudewyns et al., 1993; Jensen, 1994), and other studies suggesting the opposite (Carlson et al., 1998; Devilly et al., 1999). However, it should be noted that only two of the cited studies had a full course of treatment – all the others were short duration studies. Studies of other CBT modalities and SSRIs have also shown inconsistent results in combat veterans, and thus, based on current evidence, there is no reason to believe that EMDR would not be as effective as other trauma-focused CBTs in this population.

Overall, there are rigorously controlled studies to support the conclusion that EMDR is effective in the treatment for PTSD.

EVIDENCE TABLE

	Recommendation	Sources	LE	QE	R
1	EMDR is an effective treatment for PTSD (compared with wait-list, routine care, and active treatment controls)	Chemtob et al., 2000 Davidson & Parker, 2001 Maxfield & Hyer, 2002 Sheppard et al., 2000 Van der Kolk et al., 2007	I	Good	A
2	Eye movements are *not* critical to the effects of EMDR	Davidson & Parker, 2001 Spates et al., 2009	I	Fair	B
3.	EMDR compared with ET show consistent comparable results	Davidson & Parker, 2001 Foa & Meadows, 1997 Ironson et al., 2002 Lee et al., 2002 Power et al., 2002 Rothbaum et al., 2005 Servan-Schrieber, 2000 Sheppard et al., 2000 Taylor et al., 2003 Van Etten and Taylor, 1998	I	Good	A

LE=Level of Evidence QE=Quality of Evidence; SR= Strength of Recommendation (see Appendix A)

B5. Imagery Rehearsal Therapy (IRT)

Occurrence of nightmares as a problem is frequent (4 to 8 percent in the general population and 60 percent in PTSD). Evidence shows that nightmares are associated with psychological distress and sleep impairment. A conditioning pattern similar to classic psychophysiological insomnia is produced in the nightmare-disturbed loop, along with the negative cognition of "fear of going to sleep." Studies using brief CBT (desensitization and imagery rehearsal) have demonstrated a large reduction in

nightmares. Many studies, including Forbes et al. (2001), suggest that PTSD is associated with a propensity toward image, particularly where the post-traumatic symptom picture is characterized by nightmares and flashbacks. IRT incorporates a system to increase the imagery control.

IRT is aimed at changing the content of the patient's nightmares to promote mastery over the content-threat, thereby altering the meaning, importance, and orientation to the nightmare. IRT includes elements of 1) psychoeducation about nightmares, insomnia, and PTSD; 2) positive coping skill building (thought stopping, breathing, grounding, writing/talking about issues and others); 3) cognitive restructuring; 4) sleep hygiene, stimulus control, and sleep restriction; and 5) focused use of pleasant imagery to replace negative imagery in recurrent nightmares. While discussion of trauma imagery occurs, the model includes a de-emphasis of discussion of this content in group sessions. The model has been tested primarily in a group format.

DISCUSSION

Several studies have examined IRT with some promising results. While not with a primary PTSD population, Krakow et al. (1995) studied 58 chronic nightmare sufferers who were randomly assigned to a treatment group (n = 39) or a wait list control group (n = 19). The IRT group demonstrated significant reductions in nightmares and improved sleep quality. Further, reduction in nightmares was a significant predictor of improvement in sleep. The authors concluded that for some chronic sufferers, nightmares may be conceptualized as a primary sleep disorder that can be effectively and inexpensively treated with CBT.

Krakow et al. (2001a) randomly assigned 168 female survivors of sexual assault (95 percent of the sample met the criteria for PTSD) to receive IRT (n = 88) or wait list (n = 80) and found that among completers, those women assigned to IRT had a larger reduction in self-reported PTSD severity at the 3-month follow-up than wait list. Further, the impact of nightmares was reduced and sleep quality improved. In a pilot study of IRT with crime survivors with PTSD, Krakow et al. (2001b) reported significant reductions in nightmares, improved sleep, and reduced PTSD severity at the 3-month follow-up.

Forbes et al. (2001) completed an open trial of group IRT with 12 Vietnam veterans with combat-related nightmares and PTSD. Veterans reported significant reduction in nightmare frequency and intensity for the target nightmare. In addition, self-reported PTSD symptoms were significantly reduced. Follow-up data demonstrated maintenance of gains at 12 months following the conclusion of treatment (Forbes et al., 2003).

A recent large RCT comparing IRT with a group nightmare management treatment (N=124) among Vietnam veterans with PTSD (receiving treatment in a VA clinical setting) found that neither treatment produced significant or sustainable improvement in overall PTSD symptom severity, nightmare frequency or sleep quality (Cook et al, 2010). While much of the research to date has focused on IRT, other versions of nightmare reduction programs, such as Emotional Relaxation and Rescripting, are under empirical examination.

EVIDENCE TABLE

	Recommendation	Sources	LE	QE	SR
1	IRT can be considered for treatment of PTSD (nightmare and sleep disruption, in particular)	Krakow et al., 1995, 2001a 2001b Forbes et al., 2001, 2003 Cook et al., 2010	I II-1 I	Fair	C

LE=Level of Evidence QE=Quality of Evidence; SR= Strength of Recommendation (see Appendix A)

B6. Psychodynamic Therapy

BACKGROUND

In 1895, Joseph Breuer and Sigmund Freud based their *Studies on Hysteria* on the proposition that traumatic life events can cause mental disorder (Breuer & Freud, 1955). This principle, radical for its time, grew in scope and application over the next century and strongly influenced military psychiatry in World War I (Kardiner, 1941; Rivers, 1918) and World War II (Grinker & Spiegel, 1945). Psychodynamic principles were later applied to the psychological problems of Holocaust survivors (Krystal, 1968; De Wind, 1984), Vietnam veterans (Lindy, 1996), rape survivors (Rose, 1991), adult survivors of childhood sexual trauma (Courtois, 1999; Roth & Batson, 1997; Shengold, 1989), and survivors of other traumatic events (Horowitz, 1997). Psychodynamic ideas have also helped providers manage the sometimes complex issues that may surface in the relationship between survivor and psychotherapist (Pearlman & Saakvitne, 1995; Wilson & Lindy, 1994). Psychodynamic psychotherapies operate on the assumption that addressing unconscious mental contents and conflicts (including those that may have been blocked from consciousness as part of a maladaptive response) can help survivors cope with the effects of psychological trauma. Psychological meanings of post-traumatic responses are explored by examination of the fears, fantasies, and defenses stirred up by the traumatic event.

DISCUSSION

Individual case reports comprise the bulk of the psychodynamic literature on the treatment of psychological trauma, but a small group of empirical investigations are available to support recommending that 2 psychodynamically informed treatments can be considered as treatment options for PTSD.

Three RCTs have supported the efficacy of Gersons's Brief Eclectic Psychotherapy for reducing PTSD symptoms in police (Gersons et al., 2000) and community patients with PTSD (Lindauer et al., 2005). This 16-week individual psychotherapy includes both CBT (e.g., psychoeducation, imaginal exposure, cognitive restructuring) and psychodynamic elements (focus on shame and guilt, attention to the patient-therapist relationship) and a farewell ritual at the end of treatment. At present, it is unclear which elements of treatment are responsible for the improved outcomes.

Brom and colleagues (1989) conducted a RCT that compared Horowitz's (1976) Brief Psychodynamic Therapy to hypnotherapy, trauma desensitization, and a wait list control group in the treatment of PTSD. They found that symptoms of intrusion and avoidance improved significantly in each of the treatment groups but not in the control group; no differences across the three treatments were observed.

While research evidence and clinical experience suggest that psychodynamic psychotherapy can be effectively combined with other forms of psychotherapy and with psychopharmacological interventions for depression (DiMascio et al., 1979; van Praag, 1989), this approach has not been sufficiently researched in work with PTSD.

Psychodynamic ideas have, in some instances, been misapplied in clinical work with trauma survivors, giving rise to concern about the creation or elaboration of so-called *false memories* (Roth & Friedman, 1997). It may be that trauma survivors are particularly prone to this phenomenon, given their tendency towards dissociation. It is important that clinicians be properly trained before undertaking psychodynamic treatment of trauma survivors.

Because of its focus on basic problems in interpersonal relationships, psychodynamic psychotherapy may be useful in working with patients with complex PTSD. Clinical case studies suggest that psychodynamic psychotherapy may be of particular value in work with adult survivors of childhood sexual abuse (Courtois, 1999; Roth & Batson, 1997; Shengold, 1989).

EVIDENCE TABLE

	Recommendation	Sources	LE	QE	SR
1	Some forms of psychodynamic psychotherapy can be considered for the treatment of PTSD	Brom et al., 1989 Gersons, Carlier, Lamberts, & van der Kolk, 2000 Lindauer et al., 2005	I	Fair	C
2	Psychodynamic psychotherapy for patients with co-morbidity	Courtois, 1999 Roth & Batson, 1997 Shengold, 1989	II-2	Fair	C

LE=Level of Evidence QE=Quality of Evidence; SR= Strength of Recommendation (see Appendix A)

B7. Patient Education

BACKGROUND

Education of the trauma survivor is a core part of all PTSD treatments. Survivors need to better understand what they are experiencing, how to cope with reactions or symptoms, and what happens in treatment. It is also helpful to provide this information to family members or to the patient's significant others so that they can more effectively support the patient's recovery.

DISCUSSION

PTSD education involves teaching the survivor to label, recognize, and understand PTSD symptoms (and other trauma-related problems) that he or she is experiencing, providing simple advice regarding coping, explaining what he or she can do to facilitate recovery, and describing treatment options. Education can help make symptoms more understandable and predictable, decrease fear of symptoms, increase awareness of coping options, and help survivors decide whether to seek treatment or learn how to better participate in treatment.

Education should be one of the first steps of PTSD treatment. It can help establish the credibility of the treatment provider, make treatment seem immediately helpful to the patient, and help prepare the patient for next steps in treatment. In fact, education should continue throughout PTSD treatment, sometimes in brief discussions when the patient has questions and sometimes more systematically as a formal helping activity. It can be delivered to individuals or to groups. Because those with PTSD often have difficulties with concentration and memory, repetition of educational information and provision of written information are important.

The content of PTSD-related education can include the following topics:

1. *Nature of PTSD symptoms:* It is often useful to help the survivor identify and label the reactions that he or she may be experiencing, recognize that emotional and physical reactions are very common (and not dangerous), and understand that anxiety and distress are often "triggered" by reminders of the traumatic experience, which can include sights, sounds, or smells associated with the trauma; physical sensations (e.g., heart pounding); or behaviors of other people. However, it is important to include comments on positive steps

that the individual is taking, if appropriate, rather than providing a long list of possible symptoms for review. Patients can also benefit in understanding how PTSD symptoms have their basis in adaptive survival responses to life-threatening events.

2. *Practical steps to cope with trauma-related problems:* Survivors can also be educated about ways of coping with their PTSD symptoms in order to minimize their impact on functioning and quality of life. While education about coping is not a substitute for more systematic coping skills training, simple information can also be useful. Survivors can be helped to distinguish between positive and negative coping actions. Positive coping includes actions that help to reduce anxiety, lessen other distressing reactions, and improve the situation: relaxation methods, exercise in moderation, talking to another person for support, positive distracting activities, and active participation in treatment. Negative coping methods may help to perpetuate problems and can include continual avoidance of thinking about the trauma, use of alcohol or drugs, social isolation, and aggressive or violent actions.
3. *Nature of the recovery process and PTSD treatment:* Survivors will sometimes have unrealistic or inaccurate expectations of recovery and may benefit from understanding that recovery is an ongoing daily gradual process (i.e., it does not happen through sudden insight or "cure") and that healing does not mean forgetting about the trauma or having no emotional pain when thinking about it. Education about what happens in treatment is also important. This can help build motivation to participate or persist in treatment.

Despite the ubiquity of education in PTSD treatment and a strong clinical consensus as to the importance of such education, there is little evidence bearing on its impact on chronic PTSD. Education has usually been a component of empirically supported treatments, but it has not been carefully evaluated as a "stand-alone" treatment (nor is it intended to be delivered in the absence of other treatment elements).

Psychoeducation was one of several components in each study, and the effect of the psychoeducation component per se thus cannot be evaluated. There is, therefore, insufficient evidence to conclude that psychoeducation alone is an effective treatment for PTSD.

Three studies (Krupnick, 2008;Wallis, 2002; and Weine, 2008) compared group interventions containing a psychoeducation component with WL. There were 9, 12, and 16 sessions, and the sample sizes were 48, 83, and 166. Although each intervention contained a psychoeducational component, the focus and content of the group sessions differed across studies. In 2 studies, the group intervention decreased PTSD symptoms compared with WL, while in the third; PTSD symptoms were only evaluated as a mediator for effects on access to mental health services. No study included a control condition for the psychoeducation component.

EVIDENCE

	Recommendation	Sources	LE	QE	R
1	Psychoeducation is recommended as a component of PTSD treatment	Foa et al., 1999 Lubin et al., 1998 Krupnick et al., 2008 Wallis, 2002) Weine et al., 2008	III II-2	Poor Fair	C

LE=Level of Evidence QE=Quality of Evidence; SR= Strength of Recommendation (see Appendix A)

B8. Group Therapy

BACKGROUND

The most comprehensive critical review of group therapy approaches for the treatment of PTSD is Tracie Shea and colleagues' chapter within the second edition of the practice guideline *Effective Treatments for PTSD* (Shea, McDevitt-Murphy, Ready, & Schnurr, 2009). Shea et al.'s discussion builds upon the previous edition's chapter by David Foy and colleagues (Foy, Glynn, Schnurr, Jankowski, Wattenburg, Weiss, Marmar, & Gusman, 2000).

Shea and colleagues briefly review the use of group therapy for PTSD and note that there is no empirical support for the belief that group treatment is superior to individual treatment for trauma. The authors highlight potential benefits in Foy et al.(2000) of using a group format, including efficiency in treatment provision and development of support and understanding between group members that may counteract isolation and alienation. They also distinguish group treatment approaches by their emphasis on reintegration of the traumatic experience as an integral change process. Trauma-focused groups assume integration of the traumatic memory and modify the meaning of the trauma for the individual, while present-centered supportive approaches aim to decrease isolation and increase sense of competence. Shea et al. (2009) note that a focus on trauma may or may not be reflected within any one of the various theoretical orientations of therapy utilized in group approaches for PTSD, with the exception of supportive therapy, which tends to avoid direct focus on trauma material.

Shea et al. (2009) characterize three overarching group therapy orientations: Psychodynamic/Interpersonal/Process, Supportive, and Cognitive Behavioral. Most groups share common strategies designed to provide a sense of safety, trust, and develop cohesion among members. The three approaches do, however, differ in significant ways in terms of techniques and strategies used (See Table I-5).

Foy and colleagues (2000) summarized factors identified in the literature as important considerations for group treatment in general, including:

- Flexibility in personal schedule
- Ability to establish interpersonal trust
- Prior group experience, including 12-step groups
- Completion of a preparatory course of individual therapy
- Similar traumatic experiences with other group members
- Compatibility for gender, ethnicity, and sexual orientation
- Willingness to abide by rules of group confidentiality
- Not severely paranoid or sociopathic
- Stable living arrangement

The value and necessity of these factors, however, have not been examined empirically. Although most studies of group treatment for PTSD do focus on a particular trauma type, the importance of homogeneity of groups in terms of trauma type is an unanswered question. Trial participants in studies reviewed herein commonly lacked previous individual or group therapy experience. Contraindications for group therapy and exclusion criteria for trials of group treatment are usually similar and include active psychosis, cognitive deficits, and current suicidal or homicidal risk (Shea et al., 2009).

Indications for Trauma Focus versus Supportive Groups (from Foy et al., 2000)

- Individual can tolerate high anxiety arousal or other strong affects
- No active suicidality or homicidality
- Substance abuse or other co-morbidities are under control
- Individual accepts rationale for trauma-uncovering work
- Willingness to self-disclose personal traumatic experiences
- No current life crises

Table I - 5 Group Therapy in PTSD (Shea et al., 2009)

Approach	Techniques/Strategies
Supportive groups (Present-focused)	- Aim to enhance daily functioning through provision of safety, trust, acceptance, and normalization of symptoms and experiences - Help individuals develop sense of mastery over problems via group feedback, emotional support and reinforcement of adaptive behaviors - Focus on current life issues rather than traumatic experiences
Psychodynamic/ Interpersonal Process (Trauma-focused)	- Facilitate insight-based learning and change - When an explicit focus on trauma is present, trauma material arises in a less structured manner or covertly, and emphasis is on increasing awareness of unconscious fears and maladaptive patterns - Emphasize understanding the meaning of the trauma symptoms - Help individuals gain insight and make connections into how current difficulties may be linked to the trauma - The Interpersonal Therapy (IPT) model helps groups members identify their specific relationship difficulties and behavioral patterns that promote poor functioning - "Process" groups maintain emphasis on the immediate present experience of the individual, their feelings and needs, and their interactions with other members
Cognitive-behavioral therapy groups (Trauma focused)	- Include psychoeducation on trauma and skills training to manage anxiety and arousal - Trauma is directly addressed via repeated imaginal exposure techniques in session and having individuals listen to audio recordings of their trauma experiences as homework between sessions - Maladaptive thoughts and beliefs are identified and modified or restructured - In final sessions, relapse prevention strategies are planned and coping skills reviewed

Although many of the studies of PTSD group treatment reviewed excluded participants with active substance use disorders (SUD), others did not and several specifically targeted co-morbid PTSD and SUD. How SUD affects PTSD outcomes in group treatment has not been examined. Shea et al. (2009) point out that vicarious traumatization is a concern within trauma-focused groups but that no published evidence exists indicating that negative effects occur for some members in trauma-focused group treatment as a result of vicarious traumatization and systematic investigation of this possibility has not occurred.

RATIONALE

The empirical literature on group treatment for PTSD has grown since the publication of the first edition of the Treatment Guidelines for PTSD, although there remain methodological weaknesses in study designs, and there is no empirical evidence to

support a conclusion that group treatment is superior to individual treatment for trauma.

Nonetheless, it does appear that group-based treatment for individuals diagnosed with PTSD is associated with improvements in symptoms of PTSD, and there is growing belief that some unique attributes of the group treatment format provide benefits that are superior to individual treatment for trauma. Identified benefits include efficiency in treatment provision and development of support and understanding between group members that may counteract isolation and alienation.

DISCUSSION

Summary of Studies:

In their review, Shea and colleagues (2009) included studies published beginning in 1998 that targeted populations with trauma, assessed symptoms of PTSD at pre- and posttreatment, and had at least 10 participants in the group therapy being studied. Citing the small number of existing controlled studies, they did not require that studies include only participants meeting criteria for PTSD. Our search for relevant studies began with a review of studies included in Shea et al. (2009) and was limited to studies in which the sample participants met DSM criteria for PTSD and the active treatment was solely or predominantly in group format. Of the total 22 studies in Shea et al. (2009), we reviewed 14 that met these criteria, including six randomized and two nonrandomized trials comparing at least one active treatment group to a comparison or control condition, and six studies reporting pre- to posttreatment effects of a single group treatment condition. Our review also included three studies not included in the Shea et al. (2009) chapter that provide additional information to consider when weighing the effectiveness of group therapy. Two were recently published and one of these focused on a Veteran sample (Beck, Coffey Foy, Keane, & Blanchard, 2009; Ready, Thomas, Worley, Backscheider, Harvey, Baltzell, et al., 2008). The third, although dated, was a randomized trial with a Veterans sample (Rogers, Silver, Goss, Obenchain, Willis, & Whitney, 1999). Fourteen of the total 17 studies examined cognitive-behavioral interventions; two examined interpersonal therapy, and one psychodynamic.

Summary of Randomized Trials:

Eight of the 17 studies reviewed used randomized designs. Of these eight, six examined cognitive-behavioral therapy (CBT) approaches. Only three of these six compared the active treatment to a comparison condition that was not a wait-list. Schnurr and colleagues (2003) investigated Trauma-Focused Group Therapy (TFGT) in male Veterans of the Vietnam War in the largest and most rigorous study to date of group therapy for PTSD (Schnurr, Friedman, Foy, Shea, Hsieh, Lavori, et al., 2003). TFGT incorporates group-based psychoeducation, coping skills training, imaginal exposure, cognitive challenging, and relapse prevention, with one-third of all sessions devoted to individual work (Foy, Ruzek, Glynn, Riney, & Gusman, 2002). Schnurr et al (2003) did not include individual sessions. TFGT was compared with present-centered group therapy (PCGT), an approach designed to provide the “nonspecific” factors of support and interpersonal connection inherent in group treatment. Both groups experienced significant modest-sized pre- to posttreatment improvement in PTSD, which were maintained at 12 months. The primary intention-to-treat (ITT) analyses did not find differences on PTSD or any other outcomes between the group conditions (Schnurr, 2003). Rogers and colleagues (1999) compared a single group session of flooding-based exposure therapy with a single group session of eye movement desensitization and reprocessing (EMDR) in 12 Vietnam War Veterans who were undergoing inpatient treatment for combat-related

PTSD. There were no differences between groups on PTSD symptoms posttreatment, with both groups showing significant improvements. Lastly, Beck and colleagues (Beck et al., 2009) randomized 44 individuals with PTSD related to motor vehicle accidents (MVAs) to either Group Cognitive Behavior Therapy (GCBT) or a minimal contact comparison condition. The GCBT was a 14-week treatment adaptation of individual CBT to a group setting. At posttreatment, GCBT resulted in significantly greater reductions in PTSD symptoms among treatment completers, with large between group effect sizes and stability of gains at 3-months. Significantly more patients in GCBT (88.3 percent) versus in MCC (31.3 percent) no longer met criteria for PTSD at posttreatment.

Three of the trials, comparing CBT to a wait-list (WL) control, involved female populations. Imagery Rehearsal Therapy (IRT) resulted in significantly more improvement in PTSD, nightmares, and sleep, with large between-group effects on the CAPS and PTSD Symptom Scale (PSS) and improvements maintained at 6 months (Krakow, Hollifield, Johnston, Koss, et al., 2001a; Krakow, Hollifield, Scharader, et al., 2000). Large effects were also found on PTSD symptoms between an affect management group and WL control where both groups also received individual therapy and medication (Zlotnick, et al., 1997). A trial of women with PTSD related to diverse traumas, as well as co-morbid panic disorder, indicated that multichannel exposure therapy was superior to control (Falsetti et al., 2005).

Two small-scale randomized trials evaluated non-CBT approaches versus wait-list controls in women with PTSD related to sexual abuse. Comparison of a trauma-focused group and a present-focused group, both based on psychodynamic principles, showed no differences for either relative to a wait-list control (Spiegel, Classen, Thurston, & Butler, 2004) and even when combined, the composite treatment group showed significantly more improvement only on non-PTSD measures (Classen, Koopman, Nevill-Manning, & Spiegel, 2001). In contrast, Krupnick and colleagues (Krupnick, Green, Miranda, & Stockton, 2008) found significant effects with ITT analyses for Interpersonal Therapy group on PTSD, depression, and interpersonal functioning, with a medium-to-large effect for PTSD.

Caveat regarding analysis of data from group-administered treatments:

In examining the effects of these group treatments, a significant and prevalent methodological limitation warrants discussion. This limitation is that most studies failed to use analytic strategies to account for clustering of observations within treatment groups. Participants administered treatment in group format share a common therapy environment, which may homogenize response to the treatment. As explained by Baldwin and colleagues (Baldwin, Murray, & Shadish, 2005), studies that do not take into account the magnitude of the dependency among observations taken on members of the same group, or intraclass correlation (ICC), underestimate the standard error of the treatment effect by pooling the effect of the group with the effect of the treatment, so that even if treatment has no effect, an incorrect analysis can suggest a treatment effect.

Out of the 17 studies reviewed herein, only two studies corrected for unit of analysis and group ICC (Beck et al., 2009; Schnurr et al., 2003) and two studies accounted for ICC (Creamer et al., 2006; Ready et al., 2008) in analyses, with the remaining studies taking typical approach of treating the individual participants as the unit of analyses and not correcting for the ICC. It is likely that true effects for group treatments for PTSD are more modest than published effects.

Conclusions:

The empirical literature on group treatment for PSTD has grown since the publication of the first edition of the Treatment Guidelines for PTSD. However, most studies continue to utilize small sample sizes, use wait-list controls, and fail to account for clustering of observations in analyses. Advances in methodological rigor are typified within studies by Schnurr and colleagues (2003), as well as Beck and colleagues (2009). Field tests of interventions developed as part of clinical practice and evaluated on large samples, such as those by Creamer et al. (2006) and Ready et al. (2008), offer unique information yet pose numerous questions regarding complex multi-phase approaches.

With these caveats in mind, our review suggests that, overall, group-based treatment for individuals diagnosed with PTSD is associated with improvements in symptoms of PTSD. Reported pre- to post-treatment effect sizes range from small to large, but likely overestimate the true effect of the treatment. The amount of change exceeded that of wait-list controls for most studies. Psychodynamic treatment evidenced the weakest within-group effects (Classen et al., 2004). Interpersonal therapy evidenced small to large effects (Cloitre & Koenen, 2001; Krupnick et al., 2008). Significant support exists for cognitive–behavioral approaches, for both combat veterans and in adults with histories of abuse, with effects ranging from small to very large.

As noted above, few studies have directly compared different forms of group therapy. The two that have, indicated equal benefit from trauma-focused and present-centered supportive therapies in the primary analyses (Classen et al., 2004; Schnurr et al., 2003). Relatedly, it remains unknown whether improvements found in most studies of cognitive-behavioral or interpersonal/process-oriented treatments are due to the strategies employed. Shea and colleagues' (2009) examination of within-group effect sizes pre to posttreatment found no evidence that groups focusing on trauma provide superior outcomes than those who do not. Only one trial examined a group adaptation of an existing and proven individual therapy protocol (Beck et al., 2009). Reductions in PTSD from GCBD were comparable to those obtained in previous studies of individual CBT but GCBT did not reduce co-morbid anxiety and depression.

EVIDENCE TABLE

	Recommendation	Sources	LE	QE	SR
1	Consider group treatment for patients with PTSD	Beck et al., 2009 ❶ Classen et al., 2001; ❶ Cloitre & Koenen, 2001 Cook et al., 2006 Creamer et al., 2006 - Donovan et al., 2001 - Falsetti et al., 2005 ❶ Krakow et al., 2000, 2001 ❶ Krupnick et al., 2008 ❶ Lublin et al., 1998 Najavits et al., 1998 Ready et al., 2008 - Resick & Schniske, 1992 Rogers et al., 1999 ❶ Schnurr et al., 2003 v❶ Spiegel et al., 2004❶	I II	Fair- Poor	C

		Zlotnick et al., 1997❶; 2003			
2	Current findings do not favor trauma-focused versus present-focused group therapy	Schnurr et al., 2003 Classen et al., 2004 Shea et al., 2009	I	Good	I

LE=Level of Evidence QE=Quality of Evidence; SR= Strength of Recommendation (see Appendix A); ❶-RCT

B9. Dialectical Behavior Therapy

BACKGROUND

Dialectical behavior therapy (DBT) is a comprehensive cognitive-behavioral treatment for complex, difficult-to-treat mental disorders, specifically designed to treat chronically suicidal individuals and patients with multi-disorders or borderline personality disorder (BPD).

DBT has since been adapted for other seemingly intractable behavioral disorders involving emotion dysregulation, including substance dependence in individuals with BD and binge eating, to other clinical populations (e.g., depressed, suicidal adolescents) and in a variety of settings (e.g., inpatient, partial hospitalization, forensic).

While considerable evidence supports the use of exposure-based treatment for PTSD, its utilization may pose some problems for patients where the symptoms of PTSD are complicated. High rates of attrition, suicidality, dissociation, destructive impulsivity, and chaotic life problems are reasons cited by clinicians for abandoning empirically supported exposure treatment. Some practitioners have suggested that the approach of DBT, designed to address many of these issues, offers useful strategies for addressing the needs of patients considered poor candidates for exposure therapy.

The DBT approach incorporates what is valuable from other forms of therapy and is based on a clear acknowledgement of the value of a strong relationship between therapist and patient. Therapy is structured in stages, and at each stage a clear hierarchy of targets is defined. The techniques used in DBT are extensive and varied, addressing essentially every aspect of therapy. These techniques are underpinned by a dialectical philosophy that recommends a balanced, flexible, and systemic approach to the work of therapy. Patients are helped to understand their problem behaviors and then deal with situations more effectively. They are taught the necessary skills to enable them to do so and helped to deal with any problems that they may have in applying those skills. Advice and support are available between sessions. The patient is encouraged and helped to take responsibility for dealing with life's challenges.

DISCUSSION

Although DBT is becoming more common as a technique for treating patients with BPD, no clinical trials have been reported in the literature for the use of DBT in patients with PTSD. The following studies concern patients with BPD who attempted some form of self-injury; however, for patients with PTSD and co-morbid BPD, these studies may be applicable to the treatment decision process.

In a meta-analysis of RCTs of “psychosocial and/or psychopharmacological treatment versus standard or less intensive types of aftercare” for patients who had shown self-harm behaviors, Hawton et al. (2000) compared DBT versus standard aftercare and found that DBT significantly reduced rates of further self-harm (0.24; 0.06 to 0.93). The authors caution, however, that “there still remains considerable uncertainty

about which forms of psychosocial and physical treatments of self-harm patients are most effective."

Verheul et al. (2003) reported on the effectiveness of DBT in a group of 58 female BPD patients. For these women, DBT therapy "resulted in better retention rates and greater reductions of self-mutilating and self-damaging impulsive behaviors compared with usual treatment, especially among those with a history of frequent self-mutilation" (Verheul et al., 2003). In the same study group, van den Bosch et al. (2002) compared the results of therapy in women with and without co-morbid substance abuse. They found that co-morbid substance abuse did not dilute the effect of the DBT but that the DBT therapy had no effect on the womens' substance problems. Evans et al. (1999) compared the provision of self-help booklets alone to six sessions of cognitive therapy linked to the booklets, which contained elements of DBT (MACT), in 34 patients who had attempted self-harm. The authors reported that MACT therapy led to a lowering of the number of suicidal acts per month and also improved self-rated depressive symptoms.

Linehan and colleagues (1993) conducted a RCT of 39 women with BPD who were randomly assigned to DBT or usual care for one year, then followed up at six and twelve months following treatment. The authors reported that DBT patients had significantly less parasuicidal behavior, less anger, and better self-reported social adjustment during the initial 6 months and significantly fewer psychiatric inpatient days and better interviewer-rated social adjustment during the final 6 months.

Telch et al. (2001) and Safer et al. (2001) expanded the DBT concept to treatment of women with binge eating disorders. In both studies, women were randomly assigned to DBT or a wait list (Telch study – 44 women; Safer study – 31 women), and the authors' results were similar; patients improved significantly in reduction of binge/purge behaviors but did not differ on any secondary measures.

Bohus et al. (2000) treated 24 female chronically suicidal patients with DBT and found significant improvements in ratings of depression, dissociation, anxiety, and global stress and a highly significant decrease in the number of parasuicidal acts.

Gould et al. (2003) and Miller and Glinski (2000) identify DBT as a promising treatment for suicide; however, they acknowledge the need for RCTs. In their overview of the use of DBT, Koerner and Linehan (2000) also stress the need for longitudinal studies to determine suicide rates and maintenance of treatment gains.

EVIDENCE TABLE

	Evidence	Sources	QE	QE	R
1	DBT for patients with a borderline personality disorder typified by parasuicidal behaviors	Evans et al., 1999 Hawton et al., 2000 Linehan et al., 1993 Safer et al., 2001 Telch et al., 2001 van den Bosch et al., 2002 Verheul et al., 2003	I	Fair	B

LE=Level of Evidence QE=Quality of Evidence; SR= Strength of Recommendation (see Appendix A)

B10. Hypnosis

BACKGROUND

Hypnosis is not held to be an ASD or PTSD therapy per se but may significantly enhance the effectiveness of other therapies in their treatment or in the management of a variety of related clinical conditions (Kirsch et al., 1998; Spiegel & Spiegel, 1987). Historically, hypnotic treatments have played a role in the management of shell shock, battle fatigue, and traumatic neuroses.

Hypnosis is defined by the APA as "a procedure during which a health professional or researcher suggests that a client, patient, or subject experience changes in sensations, perceptions, thought, or behavior. The hypnotic context is generally established by an induction procedure" (Kirsch, 1994). An induction procedure typically entails instructions to disregard extraneous concerns and focus on the experiences and behaviors that the therapist suggests or that may arise spontaneously.

Hypnosis should only be used by credentialed healthcare professionals who are properly trained in the clinical use of hypnosis and are working within the areas of their professional expertise.

DISCUSSION

Most of the case studies that have reported that hypnosis is useful in treating post-trauma disturbances following a variety of traumas lack methodological rigor, and therefore strong conclusions about the efficacy of hypnosis to treat PTSD cannot be drawn (Rothbaum, 2001).

Brom and colleagues (1989), in a RCT, showed that hypnosis and desensitization significantly decreased intrusive symptoms, whereas psychodynamic therapy was useful for reducing avoidance symptoms in patients with various types of post-traumatic symptomatology. In a meta-analysis, Sherman (1998) compared the effects of the Brom et al. trial with those of other controlled studies and found that the major advantage of using hypnosis may appear at long-term follow-up rather than at the end of treatment: this is consistent with meta-analyses of hypnosis for conditions other than PTSD (Kirsch et al., 1999).

Various studies, including meta-analyses, of the treatment of anxiety, pain, repetitive nightmares, and other conditions often associated with PTSD imply that hypnosis can substantially reduce the severity of these problems (Daly & Wulff, 1987; Jiranek, 1993; Kirsch et al., 1995; Eichelman, 1985; Kingsbury, 1993) and enhance the effectiveness of psychodynamic and cognitive behavioral therapy (Kirsch, 1996; Kirsch et al., 1999; Smith et al., 1980). Most of the literature on the use of hypnosis for PTSD is based on service and case studies.

Shakibaei (2008) reported that hypnotherapy helped reduce both pain and re-experiencing of traumatic events among burn patients in a randomized control trial, but it should be noted that patients meeting criteria for any acute psychiatric disorder were specifically excluded from this study.

Abramowitz (2008) reports on a RCT in which hypnotherapy was compared to zolpidem treatment for insomnia among 32 patients with combat PTSD who were also suffering from insomnia. All patients were already taking an SSRI. He found significant improvement in PTSD symptoms and sleep quality, number of awakenings, ability to concentrate in the morning, and morning sleepiness in the hypnotherapy group. Sleep time improved equally in both groups.

There are a number of indications for using hypnosis in the treatment of PTSD:

1. Hypnotic techniques may be especially valuable as an adjunctive treatment for symptoms often associated with PTSD, including dissociation, anxiety, pain, nightmares, and insomnia.
2. PTSD patients who manifest at least moderate hypnotizability may benefit from the addition of hypnotic techniques to their treatment.
3. Because confronting traumatic memories may be very difficult for some PTSD patients, hypnotic techniques may provide them with a means to modulate their emotional and cognitive distance from such memories as they are worked through therapeutically.

There are a number of contraindications for using traditional hypnotic techniques in the treatment of PTSD:

1. In the rare cases of individuals who are refractory or minimally responsive to suggestion, hypnotic techniques may not be the best choice, because there is some evidence that hypnotizability is related to treatment outcome efficacy (Levitt, 1994; Spiegel et al., 1981 & 1993).
2. Some PTSD patients may be resistant to hypnotic treatment because of religious concerns or other beliefs. If resistance persists, other suggestive techniques may be tried, including emotional self-regulation therapy (ESRT), which is done with open eyes and uses sensory recall exercises rather than a hypnotic induction (Bayot et al., 1997; Kirsch et al., 1999).
3. For patients who have low blood pressure or are prone to falling asleep, hypnotic procedures, such as "alert hand," which emphasize alertness and activity rather than relaxation, may be substituted (Cardena et al., 1998).

EVIDENCE

	Recommendation	Sources	LE	QE	R
1	Hypnosis may be used to alleviate PTSD symptoms	Brom et al., 1989 Sherman, 1998 Shakibaei et al., 2008 Abramowitz et al., 2008	I	Fair-Poor	C

LE=Level of Evidence QE=Quality of Evidence; SR= Strength of Recommendation (see Appendix A)

B11. Behavioral Couples Therapy

BACKGROUND

Perceived social support has been identified as an important resilience factor in PTSD. Families report significant distress during the deployment cycle and high prevalence of family problems, such as divorce. A number of family and couples interventions have been developed, including Behavioral Family Therapy (BFT), Cognitive-Behavioral Couples Therapy (CBCT), and Support and Family Education (SAFE). However, there is as yet little support for these interventions as a first-line treatment for PTSD.

DISCUSSION

Glynn et al. (1999) conducted a RCT of couples or family treatment for PTSD, utilizing either an Exposure condition, Exposure followed by BFT, or a wait list control. While both active treatment conditions improved on PTSD symptoms, BFT did not significantly improve the PTSD symptoms, compared to the Exposure-only condition. However, BFT did demonstrate improved problem solving skills relative to the other two conditions.

Monson et al. (2004, 2005) conducted a small, uncontrolled pilot study of seven couples who received CBCT for PTSD. Significant improvements were found on PTSD, depression, and anxiety for both veterans and wives. The improvement in relationship satisfaction was more mixed, with no improvement for husbands but greater improvement for wives.

Devilly (2002) examined a Lifestyle Management course for male veterans with PTSD and their partners in a weeklong residential treatment. Both veterans and their partners experienced significant reductions in anxiety, depression, and stress; veterans also experienced significant reductions in PTSD. However, the effect size of these changes was small, and symptom improvements were considered to be of limited clinical importance.

No studies that evaluated behavioral couples therapy (BCT) for treatment of post-traumatic stress disorder (PTSD) were identified. One study (Rotunda et al., 2008) evaluated BCT for substance use disorder (SUD) in veterans with co-morbid PTSD. The study was not designed to evaluate BCT for treatment of PTSD but did assess psychological symptoms as a function of BCT. Although the effects of BCT on PTSD symptoms specifically were not reported, the results suggested that BCT may reduce general psychological distress and increase abstinence in male veterans with SUD and co-morbid PTSD. However, caution should be taken in generalizing these findings to a population with PTSD alone, given the body of literature demonstrating that those with co-morbid SUD and PTSD are different from those with PTSD alone on a number of important clinical variables (e.g., symptom severity, chronicity of illness, treatment refractory).

No review or meta-analysis publications that addressed BCT, BFT, or CBCT as a treatment for PTSD were identified.

EVIDENCE TABLE

	Evidence	Sources of Evidence	LE	QE	SR
1	BFT did not significantly improve the PTSD symptoms and was inferior to other psychotherapies	Glynn et al.,1999 Monson et al.,2004 Devilly et al.,., 2002	I	Fair - Poor	I

LE=Level of Evidence QE=Quality of Evidence; SR= Strength of Recommendation (see Appendix A)

B12. Telemedicine and Web-based Interventions

B12-1. TELEMEDICINE INTERVENTIONS

BACKGROUND

Increasingly, a range of technologies are being adapted to enhance delivery of mental health services. Such technologies include the telephone and videoconferencing tools. Some technological applications assist human providers in delivering their treatments to patients, as when videoconferences or telephones are used to reach those for whom attendance may be difficult, or increasing convenience for patients by eliminating travel to face-to-face sessions. Telephone-based services – phone-based counseling, automated telephone assessment, and interactive telephone applications – provide ways of extending assessment and treatment into the natural environment.

DISCUSSION

A burgeoning body of rigorous research has demonstrated that psychotherapy for treatment of depression and anxiety disorders, delivered either via

videoteleconferencing (VTC) or via telephone, is not only effective but clinically equivalent to face-to-face delivery (O'Reilley et al., 2007; Bee et al., 2008). However, only one study to date has conducted that level of rigorous examination of psychotherapy delivered to a PTSD population via VTC (Morland et al., 2009). This study found that Anger Management Group therapy via VTC was *as effective as* face-to-face delivery in reducing anger symptoms in PTSD patients, both immediately post-treatment and in short-term follow-up (i.e., 3 months). It also found that there were no significant differences between the two modalities in satisfaction with treatment, treatment credibility, attendance, homework completion, attrition, or alliance among group members (Morland et al., 2009). However, patients receiving treatment via VTC had lower therapeutic alliance with the group leader than those who received face-to-face delivery.

Additional trials of VTC delivery of PTSD-specific treatments have also demonstrated clinical effectiveness that was comparable to face-to-face delivery (e.g., Frueh et al., 2007). However, due to the methodologies used (e.g., small sample size, non-randomized), they were not able to test if VTC was actually equivalent to face-to-face treatment. A non-random cohort study demonstrated that CBT delivered via VTC improved PTSD symptoms at a level similar to face-to-face group delivery (Germain, Marchand, Bouchard, Drouin, & Guay, 2009). A recent pilot study found that Prolonged Exposure Therapy delivered via VTC was highly effective, safe, and feasible (Tuerk et al., 2010).

There has been somewhat inconsistent evidence of process outcomes, such as patient and provider satisfaction, patient treatment preference, comfort talking to their therapists, and homework compliance, among the different trials comparing VTC and face-to-face delivery of PTSD interventions. Although several studies have found no significant differences between the two modalities, some have found that in-person delivery has generated slightly better process outcomes (Morland, Pierce, & Wong, 2004; Frueh et al., 2007).

The effectiveness of telephone delivery of case management and support has been well proven for a wide variety of behavioral health interventions. However, it is much less studied with PTSD patients. A small cohort study demonstrated that telephone-based monitoring and support improved patient satisfaction and entry into aftercare compared to the treatment-as-usual condition (Rosen et al., 2006).

Mobile phone-based interventions present several advantages and capabilities (e.g., web-browsing, text messaging, software applications, etc.) that could address common problems in delivering evidence-based treatments (Boschen, 2010); however, the evidence to support these technologies in the PTSD interventions has yet to be generated.

In summary, telephone delivery and videoconferencing can be effectively used to overcome geographical barriers to mental healthcare. There is an abundance of evidence that the modalities are safe and effective. There is preliminary evidence to suggest that psychotherapy delivered via these modalities is as effective as face-to-face care. As the field develops, additional research needs to examine how TMH modalities affect the therapeutic process and also how mobile phone-based interventions can be effectively used for PTSD treatment.

EVIDENCE TABLE

	Evidence	Sources of Evidence	LE	QE	SR
1	Telephone delivery and videoconferencing can be effectively used to deliver psychotherapy	O'Reilley et al., 2007 Bee et al., 2008 Morland et al., 2009 Greene et al., 2010 Germain et al., 2009	I I I I II-1	Mod	C

LE=Level of Evidence QE=Quality of Evidence; SR= Strength of Recommendation (see Appendix A)

B12-2. WEB-BASED INTERVENTIONS

BACKGROUND

Increasingly, a range of computer and Internet technologies are being adapted to enhance delivery of mental health services. Web-based applications can deliver elements of treatment (such as psychoeducation or skills training) in the absence of provider contact or with reduced contact, and it is possible that access to help via technologies may increase engagement in care by reducing the stigma associated with treatment-seeking and increasing accessibility of care (e.g., for rural populations, disabled persons, individuals without easy transportation access). To date, research conducted with PTSD patients has been very limited, but services are increasingly being delivered via these technologies. Newly developed technologies can present significant challenges related to patient confidentiality and safety, and they must be addressed carefully by both the individual providers and the organization delivering these interventions.

DISCUSSION

Web-based interventions have very limited research for treatment of PTSD, although several studies have been done to assess these techniques particularly in traumatized individuals with general distress or subclinical PTSD symptoms. Web-based interventions may provide an effective delivery modality for CBT techniques that can be considered in certain circumstances. However, these interventions raise a number of privacy and confidentiality issues and have not been directly compared with other evidence-based person-to-person CBT modalities that have been shown to be efficacious.

The Internet provides a potential resource for delivery of both information (psychoeducation) and more complex interventions. At present, while there is much traumatic stress-related information available on the Web, the accuracy and authoritativeness of the information can be difficult for consumers to determine. Bremner, Quinn, Quinn, and Veledar (2006) reviewed the quality of 80 websites related to psychological aspects of trauma and found that 42 percent of sites had inaccurate information, 82 percent did not provide a source of content, and 41percent did not use a mental health professional in the development of the content. The authors concluded that although abundant, websites providing information about traumatic stress are often not useful and can sometimes provide inaccurate and potentially harmful information to consumers of medical information.

Despite these concerns, prominent authoritative websites that are grounded in research on psychological trauma and PTSD do exist, and many public organizations and universities have developed online information resources related to post-traumatic stress (e.g., National Center for PTSD site: www.ncptsd.va.gov; Center for the Study of Traumatic Stress, http://www.centerforthestudyoftraumaticstress.org/;

International Society for Traumatic Stress Studies, www.istss.org; National Institute of Mental Health, http://www.nimh.nih.gov/health/topics/post-traumatic-stress-disorder-ptsd/index.shtml).

Patients and family members should be warned that information about PTSD that is obtained from the Internet should be interpreted with caution. Internet sites from established healthcare agencies or patient advocacy organizations are recommended over chat rooms or non-specialist or commercial sites.

Several randomized controlled trials (RCTs) of web-based intervention treatment of PTSD have been conducted. Taken together, they provide preliminary support for the use of specific web-based CBT approaches. RCTs of web-based therapist-assisted interventions (Knauvelsrud & Maercker, 2007; Lange, et al., 2001; Lange et al., 2003; Litz, Engel, Bryant, & Papa, 2007) have demonstrated significant improvements in trauma-related symptoms compared to wait list and supportive counseling control conditions, with improvements being maintained over short-term (i.e., 3-month) follow-up periods. The studies have focused largely on traumatized individuals (clinical and non-clinical, such as university students) with generalized distress or subclinical PTSD symptoms. Only one of the RCTs selected patients based on a PTSD diagnosis. This study involved service members with PTSD related to the Pentagon attack of September 11, 2001 or combat in Iraq or Afghanistan (OEF & OIF) (Litz et al., 2007). However, the definition of PTSD was not based on a standard structured clinical interview, and no difference was found in the intent-to-treat analysis between the internet-based CBT and internet-based supportive therapy control. In addition, this was not a pure internet-based intervention, as it involved a 2-hour initial face-to-face session in addition to periodic telephone contact. A meta-analysis of Internet interventions for anxiety (Reger & Gahm, 2009) found that the effect sizes for PTSD symptoms fell in a large range (ES = .75; CI = .49 to 1.01), but again, this was not based on studies of patients with PTSD per se but rather persons who have sustained trauma and who have distress, subclinical PTSD, or, in some cases, actual PTSD. Reger and Gahm (2009) also noted many methodological problems with current studies and indicated that additional research is needed to determine evidence for effectiveness.

In conclusion, there is insufficient evidence to recommend web-based interventions for treatment of PTSD. The use of the Internet may have relevance as adjunctive modalities in assisting distressed traumatized individuals and complementing other evidence-based treatment interventions.

As with face-to-face treatments, it is important to recognize that existing studies have looked at the effectiveness of specific web-based protocols. Thus, it cannot be inferred that the studied modalities are generalizable to other web-based treatments. Three of the studies cited above relate to one intervention, entitled Interapy (Lange, et al., 2001; Lange et al., 2003; Knauvelsrud & Maercker, 2007). Interapy and DeStress (Litz et al., 2007) share several intervention components, including repeated writing about the traumatic experience. These evidence-supported web-based protocols are also therapist-assisted, with significant input from the provider. For example, Interapy involves a mean per-patient total of 14 hours of therapist time. Evidence from research on other mental health problems indicates that rates of attrition from web-based interventions are high in the absence of provider contact to facilitate completion. With regard to PTSD, there is relatively little evidence at present for the effectiveness of Internet interventions that are completely self-administered (e.g., Hirai & Clum, 2005).

Regardless of intervention mode, it is important that those involved in technology-assisted intervention delivery take steps to ensure that their work complies with the regulations and procedures of the organization in which they are employed, with evolving legal standards, and with the ethical standards of their professions. Newly developed technologies can present significant challenges related to patient confidentiality and safety, and these must be addressed carefully by both the individual providers and the organization delivering these interventions.

EVIDENCE TABLE

	Evidence	Sources of Evidence	LE	QE	SR
1	Insufficient evidence to recommend web-based interventions for treatment of PTSD	Knauvelsrud & Maercker, 2007	I	Poor	I
		Lange, et al., 2001	I	Poor	
		Lange et al., 2003	I	Poor	
		Litz, et al., 2007	I	Good	

LE=Level of Evidence QE=Quality of Evidence; SR= Strength of Recommendation (see Appendix A)

C. PHARMACOTHERAPY FOR PTSD

There is growing evidence that PTSD is characterized by specific psychobiological dysfunctions, which have contributed to a growing interest in the use of medications to treat trauma-related biological effects (see Table I-6).

Studies of medication classes used in therapy for PTSD in individuals exposed to trauma that assessed clinical outcomes were included in the review for this guideline update. Evidence from randomized controlled trials (RCTs) was considered to be of highest quality, followed by observational evidence. Other sources were evaluated when randomized controlled trials and observational studies were not available or did not provide adequate evidence. Studies were excluded if they did not evaluate response to pharmacotherapy and if they did not evaluate individuals exposed to trauma. The recommendations and tables address only drugs that have been studied in RCTs and are available in the U.S. Other drugs that have not been reported in published studies or were tested in open-label trials have not been considered and therefore do not appear in the table (see **Table I - 6**).

Table I - 6 Pharmacotherapy Interventions for Treatment of PTSD

	Effect = Balance of Benefit and Harm			
SR	**Significant**	**Some Benefit**	**Unknown**	**No Benefit**
A	SSRIs SNRIs		-	-
B	-	Mirtazapine Prazosin (for sleep/nightmares) TCAs Nefazodone [Caution]* MAOIs (phenelzine) [Caution]*	-	-
C			Prazosin (for global PTSD)	
D	-	-	-	Benzodiazepines [Harm] Tiagabine Guanfacine Valproate Topiramate Risperidone
I	-	-	Atypical antipsychotic (Except risperidone, as adjunct) Atypical antipsychotic (monotherapy) Conventional antipsychotics Buspirone Non-benzodiazepine hypnotics Bupropion Trazodone (adjunctive) Gabapentin Lamotrigine Propranolol Clonidine	-

*SR = Strength of recommendation (see Appendix A); * Attention to drug to-drug and dietary interactions*

RECOMMENDATIONS

General Recommendations:

1. Risks and benefits of long-term pharmacotherapy should be discussed prior to starting medication and should be a continued discussion item during treatment.
2. Monotherapy therapeutic trial should be optimized before proceeding to subsequent strategies by monitoring outcomes, maximizing dosage (medication or psychotherapy), and allowing sufficient response time (for at least 8 weeks). [C]
3. If there is some response and patient is tolerating the drug, continue for at least another 4 weeks.
4. If the drug is not tolerated, discontinue the current agent and switch to another effective medication.
5. If no improvement is observed at 8 weeks consider:
   a. Increasing the dose of the initial drug to maximum tolerated
   b. Discontinuing the current agent and switching to another effective medication
6. Recommend assessment of adherence to medication at each visit.
7. Recommend assessment of side effects and management to minimize or alleviate adverse effects.
8. Assess for treatment burden (e.g., medication adverse effects, attending appointments) after initiating or changing treatment when the patient is non-adherent to treatment or when the patient is not responding to treatment.
9. Since PTSD is a chronic disorder, responders to pharmacotherapy may need to continue medication indefinitely; however, it is recommended that maintenance treatment should be periodically reassessed.
10. Providers should give simple educational messages regarding antidepressant use (e.g., take daily, understand gradual nature of benefits, continue even when feeling better, medication may cause some transient side effects, along with specific instructions on how to address issues or concerns, and when to contact the provider) in order to increase adherence to treatment in the acute phase. [B]

Monotherapy:

11. Strongly recommend that patients diagnosed with PTSD should be offered selective serotonin reuptake inhibitors (SSRIs), for which fluoxetine, paroxetine, or sertraline have the strongest support, or serotonin norepinephrine reuptake inhibitors (SNRIs), for which venlafaxine has the strongest support, for the treatment of PTSD. [A]
12. Recommend mirtazapine, nefazodone, tricyclic antidepressants (TCAs), amitriptyline and imipramine, or monoamine oxidase inhibitors (phenelzine) for the treatments for PTSD. [B]
13. Recommend against the use of guanfacine, anticonvulsants (tiagabine, topiramate, or valproate) as monotherapy in the management of PTSD. [D]

14. The existing evidence does not support the use of bupropion, buspirone, and trazodone, anticonvulsants (lamotrigine or gabapentin) or atypical antipsychotics as monotherapy in the management of PTSD. [I]
15. There is evidence against the use of benzodiazepines in the management of PTSD. [D]
16. There is insufficient evidence to support the use of prazosin as monotherapy in the management of PTSD. [I]

Augmented Therapy for PTSD:

17. Recommend against the use of risperidone as adjunctive therapy [D]. There is insufficient evidence to recommend the use of any other atypical antipsychotic for treatment of PTSD. [I]
18. Recommend adjunctive treatment with prazosin for sleep/nightmares. [B]
19. There is insufficient evidence to recommend a sympatholytic or an anticonvulsant as an adjunctive therapy for the treatment of PTSD. [I]

DISCUSSION

Treatment of PTSD Core Symptoms

The published pharmaceutical randomized clinical controlled trials (RCTs), which target chronic PTSD symptoms, include drugs of the following drug classes: antidepressants, (e.g., SSRIs, monoamine oxidase inhibitors, tricyclics, as well as atypical antipsychotics), anticonvulsants, benzodiazepines, alpha-adrenergic blockers, and others such as d-cycloserine.

Antidepressants

Antidepressants, particularly serotonergic reuptake inhibitors (SSRIs), have proven to be effective in treating PTSD and are recommended as first-line agents in treatment guidelines (Davidson et al., 2001; Brady et al., 2000; Foa et al., 2000; Foa et al., 1999). Over 3000 patients have participated in studies of paroxetine, sertraline, and fluoxetine. Sertraline and paroxetine have FDA approval for PTSD. SSRIs have a broad spectrum of action, effectively reducing all three core symptoms of PTSD. As a class, they are generally well tolerated.

The Cochrane Collaboration published a review of the evidence regarding pharmacological treatments in PTSD (Stein et al., 2006). They found 35 short-term RCTs of PTSD (4597 participants) to review, three of which contained a maintenance component; five of those were unpublished. The authors concluded that while no clear evidence exists to show that any particular class of medication is more effective or better tolerated than any other, the greatest number of trials showing efficacy to date, as well as the largest, has been with the SSRIs. On the basis of the data, the review recommends SSRIs as first-line agents in the pharmacotherapy of PTSD and supports their value in long-term treatment.

A meta-analysis of 4 RCTs that compared SSRIs to placebo without regard to diagnostic criteria, duration, severity, or co-morbid diagnoses reported that treatment favored the drug in all 4 trials; however, only one study (with 183 subjects) reached statistical significance. Two RCTs maintained treatment with an SSRI for 64 weeks and 40 weeks, respectively. One study reported that 50 percent of patients experienced worsening symptoms when placebo was substituted for active drug, and in the second report, patients on placebo were 6.4 times more likely to relapse compared to the drug group. Although some patients may respond to an

antidepressant trial within 3 months, some patients may require more than 12 weeks to respond to SSRIs (Martenyi et al., 2002).

Results with SSRIs are conflicting with respect to wartime-related PTSD. Martenyi et al. (2002), with combat veterans of recent wars, found fluoxetine to be significantly superior to placebo. Martenyi (2007) reported a negative fixed-dose trial with fluoxetine. In addition, Friedman et al. (2007), testing Vietnam vets with chronic PTSD in a VA hospital setting, observed no difference between sertraline and placebo. One should not extrapolate the findings of Friedman's paper to all veterans, as veterans with chronic PTSD who remain symptomatic after decades of VA treatment comprise a chronic treatment refractory cohort that is not representative of all male combat veterans with PTSD.

The SSRIs citalopram, escitalopram, and fluvoxamine have been not been studied sufficiently to warrant a recommendation.

Venlafaxine, an SNRI, has been shown to have positive results in two trials of more than 800 participants with non-combat-related PTSD (Davidson, 2006a, 2006b). Duloxetine and desvenlafaxine have not been studied and can not be recommended at this time. In a 24-week comparison trial, venlafaxine performed as well as sertraline in a civilian population (Davidson, 2006b).

Other monotherapy recommendations are mirtazapine, nefazodone, TCAs, and MAOIs, although these agents have been studied in fewer patients and are considered second-line treatment options. Of the TCAs, only amitriptyline and imipramine have demonstrated positive outcomes, while data on desipramine and nortriptyline have been negative and from poor-quality studies. Nefazodone has been the subject of several small- to mid-sized RCTs and case-control studies (Davis et al., 2000; Garfield et al., 2001; Gillin et al., 2001; Hertzberg et al., 1998; Hidalgo et al., 1999; Zisook et at., 2000). In all six studies, the drug was helpful in improving CAPS, HAM-D, sleep, and anxiety. In a trial of combat- and sexual assault-origin PTSD, nefazodone was more effective than placebo (Davis, 2004). Nefazodone has demonstrated efficacy equivalent to sertraline in two fair-quality trials (McRae, 2004; Saygin, 2002). Two trials with mirtazapine (Davidson, 2003; Chung, 2004) have demonstrated positive findings. However, in the placebo-controlled trial (Davidson, 2003), both mirtazapine and placebo had large effect sizes. In a trial of military veterans, mirtazapine was as efficacious as sertraline, but there was no placebo comparison arm (Chung, 2004). Of the currently available MOAIs, only phenelzine has been studied. In a placebo comparison trial, Vietnam veterans assigned to phenelzine had significant improvement in IES compared to placebo (Kosten, 1991).

Atypical Antipsychotics

Atypical antipsychotics are not effective as monotherapy. The efficacy of atypical antipsychotics as adjunctive treatment to antidepressants has been studied in trials composed primarily of veterans. Response was predominantly in hyperarousal and re-experiencing symptom clusters. There have been ten published RCTs of two different antipsychotics, risperidone and olanzapine. Quetiapine has been studied in one small open-label trial. Olanzapine as an adjunctive treatment improved CAPS scores and sleep quality compared to placebo in a small 8-week trial (Stein M, 2002). Quetiapine improved both PANS and CAPS scores compared to baseline (Hamner & Deitsch 2003b).

Six small trials, of variable design quality, use risperidone an augmentation to other medications, rather than as a primary treatment. Risperidone has shown to improve psychotic symptoms. One trial addressed risperidone's role in co-morbid PTSD and

psychosis (Hamner, 2003a). A significant improvement in psychotic symptoms (change in PANS) was found in veterans treated with risperidone compared to placebo; both groups improved significantly in their CAPS scores. The results of the remaining 5 trials showed risperidone to have some benefit as adjunct to antidepressants, although only a small net benefit.

A seventh trial, conducted by the VA Cooperative Study Group (Krystal, 2011), randomized 247 veterans with military-related PTSD deemed resistant to antidepressants to risperidone (up to 4 mg/day) or placebo as adjunctive treatment. After 6 months the changes from baseline in CAPS scores were not significant between the two treatment arms. Changes in CAPS subscale scores for reexperiencing and hyperarousal were statistically significant favoring risperidone but the differences were not considered clinically important. No difference in the symptom scales for anxiety, depression, positive or negative symptoms, sleep or quality of life were found. The authors concluded that compared to placebo, risperidone did not reduce PTSD symptoms. This is the largest clinical trial of an atypical antipsychotic as a treatment of PTSD to date.

This study clearly shows that adjunctive risperidone does not benefit veterans with chronic PTSD; the results do not justify the risk for metabolic adverse effects. However, limited reductions in hyperarousal and reexperiencing suggest some benefit for a few patients. Low study attrition, low cross-site variability, multiple measures, and long study duration reinforce the validity of these findings. Some might object that the many other medications taken by these patients might have obscured risperidone's effects; but this objection is not clinically relevant as risperidone is always likely to be prescribed in this context. Results may not generalize to other atypical antipsychotics, to women, or to civilians.

Anticonvulsants

The existing evidence does not support the use of anticonvulsants as monotherapy for the management of PTSD core symptoms. Tiagabine has been compared to placebo in two RCTs, with no difference in response (Connor 2006; Davidson 2007). Valproate, as monotherapy, did not differ from placebo in one RCT (Davis, 2008). Anticonvulsants are frequently used as adjunctive treatments. Only topiramate has been studied in this role in veterans, with negative results (Lindley, 2007). Data on other anticonvulsants are insufficient to recommend their use in PTSD. A meta-analysis showed benefit in the use of valproate in PTSD (Adamou, 2007).

Benzodiazepines

Benzodiazepines are widely used for symptomatic control of insomnia, panic/anxiety, and irritability; there is no evidence that they reduce the core symptoms (e.g., syndromal symptoms) of PTSD, such as avoidance or dissociation (Friedman and Davidson & Stein, 2009; Viola et al., 1997). Kosten et al. (2000) present evidence that does not support the use of benzodiazepines in PTSD.

Benzodiazepine administration should be discouraged both in acute stress disorder (ASD) and post-traumatic stress disorder (PTSD), due to lack of evidence for effectiveness and risks that outweigh potential benefits. Although benzodiazepines have been frequently used “as needed” and continuously for anxiety disorders, including to augment evidence-based treatment modalities in PTSD, there is evidence to suggest that benzodiazepines may actually potentiate the acquisition of fear responses and worsen recovery from trauma. Benzodiazepine use should be considered relatively contraindicated in combat veterans with PTSD because of the very high co-morbidity of combat-related PTSD with alcohol misuse and substance

use disorders (upwards of 50 percent of co-morbidity) and potential problems with tolerance and dependence. Once initiated in combat veterans, benzodiazepines can be very difficult, if not impossible, to discontinue, due to significant withdrawal symptoms, compounded by the underlying PTSD symptoms.

The two clinical trials of benzodiazepines to treat PTSD have shown negative findings:

- Braun, et al. (1990) - In a randomized double-blind cross-over study, alprazolam showed no significant benefit in alleviating PTSD symptoms compared with placebo. A slight reduction in anxiety symptoms was offset by withdrawal effects documented after only five weeks of treatment.
- Cates, et al. (2004) - This small, single-blind cross-over placebo-controlled study compared clonazepam with placebo for the treatment of sleep dysfunction associated with combat-related PTSD. The study showed no significant difference between the benzodiazepine and placebo treatments.
- Viola et al. (1997) - At Tripler Army Medical Center, after having treated 632 patients, the vast majority of whom suffered from combat-related PTSD, between 1990 and 1996, the staff began to "explore treatment alternatives" to benzodiazepines due to the "risks attendant to benzodiazepine management of PTSD, coupled with poor clinical outcome".
- Risse et al. (1990) - This case series reflects the typical clinical experience when benzodiazapines are utilized for treating combat-related PTSD. In this study, alprazolam was used to augment treatment of anxiety symptoms in 8 combat veterans with chronic PTSD and co-morbid conditions (mostly alcohol misuse). Although anxiety initially improved with treatment, the improvement was short-lived and resulted in tolerance to increasing doses and eventual failure of the treatment. The key problem was encountered upon attempting to gradually withdraw the medication after determining that ongoing treatment was not going to be of further benefit. All 8 patients experienced severe reactions, including anxiety, sleep disturbance, rage, hyper-alertness, increased nightmares, and intrusive thoughts; 6 of the 8 veterans developed a level of rage with homicidal ideation that they had never encountered previously.
- Randal et al. (1995) and Coupland et al. (1997) - Flumazenil, a benzodiazepine/GABA receptor antagonist, provokes panic attacks in patients with panic disorder but not in healthy controls. In these two studies (one of which involved a group of male Vietnam combat veterans), flumazenil was compared with placebo to determine if it provoked anxiety, panic, or PTSD symptoms. Both studies showed that there were no significant increases in anxiety, panic, or PTSD symptoms in subjects as a result of flumazenil administration. This suggests that PTSD is dissimilar to panic disorder in terms of benzodiazepine receptor functioning and helps to explain why benzodiazepine treatment has produced no significant benefits in clinical trials.
- Several studies involving different animal models of PTSD (for example, Matar et al., 2009; Hebert et al., 1996) have shown that benzodiazepine administration in the immediate aftermath of stress exposure significantly increases vulnerability of developing more severe responses upon subsequent exposure to stress.

Sympatholytics

Prazosin, as a global treatment for PTSD, has yielded mixed results; it has shown consistent efficacy in improving sleep and reducing nightmares. In five relatively

small studies (Raskind et al., 2002, 2003, and 2007; and Taylor 2006, 2008), prazosin has demonstrated a value in reducing nightmares and in improving CAPS, CGI, and CGIC scores. The goal of these studies was not evaluation of overall PTSD symptoms, but evaluation of targeted symptoms, which showed good outcomes. (See discussion in Module I-3 A. Sleep Disturbance).

Guanfacine was studied in two trials (Neylan et al. 2006; and Davise et al., 2008). No effect was seen on measures of PTSD symptom severity for the actively treated group relative to the placebo group.

Other Agents

Buspirone, a non-benzodiazepine anti-anxiety drug, is reported to have "clinical efficacy" in two very small studies (Duffy & Malloy, 1994; Wells et al., 1991).

A single clinical investigation of the effect of the antibiotic **D-cycloserine** (Heresco-Levy et al., 2002) enrolled 11 patients in a crossover trial. While patients reported some improvement on self-reported measures of PTSD symptoms, similar improvements were seen in placebo-treated patients.

Table I - 7 Pharmacological Studies for Treatment of PTSD

Drug	Source of Evidence	Result	n	LE	QE	NB	SR
SSRI							
Sertraline *	Brady et al., 2000	Significant improvement, CAPS-2, CGI	187	I	G	Sub	A
	Chung et al, 2004	Mirtazapine is effective as sertraline Military veterans (Korean)	51	II-1	F	Mod	
	Davidson et al., 2001a	Significant responder rate, CAPS-2	208	I	G	Sub	
	Davidson et al., 2002	Study of effect on individual symptoms	?	II-2	F	Mod	
	Davidson et al.,2001b	Effective for preventing PTSD relapse	96	I	G	Sub	
	Friedman et al., 2007	No sig diff. between Tx and placebo. Combat trauma.	169	I	G	Zero	
	Londborg et al., 2001	Significant response maintained x 36 weeks	128	II-1	G	Sub	
	Rapaport et al., 2002	Significant response maintained x 64 weeks	359	I/II-1	G	Sub	
	Tucker et al., 2003	Significant improvement of primary outcome	58	I	F	Zero	
	Zohar et al., 2002	Numerical advantage (only), not sig, Israeli vets	42	I	G	Zero	
Paroxetine *	Marshal, 2007	Significant improvement, CAPS-2 & CGI	52	I	G	Sub	A
	Marshall, et al., 2001	Significant improvement, CAPS-2 & CGI	551	I	G	Sub	
	Tucker, et al., 2001	Significant improvement, CAPS-2	307	I	G	Sub	
Fluoxetine	Barnett et al., 2002	Study of tolerability. Well tolerated	65	I	G	Sub	A
	Connor et al., 1999	"Superior" response for civilian patients	53	I	G		
	Davidson, 2005	Well tolerated and effective in prevention of relapse, improved CGI. High rate of relapse	57	I	G	Sub	
	Martenyi, 2007	No significant dif. between Tx and Pbo; TOP-8	411	I	G	Zero	
	Martenyi et al., 2002a	Effective for prevention of PTSD relapse; 50% subjects - combat related	131	I	G	Sub	
	Martenyi et al., 2002b	Effective: improvement in TOP-8, CGI; 50% subjects -combat related	301	I	G	Sub	
	Meltzer-Brody, et al., 2000	Reduced all symptom clusters of PTSD;	53	II-2	F		
	Van der ko et al., 1994	Fluoxetine > placebo, more in non-VA pts	64	I	G	Sub	
Citalopram	Seedat et al., 2000	Significant improvement, CAPS-2	14	II-1	F	Zero	C
	Tucker et al, 2003	Sig improvement (↓ BP)	58	I	F	Neg	
Fluvoxamine	Escalona et al., 2002	Appears to improve PTSD symptoms	15	III	P	-	I
	Neylan et al., 2001	Improved sleep quality for Vietnam vets	21	III	P	-	

Drug	Source of Evidence	Result	n	LE	QE	NB	SR
TCA							
Amitriptyline	Davidson et al., 1990	Effective for core symptoms of PTSD	46	I	G	Mod	B
	Davidson et al., 1993	Significant improvement: IES, CGI, HAMD	62	I	G		
Desipramine	Reist et al., 1989	Did not show efficacy; no statistics	27	III	P		
Imipramine	Kosten et al., 1991	Significant improvement, CAPS-2, IES	41	I	G		
Nortriptyline	Zygmont et al., 1998	Effective for traumatic grief symptoms	22	II-1	G		
	Dow et al., 1997	Improvement in CGE for PTSD with MDD	72	II-2	F		
MAOI/RIMA							
Phenelzine	Kosten et al., 1991	Significant improvement in IES, better than placebo	37	I	G	Mod	B
SNRI							
Venlafaxine	Davidson et al, 2006	Effective in tx PTSD, Improves resilience	329	I	G	Sub	A
	Davidson et al., 2006b	Effective similar to sertraline	531	I	G	Sub	
Secondary AD							
Bupropion	Becker et al., 2007	Bupropion SR had no effect on PTSD	30	I	F	Neg	I
	Canive et al., 1998	No change in total CAPS score - male veterans	17	II-2	F		
Nefazodone	Davis et al, 2004	Nefazodone is effective and well tolerated ; Combat, sexual	42	I	G	Sub	B
	Davis et al., 2000	Significant improvement in CAPS, HAM-D	36	II-2	G		
	Garfield et al., 2001	Significant improvement in CAPS, anxiety	14	II-2	F		
	Gillin et al., 2001	Significant improvement in sleep, CAPS	12	II-2	F		
	Hidalgo et al., 1999	High response rate; pooled data, 6 studies	105	II-2	F		
	McRae et al., 2004	Nefazodone is effective as sertraline. High attrition rates	37	I	F	Sub	
	Saygin et al,, 2002	Nefazodone is effective as sertraline and well tolerated Earthquake survivors	54	I	F	Sub	
	Zisook et at., 2000	PTSD symptoms lessened, CAPS	19	II-2	F		
Trazodone	Warner et al., 2001	Reduction in nightmares; 9 reported priapism	74	III	P		I
Mirtazapine	Chung et al., 2004	Mirtazapine is effective as sertraline and well tolerated. Military veterans (Korean)	51	II-1	F	Mod	B
	Davidson et al., 2003	Significant improvement in the SPRINT, SIP, DTS as compared to placebo	26	I	F	Mod	B

Drug	Source of Evidence	Result	n	LE	QE	NB	SR
Anticonvulsants							
Gabapentin	Hamner et al., 2001	Effective for insomnia, adjunct treatment	30	II-2	P	Zero	I
Lamotrigine	Hertzberg et al., 1999	Promising results	14	I	P	Zero	I
Topiramate	Lindley, 2007	No significant effect for topiramate over placebo	40	I	F	Zero	D
	Tucker , 2007	Not significant difference from placebo (non-combat)	38	I	G	Zero	
Valproate	Davis, 2008	Divalproex monotherapy was not effective in the treatment of chronic PTSD	85	I	G	Zero	D
	Adamou, 2007 (SR)	Valproate was generally effective in reducing hyperarousal, improving irritability and anger	63	I	F	Small	
Tiagabine	Connor, 2006	No significant improvement was observed on all outcome measures	29	I	F	Zero	D
	Davidson, 2007	No difference from placebo	232	I	G	Zero	
Atypical Antipsychotics							
Olanzapine	Butterfield et al., 2001	No beneficial effect. High placebo response	15	I	G		I
	Petty et al., 2001	Significant improvement in CAPS, CGI	48	II-1	G		
	Stein et al., 2002	Adjunct to SSRI. Sig. improve measures but not global PTSD	19	I	F	Mod	
Quetiapine	Hamner et al., 2003	Significant improvement in CAPS	20	II-1	F		I
Risperidone	Bartzokis et al., 2005	Risperidone (adjunct.) > placebo; Military vets.	48	I	G	Sub	D
	Krystal et al., 2011	Risperidone (adjunct) = placebo; Veterans.	247	I	G	Zero	
	Hamner et al., 2003	Adjunct to other meds, co-morbid psychoses. Vietnam vets	40	I	F	Sub	
	Monnelly et al., 2003	Risperidone > placebo; Military combat	15	I	F	Mod	
	Padala et al., 2006	Sexual assault - risperidone monotherapy > placebo	20	I	P	Small	
	Reich et al., 2004	Child abuse - risperidone > placebo;	21	I	F	Mod	
	Rothbaum et al., 2008	Civilian - risperidone (adjunct) was helpful in subjects who did not remit with sertraline alone	45	I	F	Small	

Drug	Source of Evidence	Result	n	LE	QE	NB	SR
Sympatholytics							
Clonidine	Kinzie & Leung, 1989	Cambodian refugees improved, dual therapy	68	III	P	-	I
Guanfacine	Horrigan & Barnhill, 1996	suppression of PTSD associated nightmares in children	1	III	P	-	D
	Neylan, 2006	No effect on PTSD symptoms (in vet military)	63	I	Good	Neg	
	Davis, 2008	No effect on PTSD symptoms	65	I	Good	Neg	
Prazosin	Raskind et al., 2003	Significant improvement, CAPS, CGI	10	I	F	Small	B
	Raskind et al., 2002	Significant improvement in dream scores	59	II-2	F	Zero	
	Raskind et al., 2007	Significant improved sleep quality, reduced nightmares, better overall sense of well-being.	34	I	G	Mod	
	Taylor et al., 2006	Reduction in global PTSD illness severity	11	II	P	Mod	
	Taylor et al., 2008	Significantly improved CGI-I scores and changed PDRS scores toward normal dreaming	13	I	F	Mod	
Benzodiazepines							
Benzodiaz.	Kosten et al., 2000	Not associated with adverse outcomes	370	II-2	F	Zero	D
Alprazolam	Braun et al., 1990	Did not show efficacy. (concern: rebound anxiety)	16	I	F	Zero	D
Clonazepam	Fossey & Hamner, 1994	A source of sexual dysfunction	42	III	P	Zero	D
	Gelpin et al., 1996	No beneficial effect in PTSD, may worsen outcome	20	II-1	F	Zero	
	Shalev & Rogel 1992	No effect on auditory startle	N/A	III	F	Zero	
Temazepam	Melman et al., 2002	No benefits in preventing PTSD; may worsen outcome	11	I	P	Zero	D
	Cates et al., 2004	No difference between the benzodiazepine and placebo		I	P	Zero	D
D-cycloserine							
	Heresco-Levy, 2002	Improvements in numbing, avoidance, and anxiety symptoms	11	I	Fair	Zero	I

LE=Level of Evidence QE=Quality of Evidence (F=Fair; G=Good; P=Poor); NB=Net benefit (Sub=Substantial; Mod = Moderate; Neg=Negative)
SR= Strength of Recommendation (see Appendix A)

* FDA Approved

Table I - 8 Symptom Response by Drug Class and Individual Drug (based on controlled trials)

		Global Improvement	Re-experiencing (B)	Avoidance/ Numbing (C)	Hyperarousal (D)
SSRI	Fluoxetine	X	X	X	X
	Sertraline	X	X	X	X
	Paroxetine	X	X	X	X
SNRI	Venlafaxine	X	X	X	X
TCAs	Amitriptyline/ Imipramine	X	X		X
MAOIs	Phenelzine	X	X		X
Sympatholytics	Prazosin	X	X		X
Other Anti-depressants	Mirtazapine	X	X		X
	Nefazodone	X	X		X

Table I - 9 Drug Details

Agent	*Oral Dose	Contraindications	Adverse Events	Pregnancy Category	Remarks
Selective Reuptake Serotonin Inhibitors (SSRIs)					
Fluoxetine Paroxetine Sertraline Fluvoxamine Citalopram Escitalopram	20 – 60 mg/d 20 – 60 mg/d 50 – 200 mg/d 50 – 150 mg bid 20 – 60 mg/d 10 – 20 mg/d	Contraindications : • MAO inhibitor within 14 days • Concurrent use of pimozide or thioridazine • Hypersensitivity	• Nausea , diarrhea • Headache • Dizziness • Sexual dysfunction • Hyponatremia/SIADH (Syndrome of Inappropriate Anti-diuretic Hormone) • Nervousness, anxiety, agitation • Serotonin syndrome	• All except paroxetine are Category C • Paroxetine Category D • Women planning to breast-feed, consider an anti-depressant with the lowest excretion into breast milk: paroxetine, sertraline	• Avoid abrupt discontinuation of all except fluoxetine • Citalopram, escitalopram, and sertraline are less likely to be involved in hepatic enzyme drug interactions involving CYP2D6 or 3A4 • All except escitalopram are generically available • St. Johns Wort may in decrease the concentration of SSRIs metabolized by CYP2D6
Tricyclic Antidepressants					
Imipramine Amitriptyline Desipramine Nortriptyline Protriptyline Clomipramine	150 – 300 mg/d 150 – 300 mg/d 100 – 300 mg/d 50 – 150 mg/d 30 – 60 mg/d 150 – 250 mg/d	Contraindications : • Clomipramine – seizure disorder • MAOI use within 14 days • Acute MI within 3 months • Hypersensitivity Relative Contraindications: • Coronary artery disease • Prostatic enlargement	• Dry mouth • Dry eyes • Constipation • Orthostatic hypotension • Increased heart rate • Ventricular arrythmias • Weight gain • Drowsiness	• Category C • Women planning to breast-feed, consider an antidepressant with the lowest excretion into breast milk: nortriptyline	• Therapeutic blood concentrations not established for PTSD • Desipramine and nortriptyline have lower rate of sedation anticholinergic and hypotensive effects • Moderate CYP2D6 inhibition • St. Johns Wort may decrease the concentration of SSRIs metabolized by CYP2D6

Agent	*Oral Dose	Contraindications	Adverse Events	Pregnancy Category	Remarks
Monoamine Oxidase Inhibitors					
Phenelzine Tranylcypromine	45-75 mg/d in divided doses 10 – 60 mg/d target 1 mg/kg/d target 0.7 mg/kg/d	Contraindications: • All antidepressants within 14 days of start of a MAOI, except fluoxetine is 5 weeks • Concurrent use with CNS stimulants or depressants and decongestants • CHF, hepatic or renal disease • Pheochromocytoma • Foods high in tyramine • Hypersensitivity	• Hypertensive crisis with drug/tyramine interactions • Bradycardia • Orthostatic hypotension • Insomnia • Dry mouth • Dry Eyes • Constipation	• Category C	• Patient must maintain a low-tyramine diet and avoid foods rich in tyramine • Tranycypromine should be taken early in the day to reduce insomnia • MAOIs are to be discontinued 2 weeks prior to starting another antidepressant or serotonergic agent
Sympatholytics					
Propranolol	10-40 mg/d	Contraindications: • Sinus bradycardia, uncompensated congestive heart failure, 2nd or 3rd degree heart block, severe COPD or asthma, hypersensitivity to beta-blockers	• Hypotension, bronchospasm, bradycardia	• Category C • Breast-feeding – not recommended	• Has only been used in a single dose for prevention of PTSD
Prazosin	Target 6 – 10 mg/d Start with 1 mg at bedtime and increase as blood pressure allows.	• Hypersensitivity to quinazolines • Conurrent use of phosphodiesterase type-5 inhibitors	• First dose syncope	• Category C • Breast-feeding – effects unknown	• Primarily used for management of recurrent distressing dreams

Agent	*Oral Dose	Contraindications	Adverse Events	Pregnancy Category	Remarks
Novel Antidepressants					
Bupropion Nefazodone Trazodone Venlafaxine Mirtazapine	150 – 450 mg/d 300 – 600 mg/d 300 – 600 mg/d 150 – 375 mg/d 30 – 60 mg/d	Contraindications : • MAOI use within 14 days (all) • Hypersensitivity Bupropion – single doses of regular-release >150 mg/d and total daily dose >450 mg/d. • History of seizures, anorexia or buliemia- Nefazodone • Active liver disease or increased liver enzymes • Use with carbamazepine, pimozide, cisapride, triazolam, and alprazolam	• Bupropion: headache, insomnia, dizziness, weight loss, decreased appetite, anxiety, agitation, nervousness, sleep disturbances • Nefazodone: hepatotoxicity • Trazodone and nefazodone: sedation, rare priapism • Venlafaxine: hypertension in patients with pre-existing hypertension, headache, insomnia, somnolence, nervousness, dizziness, anorexia • Mirtazapine: weight gain, increase appetite, somnolence, dry mouth	• Category C (all)	• Need to taper venlafaxine to prevent rebound signs/symptoms • The group has a lower rate of sexual dysfunction compared to SSRIs • Obtain baseline LFTs when treating with nefazodone • Nefazodone is a potent CYP3A4 inhibitor • St. Johns Wort may increase mirtazapine's metabolism

Agent	*Oral Dose	Contraindications	Adverse Events	Pregnancy Category	Remarks
Anticonvulsants					
Carbamazepine	target 400 – 1600 mg/d	Contraindications: • bone marrow suppression, particularly leukopenia • hypersensitivity to carbamazepine, pimozide, or tricyclic antidepressants • MAOI use within 14 days • Concurrent use of nefazodone	• bone marrow suppression, aplastic anemia, leukopenia, SIADH, drowsiness, ataxia, photosensitivity, serious dermatologic reactions, including Stevens-Johnson syndrome, A-V block, and bradycardia	• Category D • Excreted into breast milk in high concentrations; measurable in infant serum.	• Therapeutic blood concentration are not established for PTSD, but monitoring may be useful in cases of suspected toxicity (usual range 4 – 12 mcg/mL) • Strong inducer of CYP 1A2, 2B6, 2C8, 2C9, 2C19, and 3A4. Induction can reduce effectiveness of many medication, such as oral contraceptives
Gabapentin	target 300 – 3600 mg/d	• renal impairment	• sedation, ataxia • peripheral edema	• Category C • Excreted into breast milk; effects unknown	
Lamotrigine	Not taking divalproex or CBZ: 25 mg once a day for 2 weeks, then 50 mg/day for 2 weeks, then 100 mg/day for 1 week	• increased rash with valproate; max dose of 200 mg	• Stevens-Johnson syndrome • Fatigue • Headache • Peripheral edema • Rash • Vision changes	• Category C • Excreted in breast milk in measurable level	Adjust dose base on renal function • Taking divalproex: 25 mg every other day for 2 weeks, then 25 mg/day for 2 weeks, then 50 mg/day for 1 week, then 100 mg/day • Taking enzyme-inducing drug (eg, CBZ): 50 mg/day for 2 weeks, then 100 mg/day for 2 weeks, then 200 mg/day for 1 week, then 300 mg/day for 1 week
Topiramate	target 200 – 400 mg/d. Start with 25 – 50 mg/d and increase by 15 – 50 mg/week to maximum dose or as tolerated.	• hepatic impairment	• angle closure glaucoma • sedation • dizziness • ataxia • cognitive impairment • weight loss • paresthesia • vision changes	• Category C • Excreted into breast milk; breast-feeding not recommended	
Valproate	target 10 – 15 mg/kg/d	• impaired liver function, thrombocytopenia	• nausea/vomiting • sedation • ataxia • thrombocytopenia • Alopecia • Weight gain • pancreatitis	• Category D • Low concentrations in breast milk and infant. Theoretical risk for hepatotoxicity or thrombocytopenia. Monitor for jaundice, liver damage, bleeding.	

Agent	*Oral Dose	Contraindications	Adverse Events	Pregnancy Category	Remarks
Benzodiazepines					
Clonazepam Lorazepam Alprazolam Diazepam	Start - 0.25 mg bid, increase by 0.25 mg every 1-2 days; maximum 20 mg/d 2 – 4 mg/d, 1.5 to 6 mg/d, 10 - 40 mg/d	Contraindications: • hypersensitivity • significant liver disease (clonazepam) • narrow angle glaucoma • severe respiratory insufficiency (lorazepam) • Caution in elderly patients and patients with impaired liver function. • Risk of abuse in patients with history of substance abuse	• sedation • memory impairment • ataxia • dependence • confusion • hypotension	• Category D (all) • All enter breast milk; breast-feeding not recommended	• If doses sustained > 2 months at therapeutic doses, then drug should be tapered over 4-week period • Alprazolam – concern with rebound anxiety
Typical antipsychotics					
Chlorpromazine Haloperidol	100 – 800 mg/d 2 – 20 mg/d	Contraindication : • Parkinson's disease • QTc prolongation or concurrent use with medications that prolong the QTc interval • Severe CNS depression • Hypersensitivity	• Sedation • Orthostatic hypotension with chlorpromazine, thioridazine· • Akathisia • Dystonia • Drug-induced parkinsonism • Tardive dyskinesia may occur with all anti-psychotics with long-term use. • Neuroleptic malignant syndrome • QTc changes	• Category C (all) • All enter breast milk; not recommended	• Therapeutic doses not established in the treatment of PTSD • Use should be well justified in medical record because of the risk of tardive dyskinesia.

Agent	*Oral Dose	Contraindications	Adverse Events	Pregnancy Category	Remarks
Atypical antipsychotics					
Olanzapine Quetiapine Risperidone	5 – 20 mg/d 300 – 800 mg/d 1 – 6 mg/d	Relative contraindication: • Parkinson's disease • Hypersensitivity	• Sedation • Weight gain • Neuroleptic malignant syndrome • Higher doses may cause akathisa, drug-induced parkinsonism, especially with risperidone doses >6 mg/d	Olanzapine: Category C Quetiapine: Category C Risperidone: Category C • All excreted into breast milk; not recommended; use with caution	• Therapeutic doses not established for PTSD • Weight gain occurs with all agents; however, olanzapine produces significantly greater gain • The relative risk of tardive dyskinesia compared to typical antipsychotics has not been established for these agents • Monitor for development of diabetes/hyperglycemia , increased cholesterol, and trigylcerides
Non-benzodiazepine					
hypnotics - Zaleplon - Zolpidem	 5 – 10 mg/d 5 – 10 mg/d	Contraindications: • Hypersensitivity Precautions: • Caution with alcohol/drug abuse history • Caution in elderly and patients with liver dysfunction	• Sedation • Ataxia • Rebound insomnia may occur • Dizziness • Headache • Behavioral changes, bizarre behavior, hazardous activities while asleep	• Category C (both) • Enters breast milk; avoid zaleplon; use zolpidem with caution	• Abuse has occurred, resulting in withdrawal reactions • Zolpidem is a CYP3A4 substrate and its metabolism can be decreased by 3A4 inhibitors
anti-anxiety - Buspirone	20 – 60 mg/d	Precaution: • MAOI use within 14 days	• Nausea • Headache • Dizziness • Drowsiness	• Category B • Excretion into breast milk unknown; not recommended	• Buspirone is a CYP3A4 substrate, and its metabolism can be decreased by 3A4 inhibitors

*Dose adjustments may be necessary in renal or hepatic impairment

D. ADJUNCTIVE SERVICES

D1. Psychosocial Rehabilitation

BACKGROUND

Patients with chronic PTSD may develop a persistent incapacitating mental illness marked by severe and intolerable symptoms; marital, social, and vocational disability; and extensive use of psychiatric and community services. These patients may sometimes benefit more from therapeutic intervention that facilitates generalizing skills for coping with PTSD from clinic to home/work/community, such as case management and psychosocial rehabilitation, than from psycho- or pharmacotherapy.

Psychosocial Rehabilitation involves clinicians providing family psychoeducation, supported employment, supported education, and supported housing; some serving as case managers; or others working with peer counselors. VHA's Uniform Mental Health Services policies (VHA Handbook, 2009) now mandate psychosocial rehabilitation, expanding such services from inpatient units to outpatient programs in Primary Care settings, Outpatient clinics, Community-Based Outpatient Clinics (CBOCs), Vet Centers, and Home-Based Care programs and in partnerships with agencies and providers in communities.

RECOMMENDATIONS

1. Consider psychosocial rehabilitation techniques once the client and clinician identify the following kinds of problems associated with the diagnosis of PTSD: persistent high-risk behaviors, lack of self-care/independent living skills, homelessness, interactions with a family that does not understand PTSD, socially inactive, unemployed, and encounters with barriers to various forms of treatment/rehabilitation services.
2. Patient and clinician should determine whether such problems are associated with core symptoms of PTSD and, if so, ensure that rehabilitation techniques are used as a contextual vehicle for alleviating PTSD symptoms.
3. Psychosocial rehabilitation should occur concurrently or shortly after a course of treatment for PTSD, since psychosocial rehabilitation is not trauma-focused.

DISCUSSION

Penk and Flannery (2000) listed seven forms of psychosocial rehabilitation as clinical practice guidelines for Post-Traumatic Stress Disorder (PTSD):

1. Patient education services
2. Self-Care and Independent Living Skills Techniques
3. Supported Housing
4. Marital/Family Skills Training
5. Social Skills Training
6. Vocational Rehabilitation
7. Case Management

A decade later, Penk & Ainspan (2009) suggested adding to this list: 8) Physical health and well-being and computer-assisted self-management training in reducing PTSD and other mental disorders, such as addictions and depression.

Table I - 10 Adjunctive Problem-Focused Method/Services

If the client and clinician together conclude that the patient with PTSD:		Service/Training
1	Is not fully informed about aspects of health needs and does not avoid high-risk behaviors (e.g., PTSD, substance use)	Provide patient education
2	Does not have sufficient self-care and independent living skills	Refer to self-care/independent living skills training services
3	Does not have safe, decent, affordable, stable housing that is consistent with treatment goals	Use and/or refer to supported housing services
4	Does not have a family that is actively supportive and/or knowledgeable about treatment for PTSD	Implement family skills training
5	Is not socially active	Implement social skills training
6	Does not have a job that provides adequate income and/or fully uses his or her training and skills	Implement vocational rehabilitation training
7	Is unable to locate and coordinate access to services, such as those listed above	Use case management services
8	Does request spiritual support	Provide access to religious/spiritual advisors and/or other resources
	OTHER CONDITIONS	
9	Does have a borderline personality disorder typified by parasuicidal behaviors	Consider Dialectical Behavioral Therapy
10	Does have concurrent substance abuse problem	Integrated PTSD substance abuse treatment

The empirical literature on group treatment for PTSD has grown since the publication of the first edition of the Treatment Guidelines for PTSD.

Evidence-based research from randomized clinical trials is now available to support recommending psychosocial rehabilitation when treating veterans (Glynn, Drebing, & Penk, 2009). Psychosocial Rehabilitations are not limited to veterans with schizophrenia or other psychoses. Psychosocial rehabilitations are recognized as efficacious in treating Post-Traumatic Stress Disorders (PTSDs), Major Depression Disorders (MDDs), and Addictions, especially when mental health practices are delivered through self-management manuals and the Internet, integrated into supported education and supported employment

The psychosocial rehabilitation model may include medication as needed, skills training designed to assist veterans to live productively in the community, and various forms of psychotherapy. Integrating trauma-focused psychotherapies with psychosocial rehabilitation is currently under-utilized, but new interventions are being empirically validated to bring together several forms of treatments and rehabilitation for PTSD.

Models of Psychosocial Rehabilitation Services

1. Education
    - Family psychoeducation is the process of providing education and coping skills for veterans and their families about relevant medical and mental disorders. Examples of such psychosocial rehabilitation are the family interventions for PTSD developed at the VA in West Los Angeles and manualized approaches designed by Sherman, Sautter, Lyons, Manguno-Mire, Han, and Perry (2005), delivered at VHA medical centers in VISN 16—Oklahoma City, Jackson, and Houston.

- Family psychoeducation generally takes place in multi-family groups (producing the added benefit of augmenting social support), but such techniques can also be given in single-family formats or even by books or online (e.g., Sherman & Sherman, 2005).
- Family psychoeducation is noted for fostering social support, challenging a key symptom in Post-Traumatic Stress Disorder (PTSD), which is characterized by social avoidance and isolation. Precautions are needed in fielding family psychoeducation among many different families, since consent of each individual is always required when information is shared about a veteran's illness and/or about families' symptoms and ways of coping. Family psychoeducation is a treatment modality in which families are a partner in providing services to each other: Families are not objects in treatment.
- Family psychoeducation is effective, particularly for PTSD (Glynn, Drebing, & Penk, 2009), and hence is well regarded in the VHA and emphasized in mental health services. Studies from different countries over the past 20 years show that family psychoeducation reduces the rates of re-hospitalization by an average of 50 percent.

2. Self-Care and Independent Living Skills Techniques
    - While social rehabilitative therapies (i.e., teaching social, coping, and life function skills) have been proven to be effective in chronic schizophrenic and other persistently impaired psychiatric cohorts, they have yet to be formally tested with PTSD clients. Since they appear to generalize well from clients with one mental disorder to another, it is reasonable to expect that they will also work with PTSD clients. There is clinical consensus that appropriate outcomes would be improvement in self-care, family function, independent living, social skills, and maintenance of employment.
    - Given the positive impact of independent skills training techniques for mental disorders in general (Halford et al., 1995), PTSD-centered modules should be developed and tested for effectiveness.

3. Supported Housing
    - VHA, for decades, has offered support for housing through residential care programs, such as residential care in inpatient units, domiciliaries, affiliations with state and local housing resources, vouchers for single-room occupancy, and congregate housing in private homes.
    - Forms of housing that are considered more effective are those in which clinical services are integrated or efforts are made by the treating staff to foster community living (Goldfinger et al., 1997; Schutt & Garrett, 1992).
    - Existing literature for persons with other forms of mental illness demonstrates that case management linked to specialized clinical services is more effective than "single-room occupancy" or "warehousing" in shelters without other forms of support (Goldfinger et al., 1997).
    - The greatest risk to ending housing arrangement and likelihood of discontinuing rehabilitation arises from addictions (Goldfinger, Schutt, Tolomiczenko, Seidman, Penk, Turner, 1999; Rog, 2000; Tsemberis & Eisenberg, 2000; Culhane, Metraux, & Hadley, 2002). Thus, interventions that provide housing support are critical to success in rehabilitation (Mares, Kasprow, & Rosenheck, 2004).

- Research on outcomes for compensated work therapy transitional residence model (CWT/TR) have shown that such endeavors indeed are quite successful in transitioning homeless, unemployed veterans who have been hospitalized in inpatient units from VA medical centers to independent living in the community (Schutt, Rosenheck, Penk, Drebing, & Seibyl, 2005). The program requires that unemployed, homeless veterans work in CWT (and, later, other jobs) in order to gain access to VHA housing for a limited time before transitioning to housing on one's own or in private congregate housing with other veterans.
- Outcome studies show that such interventions are successful in promoting tenure in jobs and in personal living arrangements and promoting healthier styles of living, as well as lowering costs due to reduce recidivism, (Cook, 2001; McKay, Johnsen, Banks and Stein, 2005; Cowell, Pollio, North, et al, 2003; Pelletier, Ngyuen, Bradley, et al,2005.

4. Marital/Family Skills Training
    - Marital and family treatments for trauma survivors fall into one of two general categories: systemic approaches designed to treat marital or family disruption, and supportive approaches designed to help family members offer support for an individual being treated for PTSD. These treatments are usually provided as an adjunct to other forms of treatment that are designed to directly address the PTSD symptoms.
    - A single, low-quality RCT compared the addition of family therapy to individual therapy for war veterans with PTSD (Glynn et al., 1999). It found no significant benefit to the addition of behavioral family therapy (BFT), largely due to a high dropout rate, nor did it add significantly to the treatment of PTSD with direct therapeutic exposure (DTE) (an individual psychotherapy technique).
    - There are no research studies on the effectiveness of marital/family therapy for the treatment of PTSD. However, because of trauma's unique effects on interpersonal relatedness, clinical wisdom indicates that spouses and families be included in the treatment of those with PTSD. Of note, marriage counselling is typically contraindicated in cases of domestic violence, until the batterer has been successfully (individually) rehabilitated.

5. Social Skills Training
    - Effectiveness of social skills training has been well demonstrated over many years in many RCTs but not specifically for PTSD (Dilk & Bond, 1996).
    - Effectiveness of social skills training has been demonstrated to reduce social isolation of persons with severe mental disorders (e.g., schizophrenia); similar techniques may be promising for PTSD, particularly if adapted to address antecedent conditions involved in trauma and its consequences (Foa & Rothbaum, 1991).

6. Vocational Rehabilitation
    - Effectiveness of vocational rehabilitation techniques in treating mental disorders has been demonstrated under controlled experimental conditions (Bell & Lysaker, 1996; Bell et al., 1996; Bell et al., 1993; Bond et al., 1997) and controlled clinical studies (Anthony et al., 1995; Drake, 1996; Lehman, 1995; Lysaker et al., 1993).

- As a form of psychosocial rehabilitation, Supported Employment (SE) means that individuals with mental health disorders learn how to find and keep regular, real-world jobs in the community. In SE, vocational rehabilitation specialists provide continuous support to assist veterans achieve success at work. Outcomes for SE have been shown to be much better than for traditional approaches, and this finding has been replicated in several countries (Bond, Drake and Mueser, 1997; Latimer, Lecomte, Becker, et al., 2006; Oldman, Thomson, Calsaferri, et al., 2005).
- Strong outcome data exist to support the efficacy of Supported Employment (SE) for veterans with medical and mental disorders (Glynn, Drebing, & Penk, 2009).
- SE consists of many different kinds of interventions, including the "place-and-train" model that uses on-the-job training within and outside VA medical centers (Penk, 2000).
- A Cochrane Report reviewed eighteen randomized controlled trials among non-veteran and veteran samples, mostly those with serious mental disorders, and found that SE was superior to programs that offered pre-vocational training (Crowther, Marshall, Bond, and Huxley, 2001).
- SE was found to be associated with fewer crises, less chaos, more structure, and on-going support from vocational rehabilitation specialists, because consumers now focus on developing their lives in the community and managing their illness more independently (Bond, Becker, and Drake, 2001).
- Effect sizes for treating PTSD with Supported Employment are sizable (e.g., Glynn, Drebing, & Penk; 2009; Drebing, Van Ormer, Rosenheck, Rounsaville, Herz, & Penk, 2005; Drebing, Van Ormer, Schutt, Krebs, Losardo, Boyd, Penk, & Rosenheck, 2004; Drebing, Van Ormer, Rosenheck, Rounsaville, Herz, & Penk, 2005; Rogers, Anthony, Lyass, & Penk, 2006; Drebing, Van Ormer, Mueller, Hebert, Penk, Petry, Rosenheck, & Rounsaville, 2007).

7. Case Management

   Although case management has been shown to be useful for a range of other psychiatric disorders, there is currently no evidence available from RCTs or from systematic reviews to support or reject the use of case management for PTSD patients.

   - Among populations with histories of trauma, the assertive community treatment models have been empirically validated under controlled (but not with random assignment) conditions (Mueser et al., 1998).
   - Most of the research that empirically validates case management has been conducted among persons with severe mental disorders (Mueser et al., 1998), presumably including persons with co-occurring PTSD and other disorders.
   - Evidence suggests that outcomes are more favorable for intensive case management (well-trained clinician teaches client psychosocial rehabilitation skills in the client's home/community) than for simple case management (clinician links client to needed services).
   - Case management has been demonstrated to reduce in-patient hospitalizations and severe symptoms, as well as stabilize housing for formerly homeless persons; however, there is little evidence to suggest that case management improves vocational adjustment/social functioning (Mueser et al., 1998).

D2. Spiritual Support

BACKGROUND

Religion may provide a framework by which many survivors of trauma construct a meaningful account of their experience and may be a useful focus for intervention with trauma survivors. The terms "religious" and "spiritual" are both used in the clinical literature to refer to beliefs and practices to which individuals may turn for support following a traumatic event.

DISCUSSION

There is a large body of anecdotal literature documenting the propensity of individuals to seek religious/spiritual comfort following a traumatic event. The terrorist attacks of September 11, 2001 provide a recent instance of this phenomenon. Meisenhelder (2002) noted that "the events of September 11, 2001 triggered . . . an increase in attendance in religious services and practices immediately following the tragic events." Schuster and colleagues (2001) performed a nationwide phone survey of 569 adults within a week of the attacks, and found that 90 percent reported coping by "turning to religion."

A study of help-seeking military veterans found significant associations between negative religious coping, lack of forgiveness, and worse PTSD and depression symptoms (Witvliet et al., 2004). Similarly, loss of religious faith was found to be associated with greater utilization of mental health services among military veterans in treatment for PTSD (Fontana & Rosenheck, 2004).

In a study of religiously active trauma survivors, positive relationships were found between a measure of positive religious coping, seeking spiritual support, and posttraumatic growth. In the same study a negative religious coping indicator, religious strain, was associated with increased post-traumatic symptoms (Harris et al., 2008). Hypothetical pathways for positive physical/mental health benefits from religious/spiritual practice include; (1) reduction of behavioral risks through healthy religious lifestyles (e.g., less drinking or smoking), (2) expanded social support through involvement in spiritual communities, (3) enhancement of coping skills and helpful cognitive appraisals resulting in meaning making, and (4) physiological mechanisms such as activation of the "relaxation response" through prayer or meditation.

Chaplains/pastoral care teams work can work in close collaboration with mental health providers to ensure that patients who desire it are presented with a spiritual care experience that results in emotional comfort and improved satisfaction with care (Clark et al., 2003). For some, Chaplains may play an important role in helping individuals regain a sense that their basic life assumptions are true. They can also provide opportunities for participation in prayers, mantras, rites, and rituals, and appropriate end-of life care as determined important by the patient (Canda & Phaobtong, 1992; Lee, 1997). Often, Chaplains represent the first source of support sought by those experiencing PTSD symptoms. The act of talking to a Chaplain is unlikely to be accompanied by the same perception of stigma as the seeking of mental health treatment, and, in active duty military settings, Chaplains are more able to provide confidentiality than their mental health provider colleagues. Therefore, in addition to providing counseling services, Chaplains can play a key role in encouraging participation in treatment for those who may require it. Finally, Chaplains can often provide an important link to the larger community for those with PTSD who have limited social participation.

EVIDENCE

	Recommendation	Sources	LE	QE	SR
1	Assess for spiritual needs and facilitate access to spiritual/religious care when sought	Canda and Phaobtong, 1992 Clark et al., 2003 Fontana. 2004 Harris et al., 2008 Lee, 1997 Witvliet et al., 2004	II, III	Poor	I

LE =Level of Evidence; QE = Quality of Evidence; SR = Strength of Recommendation (see Appendix A)

E. SOMATIC TREATMENT

E1. Biomedical Somatic Therapies

OBJECTIVE

Evaluate the evidence for efficacy of Biomedical Somatic Therapies, including Electroconvulsive Therapy (ECT), Cranial Electrotherapy Stimulation (CES), Vagal Nerve Stimulation (VNS), Repetitive Transcranial Magnetic Stimulation (rTMS), and Deep Brain Stimulation (DBS), in the treatment of PTSD.

BACKGROUND

There has been little research studying these modalities in the treatment of PTSD. ECT has strong research support in the treatment of refractory depression. VNS, rTMS, and CES have been cleared for marketing by the FDA for the treatment of depression, and DBS has been given a humanitarian exemption clearance for marketing for the treatment of Obsessive Compulsive Disorder. None of these modalities has been approved for the treatment of PTSD.

RECOMMENDATIONS

1. There is insufficient evidence to recommend the use of any of the Biomedical Somatic Therapies for first-line treatment of PTSD. [D]
2. ECT and rTMS may be considered as an alternative in chronic, severe, medication- and psychotherapy-resistant PTSD. [B]

DISCUSSION

Although there is significant interest in biomedical somatic interventions in PTSD, there is no evidence for their use as a first-line treatment for PTSD. ECT and rTMS may be beneficial in chronic, treatment-resistant PTSD; however, their use has to be further studied in larger patient populations and specifically in combat veterans.

Electroconvulsive Therapy (ECT)

Watts (2007) reports a VA retrospective chart review study of 12 hospitalized Vietnam veterans with severe refractory depression (including bipolar depression) with co-morbid PTSD who underwent a course of ECT. Results showed good response for depressive symptoms but minimal response for PTSD symptoms.

Margoob et al. (2010) reports on an open ECT trial for 20 patients (17 completers) with severe, chronic, antidepressant- and CBT-refractory PTSD who were prospectively treated with a fixed course of 6 bilateral ECT treatments on an outpatient basis. The improvement in PTSD (40 percent), measured by CAPS, was independent of the improvement in depression (57 percent), and treatment gains were maintained at 4-6 months of follow-up.

Repetitive Transcranial Magnetic Stimulation (rTMS)

Rosenberg (2002) added rTMS to standard antidepressant therapy in 12 patients with PTSD and found that depression responded strongly but that PTSD benefits were minimal.

Osuch (2009) studied rTMS as an adjunct to exposure therapy and existing medications in 9 patients with co-morbid major depression and PTSD in a double-blind crossover study that included a sham arm and found a decrease in hyperarousal symptoms alone.

Cohen (2004) reported findings of an RCT that showed significant improvement in PTSD core symptoms of re-experiencing and avoidance, but only when a 10-Hertz treatment was delivered (note that Osuch utilized no more than 5-Hertz strength).

Boggio et al. (2009) studied the efficacy of 20 Hz rTMS of either the right or left dorsolateral prefrontal cortex (DLPFC) as compared to sham rTMS in 30 patients with chronic PTSD in a double blind, placebo-controlled trial with a sham arm. Both active conditions—20 Hz rTMS of the left and right DLPFC—induced a significant decrease in PTSD symptoms, based on the PTSD Checklist and Treatment Outcome PTSD Scale; however, right rTMS induced a larger effect than left rTMS. Improvements in PTSD symptoms were still significant at the 3-month follow-up. Neuropsychological evaluation showed that active 20 Hz rTMS was not associated with cognitive worsening in patients with PTSD.

Vagal Nerve Stimulation (VNS)

There is one open pilot study of Vagal Nerve Stimulation for treatment-resistant anxiety disorders (George, 2008) that included two patients with PTSD. This study does not provide sufficient evidence on which to base a recommendation regarding the use of VNS in the treatment of PTSD.

Although there has been significant interest and widespread utilization of CES in the treatment of PTSD, there is insufficient evidence for or against its use.

Conclusion:

While intriguing, the findings from these studies are limited by a small number of patients and co-morbid symptomatology and do not provide adequate support to recommend any of the biomedical somatic interventions as a first-line treatment for PTSD. rTMS and ECT have had initial evidence of possible benefits in chronic, treatment-resistant PTSD; however, more studies in larger patient populations are needed.

EVIDENCE TABLE

	Evidence	Source	LE	QE	SR
1	Any Biomedical Somatic Therapies for first-line treatment of PTSD				D
2	ECT – for PTSD co-morbid with severe refractory depression	Watts, 2007 Margoob, 2010	II-3 II-2	Fair	B
3	rTMS - Good PTSD outcome at higher frequency Primarily for co-morbid depression added to antidepressant. Depression benefit robust	Burt ,2002	III	Poor	B
		Cohen, 2004 Boggio, 2009	I I	Good	
		Rosenberg, 2002 Osuch, 2009	I II-1	Fair	
4	VNS - vagus nerve stimulation (VNS) for treatment-resistant anxiety disorders	George, 2008	I	Poor	I

LE=Level of Evidence QE=Quality of Evidence; SR= Strength of Recommendation (see Appendix A)

E2. Acupuncture

OBJECTIVE

Improve management of PTSD symptoms, particularly when accompanied by associated symptoms of chronic pain, depression, insomnia, anxiety, or substance abuse.

BACKGROUND

The practice of needling in acupuncture to mediate pain, one of the well-accepted indications for acupuncture, is thought to occur through the production of endogenous monoamines and neuropeptides. Besides activating neurohumoral pathways, acupuncture stimulates neural connections associated with the Autonomic Nervous System, prefrontal cortex, and limbic system, all structures thought to regulate the pathophysiology of PTSD. Acupuncture investigation for the treatment of PTSD has been limited to, at best, two (English) RCTs. However, symptomatic relief of disturbances associated with PTSD symptom clusters enhances the consideration of the use of this modality.

RECOMMENDATIONS

1. Acupuncture may be considered as treatment for patients with PTSD. [B]

DISCUSSION

Research focusing on the efficacy of acupuncture is still relatively limited. The few available studies are well done and demonstrate significant improvement in both PTSD and PTSD-associated symptomatology. A larger numbers of studies exist, concluding acupuncture's efficacy in pain management, insomnia, depression, and substance abuse.

Hollifield et al. (2007) evaluated the potential efficacy and acceptability of acupuncture for the treatment of PTSD. Individuals diagnosed with PTSD were randomized to an acupuncture treatment group (ACU), a cognitive-behavioral therapy group (CBT), or a wait list control group (WLC). The primary outcome measure was self-reported PTSD symptoms at baseline, end treatment, and 3-month follow-up. Repeated measures MANOVA was used to detect predicted Group X Time effects in both intent-to-treat (ITT) and treatment completion models. Compared with the WLC condition in the ITT model, acupuncture provided large treatment effects for PTSD ($F [1, 46] = 12.60$; $p < 0.01$;

Cohen's d = 1.29), similar in magnitude to group CBT (F [1, 47] = 12.45; p < 0.01; d = 1.42) (ACU vs. CBT, d = 0.29). Symptom reductions at end treatment were maintained at the 3-month follow-up for both interventions.

A recent unpublished DoD/VA RCT studied 55 active duty members with PTSD, randomized to PTSD treatment as usual (TAU) and PTSD treatment as usual plus eight 90 minute acupuncture sessions delivered twice weekly for four weeks (TAU + Acupuncture). Outcome measures included: Clinician-Administered PTSD Scale (CAPS), PTSD Checklist (PCL), Becks Depression Inventory (BDI I-II), Numeric Rating Scale for Pain (NRS), and SF-36v2. Follow-up was at baseline and 4, 8, and 12 weeks post-randomization. Compared to usual PTSD care, a 4-week course of twice-weekly acupuncture resulted in significantly greater improvement in PTSD symptoms (Pre-post ES 1.4-1.6 versus usual care ES 0.12-0.74), significant improvement in depression, and significant improvement in pain.

EVIDENCE TABLE

	Evidence	Source	LE	QE	SR
1	There is some evidence that acupuncture may be helpful with the management of Post-Traumatic Stress Disorder, acute or chronic.	Hollified et al., 2007	I	Good	B

LE=Level of Evidence QE=Quality of Evidence; SR= Strength of Recommendation (see Appendix A)

F. COMPLEMENTARY AND ALTERNATIVE MEDICINE

OBJECTIVE

Identify interventions derived from traditional and nontraditional complementary approaches that may provide effective first-line or adjunctive treatment for PTSD.

BACKGROUND

Complementary and alternative medicine (CAM) is a group of diverse medical practices, products, and systems that are not generally considered part of conventional medicine. While there is limited evidence to suggest that any of the CAM therapies listed below are efficacious for PTSD, these interventions may be of value in dealing with other symptoms (particularly those associated with hyperarousal) or co-morbid conditions. There is little evidence that these interventions are harmful. Some patients who may be reluctant to accept mental health labels or interventions may be more accepting of these novel treatment approaches. CAM interventions are affordable and generally accessible in communities across the nation. Many CAM therapies are practiced in a group setting, which may have the added benefit of increasing socialization. CAM programs may be engaged as a family that could increase social support and reduce stress for all family members. CAM approaches often provide an increased sense of mastery and control that may promote greater resilience.

Since complementary medicine may relate to particular cultural backgrounds or other belief systems, health professionals should be aware of, and sensitive to, the needs and desires of the patient and the family. Health professionals should be willing to discuss the effectiveness of therapy and different options of care within the context of the current healthcare system.

CAM Modalities:

CAM modalities are typically grouped into broad categories reflecting putative mechanism of action: However, this is more for convenience than any specific biologic underpinnings. Many modalities, such as acupuncture, span more than one category. Generally consistent with the schema offered by the National Center for Complimentary and Alternative Medicine these groups are:

Natural Products (Biologically Based Practices): Biologically based therapies that use natural substances (e.g., herbs, foods, vitamins, dietary supplements, homeopathic remedies) to promote healing and wellness.

Mind-Body Medicine: Approaches that seek to enhance general well-being through balancing mind and body. These practices emphasize the use of the mind, the body, or a combination of mental and physical activities to harmonize mind-body functioning to promote health and well-being. Those that focus primarily on mental activity include prayer and guided imagery. Those that emphasize the integration of mind and body for experiencing more fundamental processes underlying cognitive reflection include meditation, yoga, Tai Chi, breath-oriented therapies, and expressive arts therapies (such as dance, music, and art therapies).

Biofeedback (both neurofeedback that trains one to regulate attentional states via regulation of beta, alpha, theta, and delta EEG spectral analysis, as well as psychophysiological regulation of HR, Respiration, SC, EMG, and HRV) could, like hypnosis, be considered standard approaches to psychotherapy, and be included under that category. Research has shown positive outcomes for biofeedback for pain, sleep, and anxiety. Various relaxation skills (such as progressive muscle relaxation) are utilized as part of biofeedback.

Manipulation and Body Based Practices (Exercise and Movement): These ractices are based on the manipulation of one or more parts or systems of the body. Manipulation of another's body structure including bones and joints, soft tissue, circulatory, and lymphatic systems are found in such disciplines as chiropractic spinal and joint manipulation, osteopathic manipulation of joints and soft tissue, massage therapy such as lymphatic drainage, deep and connective tissue manipulation such as Rolfing, and stimulation of specific points such as reflexology and acupressure. Active movement disciplines that focus on reducing pain through improving movement functioning use practitioners' guidance through hands-on feedback or verbal instruction as is found in Movement Therapies such as the Alexander Technique, Feldenkrais, Laban Movement Analysis, and others.

Energy medicine: Energy-focused practices that involve connecting with and balancing energetic fields that purportedly surround and penetrate the human body (e.g., Qi gong, Reiki, Therapeutic touch).

Whole Medical Systems: Traditional medicine systems based upon comprehensive systems of theory and practice for improving overall health and correcting health imbalances that focus on both improving overall lifestyle (diet exercise, social and emotional functioning), and specific methods of herbal and somatic interventions, such as Ayurvedic Medicine, Traditional Chinese Medicine (including acupuncture), and naturopathy (which combines disciplines from the above categories).

RECOMMENDATIONS

1. There is insufficient evidence to recommend CAM approaches as first line treatments for PTSD. [I]

2. CAM approaches that facilitate a relaxation response (e.g. mindfulness, yoga, acupuncture, massage, and others) may be considered for adjunctive treatment of hyperarousal symptoms, although there is no evidence that these are more effective than standard stress inoculation techniques. [I]

3. CAM approaches may be considered as adjunctive approaches to address some co-morbid conditions (e.g. acupuncture for pain). [C]

4. CAM may facilitate engagement in medical care and may be considered in some patients who refuse evidence-based treatments. However, providers should discuss the evidence for effectiveness and risk-benefits of different options, and ensure that the patient is appropriately informed.

DISCUSSION

Surveys of CAM utilization (meditation, yoga, massage, and deep breathing exercises) among the general US population indicate significant increases in acceptance of these practices over the past decade (Barnes et al., 2008). A recent White House Commission report on CAM highlighted the need for continued rigorous research regarding these approaches (White House Commission, 2002).

Although Complementary and Alternative Medicine (CAM) approaches to the treatment of many medical and mental health diagnoses, including PTSD, are in widespread use, the research base to support their effectiveness is far from complete. Evidence for their use for PTSD is sparse, yet numerous CAM modalities have been shown to be effective for symptomatic relief related to insomnia, anxiety, pain (AHRQ, 2009), and various somatic presentations associated with PTSD.

F1. Natural Products (Biologically Based Practices)

Herbal (phytotherapy) and dietary supplements have been used for the treatment of PTSD. Herbs and supplements are believed to boost health functioning through micronutrients that are directly used by body tissues, either targeting a specific organ or system, or through balancing systems that interact with each other. Although individual consumers may purchase individual herbs or vitamins, practitioners typically recommend combinations based upon both the suspected pathology as well as the patient in order to boost their host resistance. An example of specific over-the-counter supplements is omega-3 fatty acid docosahexaenoic acid (DHA), that affects catecholamines and proinflammatory cytokines, and that has been shown in several RTCs to decrease the perception of stress (Bradbury, 2004), improve mood (Lin & SU, 2007; Mischoulon et al, 2009), and decrease symptoms of ADHD in children (Johnson et al., 2009; Sinn & Bryan, 2007).

Herbal remedies such as Kava Kava have been shown to reduce anxiety (Pitler & Ernst, 2003), while others (valerian root, typically in combinations of herbs) has been shown to improve sleep (Bent et al., 2006). St. John's Wart has also been used to treat mild depression with some benefit compared to placebo (Linde, Berner, & Kriston, 2008). Although patients frequently prefer phytotherapy over prescription medication, often claiming fewer side effects, herbal benefits are typically not as effective as prescription medications, their safety has been questioned, and these have not been well studied in

patients specifically diagnoses with PTSD. Natural precursors to Seratonin and GABA sold over the counter have also shown reductions in anxiety, yet also not within the context of PTSD.

Homeopathic theory is entirely different from herbal medicine and supplements, although both are widely utilized throughout the world. Samuel Hahnemann (Hahnemann, 1996) described how substances that in larger quantities acted as a toxin, in specifically prepared microdoses served to stimulate the body's immune system to repel those symptoms that arise in consort with exposure to a toxin. Each toxic substance known to man has its symptom picture, and therefore a homeopathic preparation of that toxin is thought to stimulate the body to overcome those symptoms. A homeopathic practitioner takes an extensive history in order to best understand which single remedy (or in some cases combinations of remedies) best accounts for the cluster of symptoms, and therefore would be most optimal for stimulating the body's defenses against that symptom cluster. There are several RCT studies for anxiety, sleep, and pain in medical journals, but nothing directly for PTSD or in the context of PTSD Moreover, there is a great deal of controversy regarding the research on homeopathic efficacy, with most studies failing to find an overall benefit (Linde, Jonas, Melchart, & Willich, 2001).

Although there have been some studies of their effectiveness for addressing some components of PTSD, the results of this body of research provides insufficient evidence to draw firm conclusions about their direct effectiveness for PTSD. In addition, the quality and purity of herbals and dietary supplements available in the United States vary widely, further complicating their study and use.

F2. Mind-Body Medicine

Often referred to as "Mind-Body" approaches to health and well-being, methods such as meditation, yoga, and Tai Chi have been used for thousands of years by Eastern religious traditions for spiritual development. They share a common goal of enlightenment – that is, experiencing existence "as it is" prior to our conceptual biases stemming from our needs, fears, and desires. Traditional practice involves developing the ability to become fully immersed in the moment at hand, without cognitive reflection or pursuing or reacting to one's fears or desires. This practice allows one to break out of habitual cognitive and behavioral patterns and become more open and responsive to the situation at hand. It is also thought to lead to a more harmonious mental and physical state of being. However, in the alternative healing culture of modern Western society, these traditional practices have been taken out of their original religious context and greatly simplified to appeal to modern social interests. Several meta-analyses and reviews have shown physical and psychological health benefits from Meditation, Yoga, and Tai Chi. However there is a lack of rigorous RCTs and head to head comparisons of these approaches compared to other interventions for mental health benefits (Ospina, 2007).

Meditation and relaxation techniques, based upon Buddhist meditation practices such as Zen or Vipassana, have been adapted in the West to assist with specific concerns such as anxiety and pain. One such approach, Mindfulness Based Stress Reduction, combines elements of yogic relaxation techniques and Buddhist awareness enhancement in a simple, concrete, and brief structured format that is easy for someone not steeped in Eastern traditions to learn. Transcendental Meditation™, based on Yogic traditions, has been used to treat anxiety and depression in Vietnam veterans (Brooks 1985). There is a growing literature of RCTs showing meditation to be effective for enhancing attention, reducing anxiety and stress, improving sleep, and helping to manage pain (Grossman, Niemann, Schmidt, & Walach, 2004; Kabat-Zinn, Lipworth, & Burney, 1985; Davidson et

al, 2003). However, there is no published RCT in support of the use of meditation for PTSD per se.

Yoga is really a collection of practices to assist one to become "yoked with God." Although there are several Ashrams that emphasize this collection of practices for spiritual enhancement, for the most part, "yoga" is synonymous in the West with Hatha-style Yoga. Hatha Yoga is a series of poses that help stretch muscles, improve tone and alignment, and teach one to breathe into and release one's discomfort. Other types of yoga, such as Pranayama breathing techniques or the restorative and recuperative Yoga Nidra approach, are typically employed as part of Hatha Yoga classes in the West. Many RCTs have been conducted showing the value of yogic practices for improving sleep and reducing anxiety and stress (Kirkwood, tai Rampes, Tuffrey, et al, 2005; Sarang & Telles, 2006). However, there are currently no RCTs published in support of the use of yoga for PTSD per se.

Tai Chi Chuan (literally translated as "Grand Ultimate Fist") was initially developed as a highly effective martial art. However, two hundred years ago a version was created that could be widely practiced by the general populous in order to improve physical health and mental well-being. The series of slow continuous movements synchronized with the expansion and contraction of one's breath, is believed to harmonize mind and body in harmony with one's surroundings. Stemming from the Taoist tradition to enhance health and foster a sense of unity with mind, body, and nature, Chi Kung (also transliterated as Qi Gung), is a series of exercises that include breath and movement. Adopted as part of the Traditional Chinese Medical Model, Chi Kung Exercises are said to help balance and circulate the "chi" (life-energy). Several RCTs have shown Tai Chi (Jin, 1989), and Chi Kung to be effective in improving a sense of calm and well-being, improve sleep, and improve physical health (Wang, Bannuru, Ramel, Kupelnick, et al, 2010). However, the evidence for benefits of Tai Chi compared to regular exercise is lacking.

F3. Manipulation and Body-Based Practices (Exercise and Movement)

Although hardly fitting the definition of a CAM modality, exercise used for psychological well-being is outside of the standard of practice for psychotherapy. However, exercise has been advocated as an integrative approach in the prevention and treatment of PTSD and other combat-related mental health problems. Wald and Taylor (2008) examined the relationship between baseline physical fitness and the development of PTSD symptoms (as measured by the Impact of Event Scale) in a group of 31 soldiers undergoing military survival training. They found that higher levels of pre-study physical fitness were inversely related to both trait anxiety levels and IES scores. Studies have shown that pre-trauma levels of exercise tend to decline after developing PTSD (de Assis et al., 2008). Aside from PTSD, depression was a frequent condition for which exercise therapy was applied. The majority of reviewed studies utilized an aerobic exercise regimen—e.g., walking, running, stationary cycling (Diaz & Motta, 2008; Manger & Motta, 2005). One study emphasized the importance of participant selection of the specific type of exercises that would constitute their treatment (Donta, et al, 2003). The studies reviewed here utilized both group and individual exercise formats. All studies demonstrated either a reduction in symptoms from baseline PTSD measures or relative to a placebo or control group, but the effects were generally modest and did not always extend to other mental health disorders, such as anxiety and depression. A primary methodological limitation of the papers reviewed here is that exercise interventions were rarely conducted in isolation from other psychotherapeutic approaches.

Massage and skeletal manipulation has long been used for reducing the ill effects of physical and mental stress. Deprivation of touch has been seen as problematic for infants, and massage adherents claim that adults as well benefit from non-sexual

massage-style contact. In addition, Swedish-style massage targets the lymphatic system as well as intending to help relax large surface musculature. Deep Tissue and Sports Massage targets deeper skeletal musculature and connective tissue to correct structural imbalances. Many studies have shown the value of such approaches for at least transient stress and pain reduction and in support of sleep and anxiety. However, no research has demonstrated that these modalities are effective for PTSD per se.

F4. Energy medicine

There are a variety of CAM systems where a practitioner helps a patient to correct energy imbalances in their bodies. Practitioners place their hands over or directly upon various energy foci, and attempt to shift excess energy to deficient areas (Yogic Chakra-based approaches), and remove blockages (Traditional Chinese Medicine Chi-based approaches). Acupressure and Shiatsu are mostly based on Traditional Chinese Medicine, but use pressure on specific acupuncture points rather than needles. Reiki and Healing Touch are more energy based, and often the practitioner places their hands above the body of the client, sensing the energy and attempting to allow the energy areas to come into a balance. Chi Kung (Qi Gung) can be utilized by a Chi Kung master trained in Traditional Chinese medicine to assist another person. Many hospitals in China have departments of Traditional Chinese Medicine which include a Chi Kung Master who attempts to balance the chi in the patient by either direct hands-on methods or sitting nearby and placing the hands toward the patient.

Reiki and J*ohrei* are both energy medicine techniques that originated in Japan. In *Reiki*, the practitioner places his hands on or near the person receiving treatment with the intent to transmit *ki*, believed to be life-force energy. *Johrei,* a form of energy healing that originated in Japan, involves the practitioner facing the person receiving the treatment, where “spiritual energy” is transmitted through the practitioner (Brooks, et al., 2006).

There are no current controlled studies examining *Reiki* or *johrei* in patients with PTSD or Acute Stress Disorder. A small number of low-quality studies have been conducted, showing positive improvement in conditions commonly co-morbid with PTSD, such as depression (Collinge, Wentworth, and Sabo, 2005) or anxiety (Brooks et al., 2006).

A recent systematic review of randomized clinical trials of *Reiki* noted that the currently available RCTs are “scarce” and lack independent replication (Lee, Pittler, and Ernst, 2008). The studies that exist suffer from methodological flaws related to sample size, inadequate design, and poor reporting.

F5. Whole Medical Systems

There are two major comprehensive traditional medical systems, and several minor medical systems that integrate lifestyle and intervention across multiple dimensions. Traditional Chinese Medicine utilizes nutrition, exercise, emotional balance, massage, herbs and acupuncture to restore balance to the body in relationship to one’s environment. Chinese medicine has a sophisticated system of diagnosis through assessment of various combinations of pulses, and other physical and mental signs and symptoms. Traditionally, specialists focus on herbal treatment, acupuncture treatment, massage or Chi Kung. However, Western schools teach a combination of these and modern practitioners in the United States typically focus on lifestyle, acupuncture and herbal treatments. Ayurvedic Medicine also emphasizes a healthy lifestyle, including diet and yoga, and offers supportive intervention through expunging toxins, tonifying, and balancing, primarily through herbal treatments. Less popular and less extensively developed systems include traditional healing systems from almost every culture on

earth such as Native American healing traditions, that also focus on living in harmony with one's environment, supported by spiritual healers and herbal remedies. Although there is a great deal of research conducted on aspects of these healing systems (such as acupuncture, or the use of herbs), assessment of comprehensive systems are difficult to study, and lacking in the literature, especially with regard to mental health in general, and PTSD in particular.

F6. Other Approaches

Animal-Assisted Therapy (AAT)

AAT is a goal-directed intervention in which an animal that meets specific criteria is an integral part of the treatment process. AAT is delivered and/or directed by a health/human service provider, working within the scope of his or her profession. AAT is designed to promote improvement in human physical, social, emotional, and/or cognitive functioning. AAT is provided in a variety of settings and may be group or individual in nature. Commonly used animals include dogs and horses. There are a growing number of programs throughout the United States that utilize animals as part of PTSD treatment, including programs specifically for veterans. There are two major approaches to AAT. One simply offers the opportunity to bond with an animal. The other is more structured and occurs within a therapeutic environment. AAT for those suffering from PTSD often ask the patient to engage non-verbally with one or more animals in a structured activity, such as approaching the animal in a safe manner that engages rather than frightens them and lead them through an obstacle course without touching them. This requires developing trust, rapport, and non-verbal communication. Evidence of AAT for PTSD is ongoing but at this point lacks support.

Module I-3. MANAGEMENT OF SPECIFIC SYMPTOMS

This section includes recommendations regarding treatment interventions for a selected list of physical symptoms that are common in patients presenting with post-traumatic stress symptoms.

Survivors of trauma may not complain directly of PTSD symptoms, such as re-experiencing or avoidance. Instead, they may complain of sleeping problems. When seeking to identify PTSD, providers should consider asking specific questions about sleep problems, (including flashbacks and nightmares), pain (including musculoskeletal, headache), or hyperarousal (including an exaggerated startle response or sleep disturbance). Many individuals with PTSD experience sleep disturbances (trouble falling asleep or problems with waking up frequently after falling asleep). Chronic pain and insomnia often occur simultaneously, with the vast majority of chronic pain patients complaining of interrupted or poor quality sleep. When a person with PTSD experiences sleep disturbances, using alcohol as a way to self-medicate becomes a double-edged sword. Excessive alcohol use can impair one's ability to sleep restfully and to cope with trauma memories and stress. The need to improve sleep in these patients is clear, given increasing evidence that sleep disturbance is associated with heightened pain sensitivity and elevated disability.

Chronic pain is frequently observed in patients with PTSD and is often associated with a significant level of affective distress and physical disability. Chronic pain may develop because of an injury sustained in a traumatic event, such as a motor vehicle accident, work-related injury, or injury in military combat. Patients with chronic pain, particularly headache disorders and fibromyalgia (FM), associated with psychological traumas need a special management strategy. Diagnosis of headache disorders and FM in traumatized patients and obtaining the clinical history of a traumatic event or diagnosing PTSD in chronic pain patients are of great importance.

A. Sleep Disturbances

BACKGROUND

Many patients with PTSD have had insomnia for years, including broken sleep, frequent awakenings, and nightmares, all of which contribute to poor sleep quality. Hyperarousal behaviors, part of PTSD symptoms for many people, can be stronger at night and contribute to insomnia. Sleep problems in traumatized patients may also reflect co-morbid conditions, some of which may be of new-onset (pain may be prominent among these).

There is no evidence to suggest that insomnia, as a component of traumatic stress reactions, should be managed differently than insomnia associated with other conditions. Clinical experience does, however, show that some psychologically traumatized patients dread sleep because of intense nightmares.

Research demonstrates that non-pharmacological sleep strategies yield outcomes equal or superior to those obtained with hypnotics alone or hypnotics combined with non-pharmacological strategies. Long-term outcomes are better following non-pharmacological interventions. The aim of sleep management is to establish a regular, normalized sleep-wake pattern.

Sleep Disturbance

1. Encourage patients to practice good sleep hygiene, including:
    - Restricting the night-time sleep period to about eight hours
    - Waking at a regular time
    - Arising from bed at a regular time
    - Avoiding going to bed too early
    - Avoiding alcohol
    - Avoiding stimulants, caffeinated beverages, power/energy drinks, nicotine, and over-the-counter medications
    - Avoiding stimulating activities, light, noise, and temperature extremes before bedtime (e.g., exercise, video games, T.V.) or in the sleeping area
    - Reducing (to less than 30 minutes), or abolishing, daytime naps
    - Practicing relaxation techniques
    - Engaging in moderate exercise, but not immediately before bedtime
2. Offer Cognitive Behavioral Therapy for Insomnia, which may include:
    - Educating about proper sleep habits and sleep needs
    - Correcting false and unrealistic beliefs/concerns about sleep
    - Identifying and addressing anxious, automatic thoughts which disrupt sleep
3. Consider adjunctive therapy for nightmares using prazosin. [B]
4. Any significant change in sleep patterns should trigger clinical reassessment in order to rule out worsening or new onset of co-morbid conditions

Insomnia

1. Monitor symptoms to assess improvement or deterioration and reassess accordingly.
2. Explore cause(s) for insomnia, including co-morbid conditions.
3. Begin treatment for insomnia with non-pharmacological treatments, including sleep hygiene and cognitive behavioral treatment (see recommendation for Sleep Disturbances).
4. The selection of sleep agents for the treatment of insomnia in PTSD patients may be impacted by other treatment decisions (e.g., medications already prescribed for the treatment of PTSD, depression, TBI, pain, or concurrent substance abuse/withdrawal) and social/environmental/logistical concerns associated with deployment.
    a. **Trazodone** may be helpful in management of insomnia and may also supplement the action of other antidepressants.
    b. **Hypnotics** are a second-line approach to the management of insomnia and should only be used for short periods of time. Should hypnotic therapy be indicated, the newer generation of non-benzodiazepines (e.g.

zolpidem, eszopiclone, ramelteon) may have a safety advantage by virtue of their shorter half-life and lower risk of dependency. Patients should be warned of and monitored for the possibility of acute confusional states/bizarre sleep behaviors associated with hypnotic use. Benzodiazepines can be effective in chronic insomnia but may have significant adverse effects (confusion, sedation, intoxication) and significant risk of dependency.

c. **Atypical antipsychotics** should be avoided due to potential adverse effects but may be of value when agitation or other symptoms are severe.

d. If nightmares remain severe, consider adjunctive treatment with **prazosin [B]**

e. If symptoms persist or worsen, refer for evaluation and treatment of insomnia

Additional information of management of insomnia can be found in:

VHA Pharmacy Benefit Management (PBM) guideline for Insomnia: http://www.pbm.va.gov/ClinicalRecommendations.aspx

Sleep Hygiene Patient Education

- Avoid or limit caffeinated products, nicotine, and alcohol, especially later in the day.
- Avoid drinking excess liquids after supper to avoid having to get up during the night to go to the bathroom.
- Avoid or limit daytime naps to 30 minutes in the early afternoon before 3:00 pm.
- Go to bed only when sleepy. Sleep only as much as needed to feel refreshed. Staying in bed longer can result in fragmented/shallow sleep on following nights.
- Create a dark, quiet, temperature-controlled bedroom (e.g., change the number of blankets you use; use earplugs; close the door if noisy).
- Avoid heavy meals within 2 hours of bedtime; a light snack might help if hungry.
- Maintain a regular daily schedule of activities, including bedtime and awakening times, 7 days/week. Use an alarm clock if needed.
- Exercise regularly during the daytime. Avoid active exercise in the late evening when it is close to bedtime.
- Use the bed and bedroom only for sleeping or sexual activity. Do not eat, work, or watch television while in bed.
    - If you cannot sleep, if possible, get out of bed and go to another room; read or engage in other quiet activities; or do other relaxation activities before attempting to sleep again. Return to bed only when sleepy Repeat if necessary. Do not watch the clock; turn the clock around or cover it up
- Solve problems before retiring. If not possible, write down your worries, plans, and strategies during the early evening and not at bedtime.
- Correct extrinsic factors, such as environmental disruption (e.g., pets or snoring partner).
- Establish a "wind-down" routine going to bed and develop and maintain bedtime "rituals" that make going to sleep a familiar routine; for example:
    - Set time to relax before bed with 20-30 minutes of relaxation (e.g., soft music, meditation, breathing exercises
    - Take a warm bath
    - Have a light snack, which could include: warm milk, foods high in tryptophan, such as bananas, carbohydrates, which can help induce sleep

Adapted from Petit L, et al. Age Ageing 2003; 32: 22. Wilson S. and Nutt D. Prescriber 2005; 19: 29-41 Wolkove N, et al. CMAJ 2007; 176: 1449-54.

DISCUSSION

Use of Benzodiazepines for Sleep Disturbance

In a small, double-blind, placebo-controlled temazepam trial in acute accident/injury victims at a trauma center (Mellman et al., 2002), temazepam 30 mg was administered for 5 nights, tapered for 2 nights, then discontinued. At the 6-week follow-up, 6/11 temazepam subjects and 3/11 placebo subjects met PTSD symptom criteria. Sleep improvement was noted, however, for the duration of the trial. However, in a small randomized, controlled trial, alprazolam did not have substantial benefit for PTSD or for nightmares, although it did improve anxiety (Braun, 1990). In another small single-blind controlled study, clonazepam did not demonstrate significant benefit for sleep difficulties, including nightmares (Cates, 2004).

An argument can be made for short-term use of a benzodiazepine for the purpose of reducing hyperarousal symptoms in the immediate trauma aftermath, in order to help

normalize sleep cycles and minimize anxiety. Longer-term use of benzodiazepines, however, should be avoided, as the limited data available show that prolonged use of benzodiazepines (1-6 months in duration) is associated with a higher rate of subsequent PTSD (Gelpin et al., 1996).

Benzodiazepines use should be considered relatively contraindicated in combat veterans with PTSD because of the very high co-morbidity of combat-related PTSD with alcohol misuse and substance use disorders (upwards of 50 percent of co-morbidity) and potential problems with tolerance and dependence. Once initiated in combat veterans, benzodiazepines can be very difficult, if not impossible, to discontinue, due to significant withdrawal symptoms compounded by the underlying PTSD symptoms.

Other agents that have improved insomnia are trazodone, mirtazapine, and olanzapine. There are no trials of non-benzodiazepine hypnotics in the treatment of sleep disorders associated with PTSD.

Use of Prazosin for Sleep Disturbance

Five publications (Raskind 2000, 2002, 2003; Taylor 2006 & 2008) that examined the role of antiadrenergic medications, commonly used for treating hypertension, in the treatment of post-traumatic stress disorder (PTSD) were identified in the peer-reviewed literature.

Although Taylor et al. (2006) and Raskind et al. (2003) were excluded from the analyses (due to the small number of subjects that did not meet inclusion criteria), both have shown positive results in reducing psychological distress, specifically to trauma cues (Taylor et al., 2006). Patients taking prozosin showed significant improvement on the Clinician-Administered PTSD Scale (Raskind, 2003).

Raskind et al. (2007) evaluated prazosin effects on trauma nightmares, sleep quality, global clinical status, dream characteristics, and co-morbid depression. Forty veterans (mean age 56 +/- 9) with chronic PTSD and distressing trauma nightmares and sleep disturbance were randomized to evening prazosin (13.3 +/- 3 mg/day) or placebo for 8 weeks. In the evaluable sample (n = 34), primary outcome measures demonstrated that prazosin was significantly superior to placebo for reducing trauma nightmares and improving sleep quality. Prazosin shifted dream characteristics from those typical of trauma-related nightmares toward those typical of normal dreams.

Taylor et al. (2008) was a double blind, placebo-controlled cross-over study of 13 civilians with trauma-related PTSD. Prazosin was rapidly titrated to 3 mg/night during each 3-week treatment phase. Prazosin, compared with placebo, significantly increased total sleep time by 94 min ($p < 0.01$), and total rapid eye movement (REM) sleep and mean REM duration were also longer with prazosin. Reductions in trauma nightmares, total PTSD symptoms (using the PCL-C), and CGIC scores were significantly changed compared with placebo.

The results of these studies were consistent and positive, suggesting that prazosin therapy is safe and is associated with reduction of nighttime symptoms of PTSD. Prazosin is an effective and well-tolerated treatment for trauma nightmares, sleep disturbance, and global clinical status in veterans with chronic PTSD.

Ruff and colleagues (2009) found in an observational study that prazosin combined with sleep hygiene counseling was an effective initial treatment for a group of OIF/OEF veterans (n=74) with headaches associated with histories of mild TBI from exposure to an explosion in combat and with PTSD. Prazosin was well tolerated. Nine weeks after providing sleep counseling and initiating an increasing dosage schedule of prazosin at bedtime, 65 veterans had reduced headache intensity and frequency, reduced daytime

sleepiness, and improved cognitive performance. These gains were maintained 6 months later.

Sleep hygiene counseling is beneficial in terms of improving sleep duration and reducing the time it takes for a person to fall asleep (Morin et al., 1999). By blocking nightmares in people with PTSD, prazosin prolongs sleep duration by preventing sleep interruptions. Thus, the two interventions may have synergized, with sleep hygiene counseling reducing the time it took for veterans to fall asleep and prazosin prolonging sleep.

A systematic review by Dierks et al. (2007) did not find any additional publication to the above. The authors concluded that despite various limitations, all of the studies showed significant improvements in the sleep-related symptoms of PTSD following the addition of prazosin therapy, based on the Clinician-Administered PTSD Scale recurrent distressing dreams item and the Clinical Global Impression of Change scale.

EVIDENCE TABLE

	Evidence	Source	LE	QE	NB	SR
1	Prazosin improved sleep quality, reduced trauma nightmares	Dierks et al., 2007 § Raskind et al., 2002, 2007 Taylor et al., 2008	I	Good	Mod	B
2	Benzodiazepines for sleep disturbance, insomnia	Gelpin et al., 1996 Mellman et al., 1998	II-2	Fair	Small/ Neg	C

LE =Level of Evidence; QE = Quality of Evidence; SR = Strength of Recommendation; NB=Net benefit (see Appendix A)

Table I - 11 Pharmacological Studies - Prazosin for Sleep Disturbances

Author, Year	Results	n	Trauma	LE	QE	NB
Raskind et al., 2002	Significant improvement in dream scores after 8 weeks of prazosin	59	Retrospective study - Veterans	II-2	Poor	Mod
Raskind et al., 2003	Significant improvement, CAPS, CGI. Prazosin > placebo	10	Veterans	I	Poor	Mod
Raskind et al., 2007	No difference	34	Veterans	I	Good	Mod
Taylor et al., 2006	Reduction in global PTSD illness severity	11	Civilian	II	Poor	Mod
Taylor et al., 2008	Reductions of nighttime, significantly increased total sleep time	13	Civilian	I	Fair	Mod

LE =Level of Evidence; QE = Quality of Evidence; \NB=Net benefit (see Appendix A)

B. Pain

BACKGROUND

There is a growing body of research that indicates clearly that PTSD and chronic pain frequently co-occur. People with both PTSD and chronic pain tend to have greater distress and impairment compared to those with only one of these conditions, and assessment and treatment are more complicated. It is therefore important to include a pain assessment (acute or chronic) in the examination of patient with ASD or PTSD, and to consider the extent to which PTSD symptoms may be influenced by pain or the extent to which pain symptoms are being exacerbated by PTSD.

Certain types of chronic pain are more common in individuals who have experienced specific traumas. Among adult survivors of physical, psychological, or sexual abuse the most common forms of chronic pain involve: pain in the pelvis, lower back, face, and bladder; fibromyalgia; interstitial cystitis; and non-remitting whiplash syndromes. Chronic pain is a common problem among returning soldiers. In service persons from OEF/OIF, head, neck, back, shoulder, and knee pain have been found to be most common (Lew, 2009). Co-morbidity of physical and emotional problems; and in particular the combination of chronic pain, PTSD symptoms, and post-concussion syndrome (PCS); is unique to the OEF/OIF population and appears to be more common in blast-injured patients and may be more difficult to treat than each condition independently.

Understanding the development and maintenance of chronic pain and PTSD and how they interact is of essential importance and is often overlooked in practice. Fear-based avoidance is a central theme in both PTSD and chronic pain. While the underlying basis for the avoidance may differ, avoidance behaviors may exacerbate or maintain the severity of either or both conditions. Although pharmacological agents have been examined in the treatment of pain and PTSD individually, little is known regarding the relationship of medication use with functioning in patients with co-morbid conditions. Pain should be assessed and aggressively treated in early phases of post-trauma, and providers across disciplines need to work together to develop treatments that are complementary, based on theory, and supported by empirical evidence.

RECOMMENDATIONS (BASED ON CONSENSUS OF THE WORKING GROUP CLINICAL EXPERTS)

1. Recommend pain assessment using a ‘0 to 10’ scale.
2. Obtain a thorough biopsychosocial history and assess for other medical and psychiatric problems, including risk assessment for suicidal and homicidal ideation and misuse of substances, such as drugs or alcohol and over-the-counter and prescription drugs or narcotics.
3. Assessment should include questions about the nature of the pain and likely etiology (i.e., musculoskeletal and neuropathic), locations, quality, quantity, triggers, intensity, and duration of the pain, as well as aggravating and relieving factors.
4. Assessment should include evaluation of the impact of pain on function and activities, pain-related disability, or interference with daily activities.
5. Assessment should include the identification of avoidance behaviors that contribute to emotional distress and/or impaired functioning.
6. Management of pain should be multidisciplinary, addressing the physical, social, psychological, and spiritual components of pain in an individualized treatment plan that is tailored to the type of pain. [C]

7. Selection of treatment options should balance the benefits of pain control with possible adverse effects (especially sedating medications) on the individual's ability to participate in, and benefit from, PTSD treatment. [I]
8. Musculoskeletal pain syndromes can respond to correcting the underlying condition and treatment with non-steroidal anti-inflammatory drugs (NSAIDs).
9. When appropriate, recommend use of non-pharmacological modalities for pain control, such as biofeedback, massage, imaging therapy, physical therapy, and complementary alternative modalities (yoga, meditation, acupuncture). [C]
10. Centrally acting medications should be used in caution in patients with PTSD, as they may cause confusion and deterioration of cognitive performance and interfere with the recovery process.
    a. If required, lower doses of opioid therapy or other centrally acting analgesics should be used for short duration with transition to the use of NSAIDs. [C]

5. Consider offering Cognitive Behavioral Therapy, which may include:
    b. Encouraging increasing activity by setting goals
    c. Correcting false and unrealistic beliefs/concerns about pain
    d. Teaching cognitive and behavioral coping skills (e.g., activity pacing)
    e. Practicing and consolidation of coping skills and reinforcement of use

DISCUSSION

Prevalence

PTSD and chronic pain disorder are highly co-morbid (Sharp, 2001). The literature indicates a high degree of co-occurrence between pain and PTSD, regardless of whether the pain is being assessed in patients with PTSD or PTSD is being assessed in patients with chronic pain. Chronic pain and PTSD are frequently observed to be co-morbid following traumatic injury (Bryant et al., 1999; Hickling and Blanchard, 1992). Studies have shown that PTSD symptoms tend to be elevated in patients with chronic pain and fibromyalgia (Amir, 1997; Engel, 2000; Sherman, 2000), chronic low back pain, and other musculoskeletal disorders (Sherman, 2000).

Sharp (2004) described four studies of patient populations that were drawn from MVA victims, combat veterans, fire fighters, and chronic pain clinic patients. In each instance, they found a high prevalence of pain in patients diagnosed with PTSD or a high prevalence of PTSD in patients diagnosed with chronic pain. Schwartz et al. (2006) noted that between 10 percent and 50 percent of patients treated in tertiary care settings for chronic pain and related conditions have symptoms that meet criteria for PTSD. Muse (1986) reported that 9.5 percent of a sample of patients attending a multidisciplinary chronic pain center met criteria for "post-traumatic pain syndrome."

Norman et al. (2007, 2008) found that self-reported pain levels within 24-48 hours after serious injury were significantly and strongly associated with the subsequent risk of PTSD. The author suggests that high levels of peri-traumatic pain could be used to identify individuals at elevated risk for PTSD following traumatic injury. Similarly, in a study of 2931 seriously injured patients admitted to acute care hospitals in the United States, Zatzick and Galea (2007) found that pain after injury was significantly associated with an increased risk of PTSD one year after hospitalization. The prevalence of PTSD is particularly high when the chronic pain results directly from a traumatic event (Hickling & Blanchard, 1992; Taylor & Koch, 1995; Chibnall, 1994; Asmundson et al., 1998; Otis

et al., 2003), and the presence of both PTSD and chronic pain can increase the symptom severity of either condition (Otis, 2003).

Beckham (1997) reported that 80 percent of combat Vietnam veterans with PSTD who completed self-report questionnaires assessing PTSD reported the presence of a chronic pain condition. Increased levels of PTSD re-experiencing symptoms were associated with increased pain level and pain-related disability.

The co-occurrence of chronic pain and PTSD has implications for the experience of both conditions. Persons with co-morbid pain and PTSD may experience less symptom improvement after treatment for these conditions (Asmundson, 2002; Baker et al., 1997; Clark et al., 2009; Hickling, 1992; McClean, 2005; Muse, 1986). Patients with chronic pain related to trauma or PTSD experience more intense pain and affective distress (Geisser, 1996), higher levels of life interference (Turk et al., 1996), and greater disability than pain patients without trauma or PTSD (Sherman, 2000).

Co-morbidity of Pain and PTSD (and PCS) in OEF/OIF

Because of the nature of injuries and the physical demands of OEF and OIF deployments, there are data to suggest that a significant majority of returned warriors report ongoing pain problems (Clark, 2004; Clark et al., 2009a; Gironda et al., 2006; Kalra et al., 2008).

Post-traumatic headaches are a common complaint (Gironda et al., 2009; Clark et al., 2007; Gironda et al., 2006; Lew et al., 2007; Ruff et al., 2008). Other commonly reported pain problems are low back pain and joint pain (Clark et al., 2007; Clark et al., 2009a). The high prevalence of chronic pain (pain that lasts longer than 3 months) places OEF/OIF soldiers at long-term risk for impaired functional ability, significant emotional distress, interpersonal conflict, substance misuse, and vocational limitations. Substance misuse (including opioid medications) has also been found in OEF/OIF returnees, although at lesser prevalence than pain (published rates range from 3 to 28 percent) (Clark et al., 2007; Kalra et al., 2008; Kang & Hyams, 2007; Shipherd et al., 2007).

Pain symptoms are a common complaint among post-deployment populations (back pain and headache). In one study of 1800 OEF/OIF veterans, 46.5 percent reported some pain, with 59 percent of those exceeding the VA clinical threshold of $\geq$ 4 on a 0-10 pain scale (Gironda et al., 2006). Recent literature suggests that many returning service members have multiple co-morbid symptoms of post-concussion syndrome, chronic pain, and PTSD (Clark et al., 2007; Clark et al., 2009; Lew et al., 2009; Sayer et al., 2008). In a sample of OEF/OIF veterans, pain was the single most common complaint recorded, and 42 percent of the sample reported concurrent PCS, chronic pain, and PTSD symptoms.

“The mechanism by which chronic pain and PTSD (and Post-Concussion Syndrome) interact is still unclear. Researchers evaluating co-morbid pain and PTSD have presented a variety of models to explain this phenomenon, including a Shared Vulnerability model, a Mutual Maintenance model, and a Triple Vulnerability model (Asmundson et al., 2002; Otis et al., 2003; Sharp & Harvey, 2001). These models propose mechanisms of interaction via the dispositional tendency to be fearful or anxious, the belief that anxiety states cause harmful consequences, and the cognitive distortions and behavioral patterns of PTSD and chronic pain that maintain or exacerbate symptoms of the other syndrome. These models have yet to be fully tested, and there are no available outcomes data regarding the success of integrated treatment of co-morbid pain and PTSD symptoms (Otis et al., 2003). However, such research is now being conducted." (Otis, 2008) (Walker, 2010).

Pain & PTSD Sensitivity Assessment

Given the high rates of co-morbidity of chronic pain and PTSD, clinicians should assess for both disorders. Several well-validated self-report questionnaires are available to help determine a diagnosis and the severity of symptoms. Self-report measures of pain, including the 0 to 10 numerical pain rating scale, the McGill Pain Questionnaire, the West Haven-Yale Multidimensional Pain Inventory, or the Pain Outcomes Questionnaire, were developed and validated specifically for veterans.

Asmundson et al. (2002) recommended that clinicians who conduct diagnostic assessments of patients presenting with PTSD symptoms also screen for the presence of existing pain conditions, such as fibromyalgia or chronic musculoskeletal pain using a self-reported questions or a structured clinical interview format

Interdisciplinary Approach to Management

Only a few studies have reported the results of treatments designed to address co-occurring chronic pain and PTSD. Given the current state of the literature, few recommendations can be made regarding preferred treatment modalities for individuals with co-morbid pain and PTSD. Several authors support the use of a multidisciplinary treatment approach for patients with PTSD and chronic pain (Muse, 1986).

Given the broad range of emotional and physical symptoms characteristic of veterans with co-occurring PTSD, chronic pain, and possible PCS, an integrated treatment approach is required (Walker, 2010). Treatment goals need to be clarified (e.g., reduce symptom severity, increase occupational or interpersonal functioning, reduce ongoing use of healthcare services) (Clark, 2008).

The focus of the integrated approach should be on education and management of symptoms and reducing pain and suffering, improving function, and enhancing quality of life. The interventions and treatment modalities employed should follow the current evidence-based recommendations for PCS, chronic pain, and PTSD (see VA/DoD guidelines for mTBI/Concussion and the VHA National Pain Management Strategy [NPMS], 2003); the specific practice guidelines for managing acute and chronic pain associated with certain conditions, like low back pain (APS-AAPM, 2005; VHA/DoD, 2007); and the guideline for the use of opioids with chronic pain (VHA/DoD, 2010).

For the OEF/OIF population of returning soldiers, treatment should be individualized on an inpatient or outpatient basis, depending upon needs within the group and individual treatment formats. Treatment should be goal-oriented and time-limited, with increased patient function and independence as major goals (Clark, 2009).

Non-Pharmacological Treatment

Initial treatment of PTSD focuses on providing psychoeducation about the disorder. This may include specifically addressing how fears and avoidance of the trauma may serve to maintain the symptoms and decrease the ability to function. This may also include discussing how pain may serve as a trigger or reminder of the trauma and increase arousal, fear, and avoidance and thereby increase disability and pain (Sharp, 2004).

Non-pharmacological ways to manage chronic pain may include **Relaxation (**e.g., relax the locus of the pain problems by relaxing muscle tension). **Increasing Activity and Fitness (**e.g., gradual return to more normal levels of activities and slowly increase patient's stamina for physical activities), **Reducing Emotional Over-Reactivity (**e.g., practice specific methods of emotional reaction to stressful triggers); and **External Focusing/Distracting** (e.g., learn to shift and manipulate the focus of attention in a positive way, which will minimize the experience of the pain).

Complementary Alternative medicine - There are numerous interventions that are being used to help manage chronic pain, including breathing, muscle relaxation, visual imagery, music, cold/heat, stretching, massage therapy, stress management, acupuncture, acupressure, hydrotherapy, and others.

Tan et al. (2007) examined various CAM therapies for chronic pain. For example, heart rate variability (HRV) biofeedback (using a stress eraser portable biofeedback device that easily can be used by veterans at home for the purpose of increasing HRV) has been shown to be effective for reducing the symptoms of PTSD (e.g., Tan et al., 2009; Zucker, 2009) and for persistent pain associated with fibromyalgia (Hassett et al., 2007). Regulating heart rhythm coherence, using biofeedback devices that computes the heart rhythm patterns, has been shown to improve symptoms, such as depression, anxiety, panic disorder, and PTSD symptoms (McCraty, Atkinson, Tomasino, & Stuppy, 2001).

Pharmacotherapy

There are no studies evaluating the pharmacotherapy for acute dissociation or traumatic pain associated with ASR.

The most common first-line treatments for pain have traditionally been analgesics, which include opioids, NSAIDS (non-steroidal anti-inflammatory drugs), anti-epileptic drugs, and tricyclic antidepressants for neuropathic pain, and antidepressants that target the inhibition of norepinephrine reuptake (SNRIs). With respect to trauma exposure, some data suggest that pain patients with co-morbid PTSD use analgesic medications at higher rates than their non-PTSD counterparts (Schwartz et al., 2006).

Selective serotonin reuptake inhibitors (SSRIs) are recommended as the first-line pharmacological intervention for PTSD (see Intervention for PTSD– Module B). SSRIs also have been examined for use in the treatment of chronic pain, but support for their efficacy in this population is limited (see reviews by McCleane, 2008; Dworkin et al., 2007). Sedative and anxiolytic medications are sometimes prescribed to alleviate symptoms associated with both PTSD and chronic pain but are not recommended due to the addictive properties of many anxiolytic agents (American Psychiatric Association, 2004; Sanders et al., 2005). The relationship between these pharmacological agents and functioning among patients with co-morbid pain and PTSD has not been examined.

Opioid Therapy

While controversial, the use of opioid medications in the treatment of chronic, non-malignant pain has increased significantly over the past three decades (Caudill-Slosberg et al., 2004). The efficacy of opioids in alleviating acute pain is well established, but less is known regarding their utility in treating chronic pain or their relationship with patient functioning over extended periods of use (Ballantyne and Shin, 2008). Side effect profiles associated with opioid use – tolerance, physical dependence, cognitive impairment – often are cited as factors contributing to potential decreases in functioning (Ballantyne and Shin, 2008; Eriksen et al., 2006). Clinicians need to recognize the interrelationships of chronic pain, PTSD, and opioid use. The co-morbid psychiatric disorders are known to increase risk of abuse and dependency among persons with chronic pain (Edlund et al., 2007). Some data suggest that pain patients with co-morbid PTSD use analgesic medications at higher rates than their non-PTSD counterparts (e.g., Schwartz et al., 2006). It may suggest that the experience of pain in the present may be affected by previous emotional trauma and ongoing trauma-related stress disorders. Some findings suggest the possibility that long-term use of opioids may lead to opioid-induced hyperalgesia (Angst & Clark, 2006).

Given inconsistent findings regarding the efficacy of opioids for long-term pain control, potential for reductions in overall functioning, and the increased risk of abuse and

dependency, providers should consider the benefits and potential harm of extended opioid therapy for patients with chronic pain subsequent to traumatic injury (Clapp, 2010) (VA/DoD COT CPG, 2010).

Cognitive Behavior Therapy (CBT)

Cognitive behavioral therapy is recommended as first-line therapy for PTSD (see Intervention for PTSD Module B). CBT for pain uses a similar approach and a variety of techniques that are aimed at changing maladaptive thoughts and behaviors that serve to maintain and exacerbate the experience of pain. CBT for chronic pain involves teaching patients ways of safely reintroducing enjoyable activities into their lives.

Using components of cognitive processing therapy (CPT) for PTSD and cognitive behavioral therapy (CBT) for chronic pain management, a 12-session integrated treatment for veterans with co-morbid chronic pain and PTSD was developed (Otis, 2009). The key components of the CBT for chronic pain include cognitive restructuring (i.e., teaching patients how to recognize and change maladaptive thoughts), relaxation training (e.g., diaphragmatic breathing, progressive muscle relaxation), time-based activity pacing (i.e., teaching patients how to become more active without overdoing it), and graded homework assignments designed to decrease patients' avoidance of activity and reintroduce a healthy, more active lifestyle (Otis, 2007). The therapy includes weekly readings and homework assignments, pre- and post-treatment evaluations using measures of pain, PTSD, physical disability, and psychological distress.

The result of implementing the program in a pilot study demonstrates the importance of establishing participant trust and regular therapy attendance and addressing participant avoidance. Participants reported that they generally liked the format of treatment and appreciated learning about the ways that chronic pain and PTSD share some common symptoms and ways that the two disorders can interact with one another. The authors concluded, based on this initial small pilot study, that the participants appeared to benefit from receiving the integrated treatment for pain and PTSD (Otis, 2009).

C. Irritability, Severe Agitation, or Anger

BACKGROUND

In the most general sense, anger is a feeling or emotion that ranges from mild irritation to intense fury and rage. Anger is often a central feature of response to trauma and can be seen as a core component of the survival response in humans. Mismanaged or uncontrolled anger and rage can lead to a continued sense of being out of control and may cause conflicts in personal and professional relationships. Anger and irritability may be associated with domestic violence and abuse, road rage, and workplace violence, even if there is no intent to cause harm to others. It is important to distinguish between anger and aggression. Aggression is behavior that is intended to cause harm to another person or damage property. This behavior can include verbal abuse, threats, or violent acts. Anger, on the other hand, is an emotion and does not necessarily lead to aggression. Therefore, a person can become angry without acting aggressively.

Anger becomes a problem when it is felt too intensely, is felt too frequently, or is expressed inappropriately. Anger management interventions include a range of methods, including teaching individuals to recognize signs of becoming angry, self-calm, avoid escalating conflicts, and respond to anger-eliciting situations in more positive ways.

RECOMMENDATIONS (BASED ON CONSENSUS OF THE WORKING GROUP CLINICAL EXPERTS)

1. Assess the nature of symptoms, severity, and dangerousness. Consider using standardized Anger Scales, such as Spielberger's State-Trait Anger Expression Inventory, to quantify.
2. Explore for cause of symptoms and follow-up to monitor change.
3. Consider referral to specialty care for counseling or for marital or family counseling as indicated. Offer referral for:
    a. Anger Management therapy
    b. Training in exercise and relaxation techniques
4. Promote participation in enjoyable activities - especially with family/ loved ones.
5. Promote sleep and relaxation.
6. Avoid stimulants and other substances (caffeine, alcohol).
7. Address pain (see pain management).
8. Avoid benzodiazepines.
9. Consider SSRIs/SNRIs
    a. If not responding to SSRIs/SNRIs and other non-pharmacological interventions, consider low-dose anti-adrenergics or low-dose atypical antipsychotics (risperidone, quetiapine).
    b. If not responding or worsening, refer to specialty care.

DISCUSSION

In anger management treatments, physical arousal, problem behaviors, and anger-provoking thoughts/beliefs are all addressed in different ways (Chemtob, 1997).

Cognitive-behavioral treatment, such as anxiety management, shows positive results when used to address anger and applies many techniques to manage these three anger components.

DISCUSSION

Prevalence

A study of sample OEF/OIF veterans found that over half of the veterans with PTSD indicated that they had been aggressive in the past 4 months, such as threatening physical violence, destroying property, and having a physical fight with someone. Veterans with sub-threshold PTSD syndrome reported just about the same amount of aggressive behavior as the veterans with PTSD. In fact, anger has been shown to be associated with other co-morbid conditions to PTSD, such as head injury and alcohol (substance) abuse. Each of these conditions has been associated with elevated anger and hostility in veterans from previous conflicts. High levels of anger have been observed in veterans of the Iraq and Afghanistan Wars. (Jakupcak, 2007)

In another survey of 2797 US soldiers returning from deployment, overall, 40 percent of soldiers reported killing or being responsible for killing during their deployment. Even after controlling for combat exposure, killing was a significant predictor of PTSD symptoms, alcohol abuse, anger, and relationship problems Maquan et al., 2010).

A study assessing Vietnam combat veterans and comparing them to veterans who did not serve in war found that the combat veterans were not significantly angrier than their veteran peers who did not serve in Southeast Asia. Additionally, various parameters of

war zone duty were not highly associated with anger scores. However, combat veterans with PTSD scored significantly higher than veterans without PTSD on measures of anger, arousal, and range of anger-eliciting situations, hostile attitudinal outlook, and tendency to hold anger in. These results suggest that PTSD, rather than war zone duty, is associated with various dimensions of angry affect (McFalls et al., 1999).

Anger can be a very difficult emotion to deal with and can lead to a number of legal and interpersonal problems, such as domestic violence. In fact, individuals with PTSD are particularly at risk for the perpetration of relationship violence.

Research has identified anger as prominent in and an influence on treatment outcomes for military veterans with PTSD. To improve treatment effectiveness, clinicians need to assess veterans' anger, aggression, and alcohol use, as well as their current fear of anger and elucidate the relationship between these factors (Forbes, 2008).

Chemtob et al. (1997) described three components of post-traumatic anger that can become maladaptive or interfere with one's ability to adapt to current situations that do not involve extreme threat:

- **Arousal**: Anger is marked by the increased activation of the cardiovascular, glandular, and brain systems associated with emotion and survival. It is also marked by increased muscle tension. Sometimes with individuals who have PTSD, this increased internal activation can become reset as the normal level of arousal and can intensify the actual emotional and physical experience of anger. This can cause a person to feel frequently on edge, keyed up, or irritable and can cause a person to be more easily provoked. It is common for traumatized individuals to actually seek out situations that require them to stay alert and ward off potential danger. Conversely, they may use alcohol and drugs to reduce overall internal tension.
- **Behavior**: Often, the most effective way of dealing with extreme threat is to act aggressively, in a self-protective way. Additionally, many people who were traumatized at a relatively young age do not learn different ways of handling threat and tend to become caught in their ways of reacting when they feel threatened. This is especially true of people who tend to be impulsive (who act before they think). Again, as stated above, while these strategies for dealing with threat can be adaptive in certain circumstances, individuals with PTSD can become stuck in using only one strategy, when other approaches would be more constructive. Behavioral aggression may take many forms, including aggression toward others, passive-aggressive behavior (e.g., complaining, "backstabbing," deliberately being late or doing a poor job), or self-aggression (self-destructive activities, self-blame, being chronically hard on oneself, self-injury).
- **Thoughts and Beliefs**: The thoughts or beliefs that people have to help them understand and make sense of their environment can over-exaggerate threat. Often, the individual is not fully aware of these thoughts and beliefs, but they cause the person to perceive more hostility, danger, or threat than others might feel is necessary. For example, a combat veteran may become angry when others around him (wife, children, and coworkers) don't "follow the rules." The strength of his belief is actually related to how important it was for him to follow rules during the war in order to prevent deaths. Often, traumatized persons are not aware of the ways their beliefs are related to past trauma. For instance, by acting inflexibly toward others because of their need to control their environment, they can provoke others into becoming hostile, which creates a self-fulfilling prophecy. Common thoughts that people with PTSD have include: "You can't trust anyone," "If I got out of control, it would be horrible/life-threatening/intolerable," "After all

I've been through, I deserve to be treated better than this," and "Others are out to get me, or won't protect me, in some way."

How can individuals with post-traumatic anger get help?

In anger management treatment, arousal, behavior, and thoughts/beliefs are all addressed in different ways. Cognitive-behavioral treatment, a commonly utilized therapy that shows positive results when used to address anger, applies many techniques to manage these three anger components:

- For **increased arousal**, the goal of treatment is to help the person learn skills that will reduce overall arousal. Such skills include relaxation, self-hypnosis, and physical exercises that discharge tension.

- For **behavior**, the goal of treatment is to review a person's most frequent ways of behaving under perceived threat or stress and help him or her to expand the possible responses. More adaptive responses include taking a time-out; writing thoughts down when angry; communicating in more verbal, assertive ways; and changing the pattern "act first, think later" to "think first, act later."

- For **thoughts/beliefs**, individuals are given assistance in logging, monitoring, and becoming more aware of their own thoughts prior to becoming angry. They are additionally given alternative, more positive replacement thoughts for their negative ideas (e.g., "Even if I am out of control, I won't be threatened in this situation." or "Others do not have to be perfect in order for me to survive/be comfortable."). Individuals often role-play situations in therapy so they can practice recognizing their anger-arousing thoughts and apply more positive thoughts.

There are many strategies for helping individuals with PTSD deal with the frequent increase of anger they are likely to experience. Most individuals have a combination of the three anger components listed above, and treatment aims to help with all aspects of anger. One important goal of treatment is to improve a person's sense of flexibility and control so that he or she does not feel re-traumatized by his or her own explosive or excessive responses to anger triggers. Treatment is also meant to have a positive impact on personal and work relationships.

APPENDICES

Appendix A. Guideline Development Process 199

Appendix B. Acronym List 206

Appendix C. PTSD Screening Tools 209

Appendix D. Participant List 213

Appendix E. Bibliography 222

APPENDIX A: Guideline Development Process

The update of the VA/DoD Clinical Practice Guideline for Management of Post-Traumatic Stress was developed following the steps described in "Guideline for Guidelines," an internal working document of the VA/DoD Evidence Based Practice Working Group, that requires an ongoing review of guideline works in progress.

The Offices of Quality Performance and Patient Care Services of the VA, and the Army Medical Command of the DoD identified clinical leaders to champion the guideline development process. During a preplanning conference call, the clinical leaders defined the scope of the guideline and identified a group of clinical experts from the VA and DoD to form the Management of Post-Traumatic Stress Working Group (WG). For this guideline these WG participants were drawn from the fields of primary care, psychiatry, psychology, internal medicine, pharmacology, nursing, and social work.

The WG participated in 2 face-to-face meetings to reach consensus about the guideline algorithm and evidence-based recommendations and to prepare a draft update document. The draft continued to be revised by the Working Group through numerous conference calls and individual contributions to the document.

Recommendations for the management of post-traumatic stress were derived through a rigorous methodological approach that included the following:

- Determining appropriate criteria such as effectiveness, efficacy, population benefit, or patient satisfaction
- Reviewing literature to determine the strength of the evidence in relation to these criteria
- Formulating the recommendations and grading the level of evidence supporting the recommendation

After orientation to the goals and scope of the guideline update, the WG developed a set of 13 researchable questions within the focus areas of the guideline and identified associated key terms. For this guideline, two sets of questions were developed. The First (A) addressed *acute and early intervention aimed at/prevention of PTSD in adults with recent exposure to trauma*. The second set (B) focused on *therapy of adult patients with PTSD to achieve resolution of symptoms and functional outcome.* This approach ensured that the guideline development work outside of meetings focused on issues that practitioners considered important and also produced criteria for the literature search and selection of included studies that formed the body of evidence for this guideline update.

All questions specified (adapted from the Evidence-Based Medicine toolbox, Center for Evidence-Based Medicine, [http://www.cebm net]):

- **P**opulation – Characteristics of the target patient population
- **I**ntervention – Exposure, diagnostic, or prognosis
- **C**omparison – Intervention, exposure, or control used for comparison
- **O**utcome – Outcomes of interest

These specifications served as the preliminary criteria for selecting studies. See *PICO Questions to Guide Literature Search* for a complete listing and categorization of the questions *(end of this appendix).*

Literature Search

An initial global literature search yielded 59 systematic reviews/meta-analyses addressing pharmacotherapy, psychotherapy, combination, enhancement, complementary and other topics. One hundred and seventy eight (178) RCTs were found on the same subjects. Twenty-four controlled trials (CT) addressed combination, enhancement, and other areas. Refinement of the review process with input from the WG members resulted in the studies being identified that met the baseline criteria for inclusion,

addressed one or more of the researchable questions, and covered topic areas that had either not been addressed in the previous version of this guideline or had been included but not fully developed . A more detailed (full) search was conducted on each question, supplemented by hand searches and cross-referencing to search for relevant articles. The searches for these questions covered the period since the publication of the first VA/DoD CPG on management of post-traumatic stress (between January 1, 2002 and August, 2009).

Selection of Evidence

The evidence selection process was designed to identify the best available evidence to address each key question and ensure maximum coverage of studies at the top of the hierarchy of study types. Published, peer-reviewed RCTs, as well as meta-analyses and systematic reviews that included randomized controlled studies, were considered to constitute the strongest level of evidence in support of guideline recommendations. This decision was based on the judgment that RCTs provide the clearest, most scientifically sound basis for judging comparative efficacy. The WG also recognized the limitations of RCTs, particularly considerations of generalizability with respect to patient selection and treatment quality. When available, the search sought out critical appraisals already performed by others that described explicit criteria for deciding what evidence was selected and how it was determined to be valid. The sources that have already undergone rigorous critical appraisal include Cochrane Reviews, Best Evidence, Technology Assessment, AHRQ systematic evidence reports, and other published Evidence-based Clinical Practice Guidelines.

The following databases were searched: Medline/Pubmed, Embase, PsycINFO, OVID, PILOT, and Cochrane Central Register of Controlled Trials. Limits were set for language (English), and type of research (RCT, systematic reviews including EPC and HTA reviews and meta-analyses). For prognostic and diagnostic questions (e.g., does test improve outcome?); cohort or other prospective non-RCT designs were considered.

The following inclusion criteria were used to select the articles identified in the literature search for possible inclusion:

- Published in United States, United Kingdom, Europe, Australia, Japan, New Zealand
- Full articles only published in English
- Study populations: age limited to adults 18 years of age or older; all races, ethnicities, and cultural groups
- Relevant outcomes able to be abstracted from the data presented in the articles
- Sample sizes appropriate for the study question addressed in the paper. RCTs were included if they were initiated with 30 or more participants

Preparation of Evidence Tables (Reports) and Evidence Rating

The results of the searches were organized in evidence reports, and copies of the original studies were provided to the WG for further analysis. Each reference was appraised for scientific merit, clinical relevance, and applicability to the populations served by the VA and DoD health care systems.

Recommendation and Quality Rating

Evidence-based practice involves integrating clinical expertise with the best available clinical evidence derived from systematic research.

A group of research analysts read and coded each article that met inclusion criteria. The articles were assessed for methodological rigor and clinical importance. Clinical experts from the VA and DoD WG reviewed the results and evaluated the strength of the evidence, considering quality of the body of evidence (made up of the individual studies) and the significance of the net benefit (potential benefit minus possible harm) for each intervention.

The overall strength of each body of evidence that addresses a particular Key Question was assessed using methods adapted from the U.S. Preventive Services Task Force (Harris, 2001). To assign an overall quality [QE] (see Table A-2) of the evidence (good, fair, or poor), the number, quality, and size of the studies; consistency of results between studies; and directness of the evidence were considered. Consistent results from a number of higher-quality studies [LE] (see Table A-1) across a broad range of populations; supports with a high degree of certainty that the results of the studies are true and therefore the entire body of evidence would be considered ‘‘good” quality. A ‘‘fair” quality was assigned to the body of evidence

indicating that the results could be due to true effects or to biases present across some or all of the studies. For a ‘‘poor” quality body of evidence, any conclusion is uncertain due to serious methodological shortcomings, sparse data, or inconsistent results.

The Strength of Recommendation [SR] was then determined based on the Quality of the Evidence [QE], and the clinical significance of the net benefit [NB] (see Table A-3) for each intervention, as demonstrated by the body of evidence. Thus, the grade (i.e., A, B, C, D or I) assigned to guideline recommendations reflect both variables; the Quality of the evidence and the potential clinical benefit that the intervention may provide to patients (see Table A4).

Table A-1: Level of Evidence (LE)	
I	At least one properly done RCT
II-1	Well-designed controlled trial without randomization
II-2	Well-designed cohort or case-control analytic study, preferably from more than one source
II-3	Multiple time series evidence with/without intervention, dramatic results of uncontrolled experiment
III	Opinion of respected authorities, descriptive studies, case reports, and expert committees

Table A-2: Overall Quality [QE]	
Good	High grade evidence (I or II-1) directly linked to health outcome
Fair	High grade evidence (I or II-1) linked to intermediate outcome; *or* Moderate grade evidence (II-2 or II-3) directly linked to health outcome
Poor	Level III evidence or no linkage of evidence to health outcome

Table A-3: Net Effect of the Intervention [NB]	
Substantial	More than a small relative impact on a frequent condition with a substantial burden of suffering; *or* A large impact on an infrequent condition with a significant impact on the individual patient level.
Moderate	A small relative impact on a frequent condition with a substantial burden of suffering; *or* A moderate impact on an infrequent condition with a significant impact on the individual patient level.
Small	A negligible relative impact on a frequent condition with a substantial burden of suffering; *or* A small impact on an infrequent condition with a significant impact at the individual patient level.
Zero or Negative	Negative impact on patients; *or* No relative impact on either a frequent condition with a substantial burden of suffering, or an infrequent condition with a significant impact on the individual patient level.

Table A-4: Final Grade of Recommendation [SR]				
	The net benefit of the intervention			
Quality of Evidence	Substantial	Moderate	Small	Zero or Negative
Good	A	B	C	D
Fair	B	B	C	D
Poor	I	I	I	I

Strength of Recommendation Rating [SR]

A	A strong recommendation that the clinicians provide the intervention to eligible patients. *Good evidence was found that the intervention improves important health outcomes and concludes that benefits substantially outweigh harm.*
B	A recommendation that clinicians provide (the service) to eligible patients. *At least fair evidence was found that the intervention improves health outcomes and concludes that benefits outweigh harm.*
C	No recommendation for or against the routine provision of the intervention is made. *At least fair evidence was found that the intervention can improve health outcomes, but concludes that the balance of benefits and harms is too close to justify a general recommendation.*
D	Recommendation is made against routinely providing the intervention to asymptomatic patients. *At least fair evidence was found that the intervention is ineffective or that harms outweigh benefits.*
I	The conclusion is that the evidence is insufficient to recommend for or against routinely providing the intervention. *Evidence that the intervention is effective is lacking, of poor quality, or conflicting, and the balance of benefits and harms cannot be determined.*

Algorithm Format

The clinical algorithm incorporates the information presented in the guideline in a format which maximally facilitates clinical decision-making. The use of the algorithmic format was chosen because of evidence showing that such a format improves data collection, facilitates diagnostic and therapeutic decision-making, and changes patterns of resource use.

The algorithmic format allows the provider to follow a linear approach to critical information needed at the major decision points in the clinical process and includes:

- An ordered sequence of steps of care
- Recommended observations
- Decisions to be considered
- Actions to be taken

A clinical algorithm diagrams a guideline into a step-by-step decision tree. Standardized symbols are used to display each step in the algorithm (Society for Medical Decision-Making Committee, 1992). Arrows connect the numbered boxes indicating the order in which the steps should be followed.

	Rounded rectangles represent a clinical state or condition.
	Hexagons represent a decision point in the guideline, formulated as a question that can be answered Yes or No. A horizontal arrow points to the next step if the answer is YES. A vertical arrow continues to the next step for a negative answer.
	Rectangles represent an action in the process of care.
	Ovals represent a link to another section within the guideline.

A letter within a box of an algorithm refers the reader to the corresponding annotation. The annotations elaborate on the recommendations and statements that are found within each box of the algorithm. Included in the annotations are brief discussions that provide the underlying rationale and specific evidence

tables. Annotations indicate whether each recommendation is based on scientific data or expert opinion. A complete bibliography is included in the guideline.

Lack of Evidence – Consensus of Experts

Where existing literature was ambiguous or conflicting, or where scientific data was lacking on an issue, recommendations were based on the clinical experience of the Working Group.
This update of the Stroke Rehabilitation Guideline is the product of many months of diligent effort and consensus building among knowledgeable individuals from the VA, DoD, and academia, as well as guideline facilitators from the private sector. An experienced moderator facilitated the multidisciplinary Working Group. The list of participants is included in Appendix D

REFERENCES

Agency for Health Care Policy and Research (AHCPR). Manual for conducting systematic review. August 1996. Prepared by Steven H. Woolf.

Harris RP, Helfand M, Woolf SH, Lohr KN, Mulrow CD, Teutsch SM, Atkins D; Methods Work Group, Third US Preventive Services Task Force, Current methods of the U.S. Preventive Services Task Force: a review of the process. Am J Prev Med 2001 Apr;20(3 Suppl):21-35. Available at; http://www.ahrq.gov/clinic/ajpmsuppl/harris1.htm

Society for Medical Decision-Making Committee (SMDMC). Proposal for clinical algorithm standards, SMDMC on Standardization of Clinical Algorithms. Med Decis Making 1992 Apr-Jun; 12(2):149-54.

United States Preventive Service Task Force (USPSTF). Guide to clinical preventive services. 2nd edition. Washington, DC: US Department of Health and Human Services, Office of Disease Prevention and Health Promotion, 1996.

Woolf SH. Practice guidelines, a new reality in medicine II; Methods of developing guidelines. Arch Intern Med 1992 May; 152(5):946-52.

Management of Post-Traumatic Stress
UPDATE 2010 PICO QUESTIONS

A. Acute Intervention / Prevention in adults with recent exposure to trauma or diagnosed with ASD

1. Is **debriefing** more effective than no intervention or any other intervention for prevention of PTS disorder?
2. Is **pharmacotherapy** more effective than no intervention or any other intervention for prevention of full PTS disorder?
    a. **alpha–blockers**
    b. **beta–blockers**
    c. Sympatolitic
    d. **DCS and CBT**
3. Are any **psychotherapy techniques** more effective than no intervention or any other intervention for prevention of full PTS disorder?
4. Is **psychoeducation** more effective than no intervention or any other intervention for prevention of full PTS disorder?
5. Are any **Complimentary Alternative Medicine (CAM) approaches** more effective than no intervention or any other intervention for prevention of full PTS disorder?
6. Is **early intervention** more effective than **later intervention** for prevention of full PTS disorder**?**
7. Is **combination** of pharmacotherapy and psychotherapy more effective than no intervention or any other intervention for prevention of full PTS disorder?
8. **Is peer counseling** more effective than **counseling** by an outside team for prevention of full PTS disorder?
9. **Is outreach (screening, repeated screening)** more effective than no intervention or any other intervention for prevention of full PTS disorder?

B. Treatment for PTSD

Which of the following treatment interventions for adult patients with PTSD lead to achieve Resolution of symptoms and Functional outcome? (Consider effectiveness in special population (e.g., Gender, Combat veterans, Elderly)

10. Psychotherapy Techniques:

Is **prolonged exposure** more effective interventions in the treatment of PTSD**?**
Is EMDR more effective than other interventions in the treatment of PTSD?
Is cognitive processing therapy more effective than other interventions in the treatment of PTSD?
Is DBT, MBCT, ACT or mindfulness more effective than other interventions in the treatment of PTSD?
Psychoeducation (Battlemind, stress control) more effective than other interventions in the treatment of PTSD?

11. Pharmacotherapy Classes:

-MAOI and TCAs
-SSRIs
-SNRIs
-DNRI
-Novel antidepressant (trazodone, nefazodone)
-Conventional antipsychotics
-Atypical antipsychotics
-Anticonvulsants
-Anxiolytic (Benzodiazepine)
-Sedative hypnotics (for sleep)
-Antiadrenergics

12. Somatic:

-ECT
-rTMS

13. Complementary Alternative Mdedicine (CAM)

-Acupuncture
-Meditation
-Herbal, food suppl.,
-Yoga, Tai Chi

APPENDIX B
Acronym List

ABCs	Airway, breathing, circulation
AHCPR	Agency for Healthcare Policy and Research
APA	American Psychiatric Association
ASD	Acute stress disorder
ASR	Acute stress reaction
AUDIT	Alcohol Use Disorders Identification Test
BEP	Brief Eclectic Psychotherapy
BFT	Behavioral Family Therapy
BL	baseline;
CAGE	Alcohol abuse/dependence screening test mnemonic
CAPS	Clinician-Administered PTSD Scale;
CAPS	Clinician Administered PTSD Scale
CAPS-1	Clinician-Administered PTSD Scale1-month version;
CAPS-2	Clinician-Administered PTSD Scale1-week version;
CAPS-2	Clinician-Administered PTSD Scale Part 2;
CAPS-D	Clinician-Administered PTSD Scale hyperarousal subscale;
CBC	Complete blood count
CBT	Cognitive Behavioral Therapy
CCTR	Cochrane Central Register of Controlled Trials
CDR	Commander
CGI	Clinical Global Impression;
CGI-I	Clinical Global Impression-Improvement;
CGI-S	Clinical Global Assessment of Severity;
CGIC	Clinical Global Impression of Change;
CI	confidence interval;
CISD	Critical Incident Stress Debriefing
CNS	Central nervous system
COSR	Combat and operational stress reactions
CPT	Cognitive Processing Therapy
CPT-C	CPT-Cognitive
CT	Cognitive Therapy
CTT	Cognitive Trauma Therapy
CV	Cardiovascular
DARE	Database of Abstracts of Reviews of Effectiveness
DAST	Drug Abuse/Dependence Screener
DBT	Dialectical Behavioral Therapy
DCS	D-cycloserine;
DoD	Department of Defense
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders (4th ed.)
DTS	Davidson Trauma Scale;
dx	diagnosis
EBM	Evidence-based medicine
EBPTU	Evaluation and Brief PTSD Treatment Unit
ED	Emergency Department;
EEG	Electroencephalography
EKG	Electrocardiogram

EMDR	Eye Movement Desensitization and Reprocessing
EMTs	Emergency Medical Teams
ES	effect size
ESRT	Emotional Self-Regulation Therapy
ET	Exposure Therapy
EtoH	Ethanol
FDA	U. S. Food and Drug Administration
GAF	Global Assessment of Function
GI	Gastrointestinal
grp(s)	group(s);
GT	group therapy
GU	Genitourinary
HIV	Human immunodeficiency virus
Interapy	Internet Therapy
IRT	Imagery Rehearsal Therapy
IRT	Image Rehearsal Therapy
ITT	Intention to Treat
LOC	Level of consciousness
LOF	Level of function
MAOIs	Monoamine oxidase inhibitors
MAST	Michigan Alcohol Screening Test
MDD	Major Depressive Disorder
MHP	Mental health providers
MI	Myocardial infarction
MISS	Mississippi Scale for Combat-Related PTST-civilian version;
MMSE	Mini-Mental State Examination
MRI	Magnetic resonance imaging
MSE	Mental status examination
MVA	motor vehicle accident;
N/R	not reported;
NET	Narrative Exposure Therapy (a form of Exposure Therapy)
NIMH	National Institute of Mental Health
NS	Nervous system
OMO	Ongoing military operations
OTC	Over-the-counter
PCL	Posttraumatic Stress Disorder Checklist;
PCL-C	PTSD Checklist – Civilian Version
PCL-M	Patient Checklist for PTSD-Military Version;
PCL-M	PTSD Checklist – Military Version
PCL-S	PTSD Checklist – Stressor Specific Version
PCP	Primary care provider
PE	Physical examination
PE (Interventions)	Prolonged Exposure
PIES	Proximity, Immediacy, Expectancy, Simplicity
PSQI	Pittsburgh Sleep Quality Index
PsychEd	Psychological Education
pt(s)	patient(s);
PTSD	posttraumatic stress disorder;
PTSD	Post-traumatic Stress Disorder

QE	Quality of evidence
RA	Repeated Assessment
RCS	Readjustment Counseling Services
RCT	Randomized controlled trial
RLX	Relaxation Training
RTD	Return-to-duty
SC	Supportive Counseling
SC	Supportive Counseling
SIADH	Syndrome of inappropriate antidiuretic hormone
SIPU	Specialized Inpatient PTSD Unit
SIT	Stress Inoculation Therapy
SM	Service member
SR	Strength of recommendation
SSRI	Selective Serotonin Reuptake Inhibitors
SUD	Substance Use Disorder
SUNY	State University of New York
TAU	Treatment as Usual
TCAs	Tricyclic Antidepressants
TOP-8	Treatment Outcome PTSD rating scale;
TSH	Thyroid Stimulating Hormone
Tx or RX	treatment
USPSTF	U.S. Preventive Service Task Force
VA	Veterans Affairs
VAMC	Veterans Affairs Medical Center
Vets	Veternas
VHA	Veterans Health Administration
WL	wait list

APPENDIX C
PTSD Screening Tools

Primary Care PTSD Screen (PC-PTSD)

The table below shows the Primary Care PTSD Screen (PC-PTSD) that has been designed for use in primary care and other medical settings. The PC-PTSD is brief and problem-focused. The screen does *not* include a list of potentially traumatic events. There are two reasons for this:

- Studies on trauma and health in both male and female patients suggest that the active mechanism linking trauma and physical health is the diagnosis of PTSD. In other words, the relationship between trauma and health appears to be mediated through a current PTSD diagnosis.
- A symptom-driven screen, rather than a trauma-focused screen, is attractive to primary care staff who may not be able to address a patient's entire trauma history during their visit with the patient. Such a trauma inquiry might be especially problematic with a VA population where the average number of traumatic events meeting criterion A for PTSD is over four.

A positive response to the screen does not necessarily indicate that a patient has Post-traumatic Stress Disorder. However, a positive response does indicate that a patient *may* have PTSD or trauma-related problems and further investigation of trauma symptoms by a mental-health professional may be warranted.

Primary Care PTSD Screen **In your life, have you ever had any experience that was so frightening, horrible, or upsetting that, *in the past month,* you…**
1. Have had nightmares about it or thought about it when you did not want to? **YES** **NO**
2. Tried hard not to think about it or went out of your way to avoid situations that reminded you of it? **YES** **NO**
3. Were constantly on guard, watchful, or easily startled? **YES** **NO**
4. Felt numb or detached from others, activities, or your surroundings? **YES** **NO**
Current research suggests that the results of the PC-PTSD should be considered "positive" if a patient answers "yes" to any two items.

PTSD CheckList – Civilian Version (PCL-C)

Patient's Name: _____

Instruction to patient: Below is a list of problems and complaints that veterans sometimes have in response to stressful life experiences. Please read each one carefully, put an "X" in the box to indicate how much you have been bothered by that problem *in the last month.*

No.	Response:	Not at all (1)	A little bit (2)	Moderately (3)	Quite a bit (4)	Extremely (5)
1.	Repeated, disturbing *memories*, *thoughts*, or *images* of a stressful experience from the past?					
2.	Repeated, disturbing *dreams* of a stressful experience from the past?					
3.	Suddenly *acting* or *feeling* as if a stressful experience *were happening again* (as if you were reliving it)?					
4.	Feeling *very upset* when *something reminded* you of a stressful experience from the past?					
5.	Having *physical reactions* (e.g., heart pounding, trouble breathing, or sweating) when *something reminded* you of a stressful experience from the past?					
6.	Avoid *thinking about* or *talking about* a stressful experience from the past or avoid *having feelings* related to it?					
7.	Avoid *activities* or *situations* because *they remind you* of a stressful experience from the past?					
8.	Trouble *remembering important parts* of a stressful experience from the past?					
9.	Loss of *interest in things that you used to enjoy*?					
10.	Feeling *distant* or *cut off* from other people?					
11.	Feeling *emotionally numb* or being unable to have loving feelings for those close to you?					
12.	Feeling as if your *future* will somehow be *cut short*?					
13.	Trouble *falling* or *staying asleep*?					
14.	Feeling *irritable* or having *angry outbursts*?					
15.	Having *difficulty concentrating*?					
16.	Being *"super alert"* or watchful on guard?					
17.	Feeling *jumpy* or easily startled?					

Weathers, F.W., Huska, J.A., Keane, T.M. *PCL-C for DSM-IV.* Boston: National Center for PTSD – Behavioral Science Division, 1991.

PTSD CheckList – Military Version (PCL-M)

Patient's Name: _____

Instruction to patient: Below is a list of problems and complaints that veterans sometimes have in response to stressful military experiences. Please read each one carefully, put an "X" in the box to indicate how much you have been bothered by that problem in *the last month.*

No.	Response:	Not at all (1)	A little bit (2)	Moderately (3)	Quite a bit (4)	Extremely (5)
1.	Repeated, disturbing *memories*, *thoughts*, or *images* of a stressful military experience?					
2.	Repeated, disturbing *dreams* of a stressful military experience?					
3.	Suddenly *acting* or *feeling* as if a stressful military experience *were happening again* (as if you were reliving it)?					
4.	Feeling *very upset* when *something reminded* you of a stressful military experience?					
5.	Having *physical reactions* (e.g., heart pounding, trouble breathing, or sweating) when s*omething reminded* you of a stressful military experience?					
6.	Avoid *thinking about* or *talking about* a stressful military experience or avoid *having feelings* related to it?					
7.	Avoid *activities* or *situations* because *they remind you* of a stressful military experience?					
8.	Trouble *remembering important parts* of a stressful military experience?					
9.	Loss of *interest in things that you used to enjoy*?					
10.	Feeling *distant* or *cut off* from other people?					
11.	Feeling *emotionally numb* or being unable to have loving feelings for those close to you?					
12.	Feeling as if your *future* will somehow be *cut short*?					
13.	Trouble *falling* or *staying asleep*?					
14.	Feeling *irritable* or having *angry outbursts*?					
15.	Having *difficulty concentrating*?					
16.	Being "*super alert"* or watchful on guard?					
17.	Feeling *jumpy* or easily startled?					

Weathers, F.W., Huska, J.A., Keane, T.M. *PCL-M for DSM-IV.* Boston: National Center for PTSD – Behavioral Science Division, 1991.

PTSD CheckList – Stressor Specific Version (PCL-S)

The event you experienced was: _____ on: _____

Instruction to patient: Below is a list of problems and complaints that veterans sometimes have in response to stressful military experiences. Please read each one carefully, put an "X" in the box to indicate how much you have been bothered by that problem in *the last month.*

No.	Response:	Not at all (1)	A little bit (2)	Moderately (3)	Quite a bit (4)	Extremely (5)
1.	Repeated, disturbing *memories*, *thoughts*, or *images* of the stressful experience?					
2.	Repeated, disturbing *dreams* of the stressful experience?					
3.	Suddenly *acting* or *feeling* as if the stressful experience *were happening again* (as if you were reliving it)?					
4.	Feeling *very upset* when *something reminded* you of the stressful experience?					
5.	Having *physical reactions* (e.g., heart pounding, trouble breathing, or sweating) when *something reminded* you of the stressful experience?					
6.	Avoid *thinking about* or *talking about the* stressful experience or avoid *having feelings* related to it?					
7.	Avoid *activities* or *situations* because *they remind you* of the stressful experience?					
8.	Trouble *remembering important parts* of the stressful experience?					
9.	Loss of *interest in things that you used to enjoy*?					
10.	Feeling *distant* or *cut off* from other people?					
11.	Feeling *emotionally numb* or being unable to have loving feelings for those close to you?					
12.	Feeling as if your *future* will somehow be *cut short*?					
13.	Trouble *falling* or *staying asleep*?					
14.	Feeling *irritable* or having *angry outbursts*?					
15.	Having *difficulty concentrating*?					
16.	Being *"super alert"* or watchful on guard?					
17.	Feeling *jumpy* or easily startled?					

Weathers, F.W., Huska, J.A., Keane, T.M. *PCL-S for DSM-IV.* Boston: National Center for PTSD – Behavioral Science Division, 1991.

This is a Government document in the public domain.

APPENDIX D
Participant List

Curtis Aberle, RN, **NP**
Chief, FMS McWethy
Brooke Army Medical Center
Fort Sam Houston, TX 78234
Phone: 210-295-4667
Email: curtis.aberle@amedd.army.mil

Ron Acierno, Ph.D
PCT Director, Ralph H. Johnson VAMC
MUSC Crime Victims Center
109 Bee Street
Charleston, SC 29401
Phone: 843-792-2945
Email: acierno@musc.edu

Carla Cassidy, RN, MSN, CRNP
Director, Evidence Based Practice Program
Department of Veterans Affairs
1717 H Street
4th Floor, Room 406
Washington, DC 20006
Phone: 202- 266-4502
Email: Carla.cassidy@va.gov

Kathleen M. Chard, PhD
VA CPT Implementation Director
Director, PTSD and Anxiety Disorders Division
Cincinnati VA Medical Center
Address: 3200 Vine Street, Cincinnati, Ohio 45220
Phone: 859-572-6741
Email: Kathleen.Chard@va.gov

Debra Dandridge, PharmD
Clinical Pharmacist
Brooke Army Medical Center
Department of Pharmacy
Fort Sam Houston, TX 78234
Phone: 210-916-2612
Email: debra.dandridge@us.army.mil

Justin S. Campbell, PhD
LCDR, U.S. Navy Medical Service Corps
Senior Analyst, Deployment Health
Bureau of Medicine and Surgery
2300 E. St. NW
Washington, DC, 20372
Phone: 202-762-3013
Email: justin.s.campbell@med.navy.mil

Daniella David, MD, M.Sc
Professor of Clinical Psychiatry, University of Miami
PTSD Program Medcial Director, Miami VA healthcare System
1201 NW 16th Street, 116A12
Miami, FL 33125
Phone: 305-575-7000, ext 3953
Email: Daniella.david@va.gov

Ernest Degenhardt, MSN, FNP, RN
Chief, Evidence-Based Practice
US Army Medical Command
2050 Worth Road, Suite 26
Fort Sam Houston TX 78234
Phone: 210-221-6527
Email: ernest.degenhardt@amedd.army mil

Martha D'Erasmo MPH
Independent Consultant
4550 North Park Ave, # 505
Chevy Chase, MD 20815
Phone: 301- 654-3152
Email: Marty@hqiinc.com

Kathryn J. Dolter, RN, PhD
Clinical Quality Program Specialist
Evidence-Based Clinical Practice Guidelines
Department of Veterans Affairs
1717 H. Street, N.W.
Washington, D.C., 20006
Phone: 202-461-1078
E-mail: Kathryn.Dolter@va.gov

Charles C Engel, MD, MPH
Colonel, MC, US Army
Dir, DoD Deployment Health Clinical Center
Assoc Chair (Research), Department of Psychiatry
Uniformed Services University School of Medicine
Phone: 202-782-8064
Email: charles.engel@amedd.army.mil

Rosalie Fishman, RN, MSN, CPHQ
President
Healthcare Quality Informatics, Inc.
2275 Shady Grove Rd, Suite 500
Rockville, MD 20850
Phone: 301- 296-4542
Email Rosalie@hqiinc.com

Joel T. Foster, PhD,
Capt, US Air Force, BSC
Licensed Psychologist
ADAPT Program Element Chief
Travis AFB/60 MDOS/SGOW
Phone: 707-423-2317
Email: joel.foster-02@travis.af.mil

Matthew J. Friedman, MD, Ph.D
Executive Director
National Center for PTSD
White River Junction VA Medical Center
215 North Main Street
White River Junction, VT 05009
Phone: 802-296-5132
Email: Matthew.Friedman@VA.gov or
Matthew.J.Friedman@Dartmouth.edu

Stella M Hayes, MD
CDR, US Navy
Family Physician / Deputy Group Surgeon
2nd Marine Logistic Group
ATTN: Group Surgeon's Office
308 G Street
Camp Lejeune, NC 28547
Phone (910)450-6675
Email: stella.hayes@med.navy.mil

MAJ Kenneth Hyde, PA-C
MAJ, US Army
Madigan Army Medical Center
ATTN: MCHJ-EM
Bldg 9040, Rm 1-54-02
Tacoma, WA 98431
Phone: 253-968-0599
Email: ken.hyde@us.army mil

Charles Hoge, MD
Director, Division of Psychiatry and Neuroscience
Walter Reed Army Institute of Research
503 Robert Grant Ave
Washington, DC 20307-5001
Phone: 301-319-9342
Email: Charles hoge@us.army mil

Matthew D. Jeffreys, MD
Associate Professor of Psychiatry, UTHSCSA
PCT Medical Director
South Texas Health care System
5788 Eckhert Rd
San Antonio, TX 78240
Phone: 210-699-2145
Email: matthew.jeffreys@va.gov

Terence M. Keane, Ph.D
Associate Chief of staff, Research & development
VA Boston Healthcare System
150 South Huntington Avenue
Boston, MA 02130
Phone: 857-364-4551
Email: Terry.Keane@va.gov

Robert L. Koffman, MD, MPH
Captain, MC, US Navy
Deputy Director for Clinical Operations
National Intrepid Center of Excellence
for Psychological Health and Traumatic Brain Injury
8901 Wisconsin Blvd
Bethesda, MD 20889-5600
Phone: 301-319-3606
Email: robert.koffman@med navy mil

Harold Kudler, MD
Coordinator, VISN 6 Mental Health Service Line
Durham VA Medical Center
508 Fulton St.
Durham , NC 27705
Phone: 919-451-3369
Email: Harold.Kudler@va.gov

James R. Liffrig, MD, MPH
COL, MC, US Army Chief, Department of Family Medicine
Womack Army Medical Center
Fort. Bragg NC 28310
Phone: 910-907-6823
Email: james.liffrig@amedd.army.mil

Patrick J. Lowry, MD
COL, MC, US Army
Psychiatrist
Munson Army Health Center
550 Pope Ave
Ft Leavenworth, KS 66027
Phone: 913-758-3751
Email: patrick.lowry@us.army mil

Sandra McNaughton, RN, NP
LTC, AN, US Army
Walter Reed Army Medical Center
6900 Georgia Ave
Washington DC 20307
Phone: 202-782-7230
Email: sandra.mcnaughton@us.arm. mil

David T. Orman, MD
Chief, PTSD-TBI/BH
Integration (PTBI)
US Army Medical Command
2050 Worth Road
Fort Sam Houston, TX 78234
Phone: 210-221-6792
Email: david.orman@amedd.army.mil

Alan L. Peterson, Ph.D
Professor
University of Texas Health Science Center at San Antonio
Department of Psychiatry
7703 Floyd Curl Dr.
San Antonio, TX 78229-3900
Phone: 210-508-5428
Email: peterson3@uthscsa.edu

Sheila M. Rauch, Ph.D
Director, SeRV-MH
VA Ann Arbor healthcare System
Assistant Professor of Psychology in Psychiatry
University of Michigan Medical School
2215 Fuller Rd (116c)
Ann Arbor, MI 48105
Phone: 734-845-3545
Email: sherauch@med.umich.edu or
Sheila rauch@va.gov

Miguel E. Roberts, Ph.D
Chief, Psychological Health Clinical Guidelines
Defense Centers of Excellence for Psychological Health
and Traumatic Brain Injury
Psychological Health Clinical Standards of Care
1335 East West Hwy, 9th Floor
Silver Spring, MD 20910
Phone: 301-295-3541
Email: miguel roberts@tma.osd mil

Josef I. Ruzek, Ph.D
Director/Chief, Dissemination and Training Division,
National Center for PTSD
VA Palo Alto Health Care System
National Center for PTSD
785 Willow Road
Menlo Park CA 94025
Phone: 650-493-5000 ext. 22977
Email: Josef.Ruzek@va.gov

Todd P. Semla, MS, Pharm.D.
Clinical Pharmacy Specialist
Department of Veterans Affairs
National Pharmacy Benefits Management Services (119D)
1st Ave-1 Blk N of Cermak Rd (Building 37, Rm 139)
Hines, Il 60141
Phone:708-786-7976
Email: Todd.semla@va.gov

Murray B. Stein, MD, MPH
Professor of Psychiatry and family Preventive Medicine, UCSD
Staff Psychiatrist VA San Diego Healthcare System
UCSD and VASDHS
9500 Gilman Dr.(Is this VA address?)
La Jolla, CA 92093-0855
Phone: 858-534-6451
Email: mstein@ucsd.edu

Steven M. Southwick, MD
Professor Psychiatry, Yale University
Deputy Director Clinical Neuroscience Division
National Center for PTSD
VA Connecticut Healthcare System
Yale University Medical School
950 Campbell Ave
West Haven, CT 05516
Phone: 203-932-5711 ext. 2464
Email:Steven.southwick@va.gov

Mark B. Stephens, MD MS FAAFP
CAPT, MC, US Navy
Associate Professor and Chair
Department of Family Medicine
Uniformed Services University
4301 Jones Bridge Rd.
Bethesda, MD 20814-4799
Phone: 301-295-3632
Email:mstephens@usuhs mil

Frances Stewart, MD
CAPT, MC, US Navy
Department of Psychiatry
National Naval Medical Center
8901 Rockville Pike
Bethesda, MD 20889
Phone: 202-685-6443
Email: frances.stewart@navy.mil

Oded Susskind, MPH
Medical Education Consultant
PO Box 112
Brookline MA 02446
Phone: 617- 232-3558
Email: Oded@tiac.net

Christopher Warner, MD, FAAFP
MAJ(P), MC, US Army
Command and General Staff College
100 Stimson Drive
Fort Leavenworth, KS 66027
Phone; 913-424-9472
Email: christopher.h.warner@us.army mil

Marjory K. Waterman, MN, RN
Nurse Consultant/ CPG Coordinator
U.S. Army Medical Command
Evidence Based Practice Office
ATTN: MCHO-CL-Q
2050 Worth Road, Suite 26
Fort Sam Houston, TX 78234
Phone: 210-221-7281
Email: Marjory.waterman@us.army mil

Robert J. Wilson, PsyD, ABPP
Col US Air Force
Director, Psychological Health Clinical Standards of Care
Defense Centers of Excellence for Psychological Health
and Traumatic Brain
Injury
Email: Robert.wilson@lakenheath.af.mil

Randon S. Welton, MD
Lt Col, USAF, MC
Chief, Modernization Division
Office of the Command Surgeon
AFMC/SGR
Phone:-937-656-3642
Email: randon.welton@wpafb.af.mil

CONTRIBUTORS AND REVIEWERS:

Nancy C. Bernardy, Ph.D.
Associate Director for Clinical Networking
PTSD Mentoring Program Manager
VA National Center for PTSD 116D
215 N. Main St.
White River Junction, VT 05009
(802) 296-5132 fax (802) 296-5135
Nancy.Bernardy@va.gov

Edward A Brusher, LCSW, BCD
LTC, MS US Army
Chief, Deputy Director, Behavioral Health Division
Office of the Surgeon General
BHD, HP&S Directorate, OTSG
Falls Church, VA 22041-3258
Phone: 703-681-4188
Email: edward.brusher@us.army mil

Bruce Capehart, MD, MBA
Psychiatry, Attending
Durham VAMC
OEF/OIF Program (122B)
508 Fulton Street
Durham, NC 27705
Phone: (919) 296-0411, ext 5112
Email: bruce.capehart@va.gov

Michael E. Clark, PhD
Clinical Director, Chronic Pain rehabilitation Program
Thomas A. Haley veterans Hospital
13000 Bruce B. Blvd
Tampa, FL 33612
Phone:813-972-2000 ext. 7484
Email: Michael.clark8@med.va.gov

Kent, Drescher, Kent
Health Science Specialist
Dept of Veterans Affairs
Nat Ctr PTSD
Palo Alto VAMC - NatCtrPTSD
795 Willow Rd Bld 352/111
Menlo Park, CA 94025
Phone: 650.493.5000 x22071
Email: kent.drescher@va.gov

Barbara A. Hermann, PhD
Associate Director for Research and Education
VA National Center for PTSD (116D)
215 North Main St.
White River Junction, VT 05009
802-296-5132 x6082 fax (802) 296-5135
Barbara.Herman@va.gov

Carolyn J. Greene, PhD
Clinical Psychologist
National Center for PTSD, Dissemination and Training Division
VA Palo Alto HCS
795 Willow Road (PTSD-334)
San Francisco, CA 94103
Phone: 650-493-5000 ext 22107
Email: Carolyn.greene3@va.gov

Julia Hoffman, Psy.D
Clinical Psychologist
National Center for Telehealth & Technology
795 Willow Road, 334-PTSD
Menlo Park, CA 94025
Phone: 650-493-5000 x20007
Email: Julia.e hofman@us.army.mil

Eric Kuhn, PhD
Co-Director for Education, PTSD
VA Sierra Pacific (VISN 21) Mental Illness Research,
Education, & Clinical Center (MIRECC)
VA National Center for PTSD, Dissemination and Training Division
795 Willow Road Menlo Park, CA 94025
Phone: (650) 493-5000 x23160
Email: eric kuhn@va.gov

Walter E. Penk, PhD, ABPP
Consultant,
Central Texas VA Healthcare System
1936 Oak Glen,
New Braunfels, Texas, 78132
Phone: 830-620-0222
Email: wepenk@att.net

James Spira, PhD, MPH
Director NCPTSD-Pacific Islands Division
Department of Veterans Affair
3375 Koapaka St. I-560
Honolulu, Hawaii 96819
Phone: 808-954-6390
Email: James.spira@va.gov

Paula P. Schnurr, Ph.D.
Deputy Executive Director
National Center for PTSD (116D)
VA Medical and Regional Office Center
White River Junction, VT 05009
Phone: (802) 296-5132 FAX (802) 296-5135
Email: spaula.schnurr@va.gov
or paula.P.Schnurr@Dartmouth.EDU

Jennifer Vasterling, PhD
Chief, Psychology Service
VA Medical Center – Boston
150 S Huntington Ave
Jamaica Plain, MA 02130
Phone: (857) 364-4038
Email: jennifer.vasterling@va.gov

APPENDIX E
Bibliography

Abramowitz EG, Barak Y, Ben-Avi I, Knobler HY. Hypnotherapy in the treatment of chronic combat-related PTSD patients suffering from insomnia: a randomized, zolpidem-controlled clinical trial. Int J Clin Exp Hypn 2008;56:270-80.

Acierno RA, Kilpatrick DG, Resnick HS et al. Violent assault, posttraumatic stress disorder, and depression. Risk factors for cigarette use among adult women. Behav Modif 1996; 20 (4):363-84.

Adamou M, Puchalska S, Plummer W, Hale AS. Valproate in the treatment of PTSD: systematic review and meta analysis. Curr Med Res Opin 2007;23:1285-91.

Adler AB, Litz BT, Castro CA, Suvak M, Thomas JL, Burrell L, McGurk D, Wright KM, Bliese PD. A group randomized trial of critical incident stress debriefing provided to U.S. peacekeepers. J Trauma Stress 2008;21:253-63.

Adler AD, Bliese PD, McGurk D, Hoge CW, Castro CA. Battlemind debriefing and battlemind training as eraly interventions with soldiers returning from Iraq: randomization by Platoon. Journal of Consulting and Clinical Psychology.2009; 77:928–940.

Aerni A, Traber R, Hock C, et al. Low-dose cortisol for symptoms of posttraumatic stress disorder. Am J Psychiatry. 2004;161(8):1488-1490.

Afifi TO, Asmundson GJ, Taylor S, Jang KL. The role of genes and environment on trauma exposure and posttraumatic stress disorder symptoms: a review of twin studies. Clin Psychol Rev 2010;30:101-12.

AHRQ, 2009 - Santaguida PL, Gross A, Busse J, Gagnier J, Walker K, Bhandari M, Raina P. Complementary and alternative medicine in back pain utilization report. Evid Rep Technol Assess (Full Rep) 2009:1-221.

Amir M, Kaplan Z, Efroni R et al. Suicide risk and coping styles in posttraumatic stress disorder patients. Psychother Psychosom 1999; 68 (2):76-81.

Amir M, Kaplan Z, Neumann L, et al: Posttraumatic stress disorder tenderness and fibromyalgia. J Psychosom Res 1997; 42:607–613.

Andrade J, Kavanagh D, Baddeley A. Eye-movements and visual imagery: A working memory approach to the treatment of post-traumatic stress disorder. British Journal of Clinical Psychology 1997; 36:209-23.

Angst MS, Clark JD. Opioid-induced hyperalgesia: a qualitative systematic review. Anesthesiology 2006;104:570-87.

Anthony WA, Rogers ES, Cohen M et al. Relationships between psychiatric symptomatology, work skills, and future vocational performance. Psychiatr Serv 1995; 46 (4):353-8.

APA (1994) - Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV). Washington, DC, American Psychiatric Association; 1994.

APA (2004) - American Psychiatric Association. Practice Guideline for the Treatment of Patients with Acute Stress Disorder and Posttraumatic Stress Disorder. Arlington, VA: American Psychiatric Association Practice Guidelines; 2004.

APA (2009), Practice Guideline for the Treatment of Patients with Acute Stress Disorder and Posttraumatic Stress Disorder: Guideline Watch, March 2009.

Armenian HK, Morikawa M, Melkonian AK, Hovanesian AP, Haroutunian N, Saigh PA, Akiskal K, Akiskal HS. Loss as a determinant of PTSD in a cohort of adult survivors of the 1988 earthquake in Armenia: implications for policy. Acta Psychiatr Scand 2000;102:58-64.

Asmundson GJ, Coons MJ, Taylor S, Katz J. PTSD and the experience of pain: research and clinical implications of shared vulnerability and mutual maintenance models. Can J Psychiatry 2002;47:930-7.

Asmundson GJ, Norton GR, Allerdings MD, Norton PJ, Larsen DK. Posttraumatic stress disorder and work-related injury. J Anxiety Disord 1998;12:57-69.

Baker DG, Mendenhall CL, Simbartl LA, Magan LK, Steinberg JL. Relationship between posttraumatic stress disorder and self-reported physical symptoms in Persian Gulf War veterans. Arch Intern Med 1997;157:2076-8.

Baldwin SA, Murray DM, Shadish WR. Empirically supported treatments or type I errors? Problems with the analysis of data from group-administered treatments. J Consult Clin Psychol 2005;73:924-35.

Ballantyne JC, Shin NS. Efficacy of opioids for chronic pain: a review of the evidence. Clin J Pain 2008;24:469-78.

Barnes PM, Bloom B, Nahin R. Complementary and alternative medicine use among adults and children: United States, 2007 (PDF). *CDC National Health Statistics Report #12*. 2008.

Barnett SD, Tharwani HM, Hertzberg MA, Sutherland SM, Connor KM, Davidson JR. Tolerability of fluoxetine in posttraumatic stress disorder. Prog Neuropsychopharmacol Biol Psychiatry 2002;26:363-7.

Barrowcliff AL, Gray NS, MacCulloch S, Freeman TC, MacCulloch MJ. Horizontal rhythmical eye movements consistently diminish the arousal provoked by auditory stimuli. Br J Clin Psychol 2003;42:289-302.

Bartone P. Hardiness as a resiliency factor for United States forces in the Gulf War. Posttraumatic stress intervention: Challenges, issues, and perspective. 2000.

Bartzokis G, Lu PH, Turner J, Mintz J, Saunders CS. Adjunctive risperidone in the treatment of chronic combat-related posttraumatic stress disorder. Biol Psychiatry 2005;57:474-9.

Basoglu M, Salcioglu E, Livanou M. A randomized controlled study of single-session behavioural treatment of earthquake-related post-traumatic stress disorder using an earthquake simulator. Psychological Medicine 2007;37:203-13.

Bayot A, Capafons A, Cardena E. Emotional self-regulation therapy: a new and efficacious treatment for smoking. Am J Clin Hypn 1997; 40 (2):146-56.

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.

Beck A. Thinking and depression: II theory and therapy. Arch Gen Psychiatry 1964; 10:561-71.

Beck JG, Coffey SF, Foy DW, Keane TM, Blanchard EB. Group cognitive behavior therapy for chronic posttraumatic stress disorder: an initial randomized pilot study. Behav Ther 2009;40:82-92.

Beck JG, Coffey SF. Group Cognitive Behavioral Treatment for PTSD: Treatment of Motor Vehicle Accident Survivors. Cogn Behav Pract 2005;12:267-77.

Becker ME, Hertzberg MA, Moore SD, Dennis MF, Bukenya DS, Beckham JC. A placebo-controlled trial of bupropion SR in the treatment of chronic posttraumatic stress disorder. J Clin Psychopharmacol 2007;27:193-7.

Beckham JC, Crawford AL, Feldman ME, Kirby AC, Hertzberg MA, Davidson JR, Moore SD. Chronic posttraumatic stress disorder and chronic pain in Vietnam combat veterans. J Psychosom Res 1997;43:379-89.

Bee PE, Bower P, Lovell K, Gilbody S, Richards D, Gask L, Roach P. Psychotherapy mediated by remote communication technologies: a meta-analytic review. BMC Psychiatry 2008;8:60.

Bell M, Lysaker P. Levels of expectation for work activity in schizophrenia: clinical and rehabilitation outcomes. Psychiatric Rehabil 1996; 19 (3):71-6.

Bell MD, Lysaker PH, Milstein RM. Clinical benefits of paid work activity in schizophrenia. Schizophr Bull 1996; 22 (1):51-67.

Bell MD, Milstein RM, Lysaker PH. Pay and participation in work activity: clinical benefits for clients with schizophrenia. Psychosocial Rehabil 1993; 17 (2):173-7.

Benattia I, Ahmed S, Pedersen R, Musgnung J. Treatment of posttraumatic stress disorder with venlafaxine extended release: a 6-month randomized controlled trial. Arch Gen Psychiatry 2006;63:1158-65.

Benotsch EG, Brailey K, Vasterling JJ et al. War zone stress, personal and environmental resources, and PTSD symptoms in Gulf War veterans: a longitudinal perspective. J Abnorm Psychol 2000; 109 (2):205-13.

Bent S, Padula A, Moore D, Patterson M, Mehling W. Valerian for sleep: a systematic review and meta-analysis. Am J Med 2006;119:1005-12.

Biddle D, Elliott P, Creamer M, Forbes D, Devilly GJ. Self-reported problems: a comparison between PTSD-diagnosed veterans, their spouses, and clinicians. Behav Res Ther 2002;40:853-65.

Birmes P, Carreras D, Ducasse JL et al. Peritraumatic dissociation, acute stress, and early posttraumatic stress disorder in victims of general crime. Can J Psychiatry 2001; 46 (7):649-51.

Bisson J, Andrew M. Psychological treatment of post-traumatic stress disorder (PTSD). Cochrane Database Syst Rev 2007:CD003388.

Bisson J, McFarlane S, Rose S, Ruzek J, Watson P. Psychological Debriefing for Adults, Chapter In EB Foa, TM Keane, MJ Friedman (Eds.), Effective treatments for PTSD: Practice Guidelines from the International Society for Traumatic Stress Studies. Second Edition; NY and London, Guilford Press:2009.

Bisson JI, Jenkins PL, Alexander J et al. Randomised controlled trial of psychological debriefing for victims of acute burn trauma. Br J Psychiatry 1997; 171:78-81.

Blanchard EB, Hickling EJ, Devineni T, Veazey CH, Galovski TE, Mundy E, Malta LS, Buckley TC. A controlled evaluation of cognitive behavioural therapy for posttraumatic stress in motor vehicle accident survivors. Behav Res Ther 2003;41:79-96.

Blanchard EB, Hickling EJ, Mitnick N et al. The impact of severity of physical injury and perception of life threat in the development of post-traumatic stress disorder in motor vehicle accident victims. Behav Res Ther 1995; 33 (5):529-34.

Bliese PD, Wright KM, Adler AB, Cabrera O, Castro CA, Hoge CW. Validating the primary care posttraumatic stress disorder screen and the posttraumatic stress disorder checklist with soldiers returning from combat. J Consult Clin Psychol 2008;76:272-81.

Board on Population Health and Public Health Practice at the National Academies of Science (2008) - Committee on Gulf War and Health: Updated Literature Review of Depleted Uranium, Institute of Medicine. Available at: http://www.nap.edu/catalog.php?record_id=12183

Boggio PS, Rocha M, Oliveira MO, Fecteau S, Cohen RB, Campanhã C, Ferreira-Santos E, Meleiro A, Corchs F, Zaghi S, Pascual-Leone A, Fregni F. Noninvasive brain stimulation with high-frequency and low-intensity repetitive transcranial magnetic stimulation treatment for posttraumatic stress disorder. J Clin Psychiatry. 2010 Aug;71(8):992-9.

Bohus M, Haaf B, Stiglmayr C et al. Evaluation of inpatient dialectical-behavioral therapy for borderline personality disorder--a prospective study. Behav Res Ther 2000; 38 (9):875-87.

Bond GR, Becker DR, Drake RE, Rapp CA, Meisler N, Lehman AF, Bell MD, Blyler CR. Implementing supported employment as an evidence-based practice. Psychiatr Serv 2001;52:313-22.

Bond GR, Drake RE, Becker DR Mueser KT. Effectiveness of psychiatric rehabilitation approaches for employment of people with severe mental illness. Journal of Disability Policy Studies 1997:10 (1);18-52.

Boschen, M. Mobile telephone and psychotherapy: I Capability and Applicability. The Behavior Therapist.2010; 33 (*1): 168-175*

Boudewyns PA, Stwerka SA, Hyer A, Albrecht W, Sperr EV. Eye movement desensitization and reprocessing. A pilot study. Behavior Therapist.1993:16;30-3.

Bradbury, J., Myers, S. Oliver, C. An adaptogenic role for omega-3 fatty acids in stress; a randomised placebo controlled double blind intervention study (pilot). Nutrition Journal 2004; 3:20.

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;283:1837-44.

Brady KT, Dansky BS, Back SE, Foa EB, Carroll KM. Exposure therapy in the treatment of PTSD among cocaine-dependent individuals: preliminary findings. J Subst Abuse Treat 2001;21:47-54.

Brady KT, Sinha R. Co-occurring mental and substance use disorders: the neurobiological effects of chronic stress. Am J Psychiatry 2005;162:1483-93.

Brailey K, Vasterling JJ, Proctor SP, Constans JI, Friedman MJ. PTSD symptoms, life events, and unit cohesion in U.S. soldiers: baseline findings from the neurocognition deployment health study. J Trauma Stress 2007;20:495-503.

Braun P, Greenberg D, Dasberg H, Lerer B. Core symptoms of posttraumatic stress disorder unimproved by alprazolam treatment. J Clin Psychiatry 1990; 51 (6):236-8.

Bremner JD, Quinn J, Quinn W, Veledar E. Surfing the net for medical information about psychological trauma: an empirical study of the quality and accuracy of trauma-related websites. Med Inform Internet Med 2006;31:227-36.

Bremner JD, Southwick S, Brett E, Fontana A, Rosenheck R, Charney DS. Dissociation and posttraumatic stress disorder in Vietnam combat veterans. Am J Psychiatry 1992; 149 (3):328-32.

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 (6):902-7.

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;55:626-32.

Breslau N. Epidemiologic studies of trauma, posttraumatic stress disorder, and other psychiatric disorders. Can J Psychiatry 2002; 47 (10):923-9.

Breslau N. Outcomes of posttraumatic stress disorder. J Clin Psychiatry 2001; 62 Suppl 17:55-9.

Breuer J, Freud S. Studies on hysteria. In: J Strachey, editor, translator and editor The standard edition of the complete psychological works of Sigmund Freud. Vol. 2. London: Hogarth Press; 1955; p. 1-335.

Brewin CR, Andrews B, Rose S, Kirk M. Acute stress disorder and posttraumatic stress disorder in victims of violent crime. Am J Psychiatry 1999;156:360-6.

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 (5):748-66.

Brewin, CR. Systematic review of screening instruments for adults at risk of PTSD. J Trauma Stress 2005: **18**(1): 53-62.

Bridges KW, Goldberg DP. Somatic presentation of DSM III psychiatric disorders in primary care. J Psychosom Res 1985;29:563-9.

Brom D, Kleber RJ, Defares PB. Brief psychotherapy for posttraumatic stress disorders. J Consult Clin Psychol 1989; 57 (5):607-12.

Brooks AJ, Schwartz GE, Reece K, Nangle G. The effect of Johrei healing on substance abuse recovery: a pilot study. J Altern Complement Med 2006;12:625-31.

Brooks JS, Scarano T. Transcendental meditation in the treatment of post-vietnam adjustment. Journal of Counseling and Adjustment,1985; 64: 212-215.

Bryant RA, Creamer M, O'Donnell M, Silove D, McFarlane AC. A Study of the Protective Function of Acute Morphine Administration on Subsequent Posttraumatic Stress Disorder. Biol Psychiatry. 2008c Dec 4. (epub]

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 (5):862-6.

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;109:341-4.

Bryant RA, Marosszeky JE, Crooks J, Baguley IJ, Gurka JA. Interaction of posttraumatic stress disorder and chronic pain following traumatic brain injury. J Head Trauma Rehab 1999;14:588–94.

Bryant RA, Mastrodomenico J, Felmingham KL, Hopwood S, Kenny L, Kandris E, Cahill C, Creamer M. Treatment of acute stress disorder: a randomized controlled trial. Arch Gen Psychiatry 2008a;65:659-67.

Bryant RA, Moulds M, Guthrie R, Nixon RD. Treating acute stress disorder following mild traumatic brain injury. American Journal of Psychiatry 2003a;160:585-7.

Bryant RA, Moulds ML, Guthrie RM, Dang ST, Mastrodomenico J, Nixon RD, Felmingham KL, Hopwood S, Creamer M. A randomized controlled trial of exposure therapy and cognitive restructuring for posttraumatic stress disorder. J Consult Clin Psychol 2008b;76:695-703.

Bryant RA, Moulds ML, Guthrie RM, Dang ST, Mastrodomenico J, Nixon RD, Felmingham KL, Hopwood S, Creamer M. A randomized controlled trial of exposure therapy and cognitive restructuring for posttraumatic stress disorder. J Consult Clin Psychol 2008b;76:695-703.

Bryant RA, Moulds ML, Nixon RV. Cognitive behaviour therapy of acute stress disorder: a four-year follow-up. Behav Res Ther 2003b;41:489-94.

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;156:1780-6.

Bryant RA. Disentangling mild traumatic brain injury and stress reactions. N Engl J Med. 2008;358(5):525-7.

Bugg, A., G. Turpin, Scholes C.. A randomised controlled trial of the effectiveness of writing as a self-help intervention for traumatic injury patients at risk of developing post-traumatic stress disorder. Behav Res Ther 2009;47:6-12.

Bullman TA, Kang HK. Posttraumatic stress disorder and the risk of traumatic deaths among Vietnam veterans. J Nerv Ment Dis 1994; 182 (11):604-10.

Burt T, Lisanby SH, Sackeim HA. Neuropsychiatric applications of transcranial magnetic stimulation: A meta-analysis. International Journal of Neuropsychopharmacology 2002;5:73-103.

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 (4):197-203.

Cacciola JS, Koppenhaver JM, Alterman AI, McKay JR. Posttraumatic stress disorder and other psychopathology in substance abusing patients. Drug Alcohol Depend. 2009 Apr 1;101(1-2):27-33.

Campfield KM, Hills AM. Effect of timing of critical incident stress debriefing (CISD) on posttraumatic symptoms. J Trauma Stress 2001; 14 (2):327-40.

Canda ER, Phaobtong T. Buddhism as a supportive system for Southeast Asia refugees. Social Work 1992; 37:61-7.

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;18:379-83.

Cardena E, Alarcon A, Capafons A, Bayot A. Effects on suggestibility of a new method of active-alert hypnosis: alert hand. Int J Clin Exp Hypn 1998; 46 (3):280-94.

Carlson JG, Chemtob CM, Rusnak K, Hedlund NL, Muraoka MY. Eye movement desensitization and reprocessing (EDMR) treatment for combat-related posttraumatic stress disorder. J Trauma Stress 1998; 11 (1):3-24.

Cates ME, Bishop MH, Davis LL, Lowe JS, Woolley TW. Clonazepam for treatment of sleep disturbances associated with combat-related posttraumatic stress disorder. Ann Pharmacother 2004;38:1395-9.

Caudill-Slosberg MA, Schwartz LM, Woloshin S. Office visits and analgesic prescriptions for musculoskeletal pain in US: 1980 vs. 2000. Pain 2004;109:514-9.

Chard KM. An evaluation of cognitive processing therapy for the treatment of posttraumatic stress disorder related to childhood sexual abuse. J Consult Clin Psychol 2005;73:965-71.

Chemtob CM, Novaco RW, Hamada RS, Gross DM, Smith G. Anger regulation deficits in combat-related posttraumatic stress disorder. J Trauma Stress 1997;10:17-36.

Chemtob CM, Tolin DF, van der Kolk BA. Guidelines for treatment of PTSD: eye movement desensitization and reprocessing. J Trauma Stress 2000; 13:569-70.

Chou R, Qaseem A, Snow V, Casey D, Cross JT, Jr., Shekelle P, Owens DK. Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society. Ann Intern Med 2007;147:478-91.

Christman S, Garvey K. Episodic versus semantic memory: Eye movements and cortical activation. In Poster presented at the 41st Annual Meeting of the Psychonomic Society, New Orleans, LA; 2000.

Chung MY, Min KH, Jun YJ, Kim SS, Kim WC, Jun EM. Efficacy and tolerability of mirtazapine and sertraline in Korean veterans with posttraumatic stress disorder: a randomized open label trial. Hum Psychopharmacol 2004;19:489-94.

Clancy CP, Graybeal A, Tompson WP, Badgett KS, Feldman ME, Calhoun PS, Erkanli A, Hertzberg MA, Beckham JC. Lifetime trauma exposure in veterans with military-related posttraumatic stress disorder: association with current symptomatology. J Clin Psychiatry 2006;67:1346-53.

Clapp JD, Masci J, Bennett SA, Beck JG. Physical and psychosocial functioning following motor vehicle trauma: relationships with chronic pain, posttraumatic stress, and medication use. Eur J Pain 2010;14:418-25.

Clark ME, Bair MJ, Buckenmaier CC, 3rd, Gironda RJ, Walker RL. Pain and combat injuries in soldiers returning from Operations Enduring Freedom and Iraqi Freedom: implications for research and practice. J Rehabil Res Dev 2007;44:179-94.

Clark ME, Scholten JD, Walker RL, Gironda RJ. Assessment and treatment of pain associated with combat-related polytrauma. Pain Med 2009a;10:456-69.

Clark ME, Walker RL, Gironda RJ, Scholten JD. Comparison of pain and emotional symptoms in soldiers with polytrauma: unique aspects of blast exposure. Pain Med 2009b;10:447-55.

Clark ME. Post-deployment pain: a need for rapid detection and intervention. Pain Med 2004;5:333-4.

Clark ME. Understanding appropriate long-term use of opioids–seventeen years and counting. Clin J Pain 2008;24:467–8.

Clark PA, Drain M, Malone MP. Addressing patients' emotional and spiritual needs. Jt Comm J Qual Saf 2003;29:659-70.

Classen C, Koopman C, Hales R, Spiegel D. Acute stress disorder as a predictor of posttraumatic stress symptoms. Am J Psychiatry 1998; 155 (5):620-4.

Classen C, Koopman C, Nevill-Manning K, Spiegel D. A preliminary report comparing trauma-focused and present-focused group therapy against a wait-listed condition among childhood sexual abuse survivors with PTSD. J Aggression Maltreat Trauma 2001;4:265-88.

Cloitre M, Koenen KC, Cohen LR, Han H. Skills training in affective and interpersonal regulation followed by exposure: a phase-based treatment for PTSD related to childhood abuse. J Consult Clin Psychol 2002;70:1067-74.

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;51:379-98.

Cohen H, Kaplan Z, Kotler M, Kouperman I, Moisa R, Grisaru N. Repetitive transcranial magnetic stimulation of the right dorsolateral prefrontal cortex in posttraumatic stress disorder: a double-blind, placebo-controlled study. Am J Psychiatry 2004;161:515-24.

Coker AL, Smith PH, Thompson MP, McKeown RE, Bethea L, Davis KE. Social support protects against the negative effects of partner violence on mental health. J Womens Health Gend Based Med 2002;11:465-76.

Collinge W, Wentworth R, Sabo S. Integrating complementary therapies into community mental health practice: an exploration. J Altern Complement Med 2005;11:569-74.

Connor KM, Davidson JR, Weisler RH, Zhang W, Abraham K. Tiagabine for posttraumatic stress disorder: effects of open-label and double-blind discontinuation treatment. Psychopharmacology (Berl) 2006;184:21-5.

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.

Cook JM, Walser RD, Kane V, Ruzek JI, Woody G. Dissemination and feasibility of a cognitive-behavioral treatment for substance use disorders and posttraumatic stress disorder in the Veterans Administration. J Psychoactive Drugs 2006;38:89-92.

Cook, J. Characteristics of EIDP Participants and the Jobs They Hold: Preliminary Findings of the Employment Intervention Demonstration Program. Available on line at http://www.psych.uic.edu/EIDP/eidpcharacteristics.pdf

Cooper NA, Clum GA. Imaginal flooding as a supplementary treatment for PTSD in combat veterans: a controlled study. Behavioral Therapy 1989; 20:381-91.

Cottraux J, Note I, Yao SN, De Mey-Guillard C, Bonasse F, Djamoussian D, Mollard E, Note B, Chen Y. Randomized controlled comparison of cognitive behavior therapy with rogerian supportive therapy in chronic post-traumatic stress disorder: A 2-year follow-up. Psychotherapy and Psychosomatics 2008;77:101-10.

Coupland NJ, Lillywhite A, Bell CE, Potokar JP, Nutt DJ. A pilot controlled study of the effects of flumazenil in posttraumatic stress disorder. Biol Psychiatry 1997;41:988-90.

Courtois CA. Recollections of sexual abuse: treatment principles and guidelines. New York: Norton; 1999.

Cowell AJ, Pollio DE, North CS, Stewart AM, McCabe MM, Anderson DW. Deriving service costs for a clubhouse psychosocial rehabilitation program. Adm Policy Ment Health 2003;30:323-40.

Creamer M, Elliott P, Forbes D, Biddle D, Hawthorne G. Treatment for combat-related posttraumatic stress disorder: two-year follow-up. J Trauma Stress 2006;19:675-85.

Creamer M, Parslow R: Trauma exposure and posttraumatic stress disorder in the elderly: a community prevalence study. Am J Geriatr Psychiatry 2008, 16:853-856.

Crowther R, Marshall M, Bond G, Huxley P. Vocational rehabilitation for people with severe mental illness. Cochrane Database Syst Rev 2001:CD003080.

Crum RM, Anthony JC, Bassett SS, Folstein MF. Population-based norms for the Mini-Mental State Examination by age and educational level. JAMA 1993;269:2386-91.

Cuijpers P, Van Straten A, Smit F. Preventing the incidence of new cases of mental disorders: a meta-analytic review. J Nerv Ment Dis 2005;193:119-25.

Culhane, DP, Metraux S, Hadley T. Public service reductions associated with placement of homeless persons with severe mental illness in supportive housing.Housing Policy Debate.2002;13:07-163

Cummings JL. Mini-Mental State Examination. Norms, normals, and numbers. Jama 1993; 269 (18):2420-1.

Daly E, Wulff J. Treatment of a post-traumatic headache. Br J Med Psychol 1987; 60 (Pt 1):85-8.

Davidson J, Baldwin D, Stein DJ, Kuper E, Benattia I, Ahmed S, Pedersen R, Musgnung J. Treatment of posttraumatic stress disorder with venlafaxine extended release: a 6-month randomized controlled trial. Arch Gen Psychiatry 2006;63:1158-65.

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 (3):259-66.

Davidson J, Pearlstein T, Londborg P, Brady KT, Rothbaum B, Bell J, Maddock R, Hegel MT, Farfel G. Efficacy of sertraline in preventing relapse of posttraumatic stress disorder: results of a 28-week double-blind, placebo-controlled study. Am J Psychiatry 2001b; 158 (12):1974-81.

Davidson J, Rothbaum BO, Tucker P, Asnis G, Benattia I, Musgnung JJ. Venlafaxine extended release in posttraumatic stress disorder: a sertraline- and placebo-controlled study. J Clin Psychopharmacol 2006;26:259-67.

Davidson JR, Brady K, Mellman TA, Stein MB, Pollack MH. The Efficacy and Tolerability of Tiagabine in Adult Patients With Post-Traumatic Stress Disorder. Journal of Clinical Psychopharmacology 2007;27:85-8.

Davidson JR, Connor KM, Hertzberg MA, Weisler RH, Wilson WH, Payne VM. Maintenance therapy with fluoxetine in posttraumatic stress disorder: a placebo-controlled discontinuation study. J Clin Psychopharmacol 2005;25:166-9.

Davidson JR, Hughes D, Blazer DG, George LK. Post-traumatic stress disorder in the community: an epidemiological study. Psychol Med 1991; 21 (3):713-21.

Davidson JR, Kudler HS, Saunders WB, Erickson L, Smith RD, Stein RM, Lipper S, Hammett EB, Mahorney SL, Cavenar JO Jr. Predicting response to amitriptyline in posttraumatic stress disorder. Am J Psychiatry 1993; 150 (7):1024-9.

Davidson JR, Landerman LR, Farfel G, Clary CM. Characterizing the effects of sertraline in post-traumatic stress disorder. Psychol Med 2002; 32 (4):661-70.

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 2001a; 58 (5):485-92.

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;53:188-91.

Davidson PR, Parker KC. Eye movement desensitization and reprocessing (EMDR): a meta-analysis. J Consult Clin Psychol 2001; 69 (2):305-16.

Davidson RJ, Kabat-Zinn J, Schumacher J, Rosenkranz M, Muller D, Santorelli SF, Urbanowski F, Harrington A, Bonus K, Sheridan JF. Alterations in brain and immune function produced by mindfulness meditation. Psychosom Med 2003;65:564-70.

Davis LL, Davidson JR, Ward LC, Bartolucci A, Bowden CL, Petty F. Divalproex in the treatment of posttraumatic stress disorder: a randomized, double-blind, placebo-controlled trial in a veteran population. J Clin Psychopharmacol 2008;28:84-8.

Davis LL, Jewell ME, Ambrose S, Farley J, English B, Bartolucci A, Petty F. A placebo-controlled study of nefazodone for the treatment of chronic posttraumatic stress disorder: a preliminary study. J Clin Psychopharmacol 2004;24:291-7.

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 (2):159-64.

Davis LL, Ward C, Rasmusson A, Newell JM, Frazier E, Southwick SM. A placebo-controlled trial of guanfacine for the treatment of posttraumatic stress disorder in veterans. Psychopharmacol Bull 2008;41:8-18.

Davydow DS, Gifford JM, Desai SV, Needham DM, Bienvenu OJ. Posttraumatic stress disorder in general intensive care unit survivors: a systematic review. Gen Hosp Psychiatry 2008;30:421-34.

de Assis MA, de Mello MF, Scorza FA, Cadrobbi MP, Schooedl AF, Gomes da Silva S, de Albuquerque M, da Silva AC, Arida RM. Evaluation of physical activity habits in patients with posttraumatic stress disorder. Clinics (Sao Paulo) 2008;63:473-8.

de Vries GJ, Olff M. The lifetime prevalence of traumatic events and posttraumatic stress disorder in the Netherlands. J Trauma Stress 2009;22:259-67.

De Wind E. Some implications of former massive traumatization upon the actual analytic process. International Journal of Psychoanalysis 1984; 65:273-81.

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.

Devilly GJ, Annab R. A randomised controlled trial of group debriefing. J Behav Ther Exp Psychiatry 2008;39:42-56.

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 (1-2):131-57.

Diaz AB, Motta R. The effects of an aerobic exercise program on posttraumatic stress disorder symptom severity in adolescents. Int J Emerg Ment Health 2008;10:49-59.

Dierks MR, Jordan JK, Sheehan AH. Prazosin treatment of nightmares related to posttraumatic stress disorder. Ann Pharmacother 2007;41:1013-7.

Difede J, Malta LS, Best S, Henn-Haase C, Metzler T, Bryant R, Marmar C. A randomized controlled clinical treatment trial for World Trade Center attack-related PTSD in disaster workers. J Nerv Ment Dis 2007;195:861-5.

Dilk MN, Bond GR. Meta-analytic evaluation of skills training research for individuals with severe mental illness. J Consult Clin Psychol 1996; 64 (6):1337-46.

DiMascio A, Weissman MM, Prusoff BA, Neu C, Zwilling M,Klerman GL. Differential symptom reduction by drugs and psychotherapy in acute depression. Arch Gen Psychiatry 1979; 36 (13):1450-6.

Dohrenwend BP, Turner JB, Turse NA, Adams BG, Koenen KC, Marshall R. The psychological risks of Vietnam for U.S. veterans: a revisit with new data and methods. Science 2006;313:979-82.

Donovan B, Padin-Rivera E, Kowaliw S. "Transcend": initial outcomes from a posttraumatic stress disorder/substance abuse treatment program. J Trauma Stress 2001;14:757-72.

Donta ST, Clauw DJ, Engel CC, Jr., Guarino P, Peduzzi P, Williams DA, Skinner JS, Barkhuizen A, Taylor T, Kazis LE, Sogg S, Hunt SC, Dougherty CM, Richardson RD, Kunkel C, Rodriguez W, Alicea E, Chiliade P, Ryan M, Gray GC, Lutwick L, Norwood D, Smith S, Everson M, Blackburn W, Martin W, Griffiss JM, Cooper R, Renner E, Schmitt J, McMurtry C, Thakore M, Mori D, Kerns R, Park M, Pullman-Mooar S, Bernstein J, Hershberger P, Salisbury DC, Feussner JR. Cognitive behavioral therapy and aerobic exercise for Gulf War veterans' illnesses: a randomized controlled trial. JAMA 2003;289:1396-404.

Dougall AL, Herberman HB, Delahanty DL, Inslicht SS, Baum A. Similarity of prior trauma exposure as a determinant of chronic stress responding to an airline disaster. J Consult Clin Psychol 2000; 68 (2):290-5.

Dow B, Kline N. Antidepressant treatment of posttraumatic stress disorder and major depression in veterans. Ann Clin Psychiatry 1997; 9 (1):1-5.

Drake RE, McHugo GJ, Becker DR, Anthony WA, Clark RE. The New Hampshire study of supported employment for people with severe mental illness. J Consult Clin Psychol 1996;64:391-9.

Drebing CE, Van Ormer EA, Krebs C, Rosenheck R, Rounsaville B, Herz L, Penk W. The impact of enhanced incentives on vocational rehabilitation outcomes for dually diagnosed veterans. J Appl Behav Anal 2005;38:359-72.

Drebing CE, Van Ormer EA, Mueller L, Hebert M, Penk WE, Petry NM, Rosenheck R, Rounsaville B. Adding contingency management intervention to vocational rehabilitation: outcomes for dually diagnosed veterans. J Rehabil Res Dev 2007;44:851-65.

Drebing, C. E., Van Ormer, A., Schutt, R. K., Krebs, C., Losardo, M., Boyd, C., Penk, W. E., & Rosenheck, R. Client goals for participating in VHA vocational rehabilitation: Distribution and relationship to outcome. Rehabilitation Counseling Bulltetin.2004; 47: 162-172.

Duffy JD, Malloy PF. Efficacy of buspirone in the treatment of posttraumatic stress disorder: an open trial. Ann Clin Psychiatry 1994; 6 (1):33-7.

Duffy M, Gillespie K, Clark DM. Post-traumatic stress disorder in the context of terrorism and other civil conflict in Northern Ireland: Randomised controlled trial. BMJ: British Medical Journal 2007;334.

Dworkin RH, O'Connor AB, Backonja M, Farrar JT, Finnerup NB, Jensen TS, Kalso EA, Loeser JD, Miaskowski C, Nurmikko TJ, Portenoy RK, Rice AS, Stacey BR, Treede RD, Turk DC, Wallace MS. Pharmacologic management of neuropathic pain: evidence-based recommendations. Pain 2007;132:237-51.

Edlund MJ, Steffick D, Hudson T, Harris KM, Sullivan M. Risk factors for clinically recognized opioid abuse and dependence among veterans using opioids for chronic non-cancer pain. Pain 2007;129:355-62.

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;60:1024-32.

Ehlers A, Clark DM, Hackmann A, McManus F, Fennell M. Cognitive therapy for post-traumatic stress disorder: Development and evaluation. Behaviour Research and Therapy 2005;43:413-31.

Eichelman B. Hypnotic change in combat dreams of two veterans with posttraumatic stress disorder. Am J Psychiatry 1985; 142 (1):112-4.

Eid J, Johnsen BH, Weisaeth L. The effects of group psychological debriefing on acute stress reactions following a traffic accident: a quasi-experimental approach. Int J Emerg Ment Health 2001;3:145-54.

Ellis A: Reason and Emotion in Psychotherapy. New York, Stuart, 1962

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.

Eriksen J, Sjogren P, Bruera E, Ekholm O, Rasmussen NK. Critical issues on opioids in chronic non-cancer pain: an epidemiological study. Pain 2006;125:172-9.

Escalona R, Canive JM, Calais LA, Davidson JR. Fluvoxamine treatment in veterans with combat-related post-traumatic stress disorder. Depress Anxiety 2002; 15 (1):29-33.

Evans K, Tyrer P, Catalan J, Schmidt U, Davidson K, Dent J, Tata P, Thornton

Falsetti SA, Resnick HS, Davis J. Multiple channel exposure therapy: combining cognitive-behavioral therapies for the treatment of posttraumatic stress disorder with panic attacks. Behav Modif 2005;29:70-94.

Farrell KR, Ganzini L. Misdiagnosing delirium as depression in medically ill elderly patients. Arch Intern Med 1995; 155 (22):2459-64.

Fear NT, Jones M, Murphy D, Hull L, Iversen AC, Coker B, Machell L, Sundin J, Woodhead C, Jones N, Greenberg N, Landau S, Dandeker C, Rona RJ, Hotopf M, Wessely S. What are the consequences of deployment to Iraq and Afghanistan on the mental health of the UK armed forces? A cohort study. Lancet 2010;375:1783-97.

Feehan M, Nada-Raja S, Martin JA, Langley JD. The prevalence and correlates of psychological distress following physical and sexual assault in a young adult cohort. Violence Vict 2001; 16 (1):49-63.

Ferrada-Noli M, Asberg M, Ormstad K et al. Suicidal behavior after severe trauma. Part 1: PTSD diagnoses, psychiatric comorbidity, and assessments of suicidal behavior. J Trauma Stress 1998; 11 (1):103-12.

Feske U. Treating low-income and minority women with posttraumatic stress disorder: a pilot study comparing prolonged exposure and treatment as usual conducted by community therapists. J Interpers Violence 2008;23:1027-40.

Finnsdottir T, Elklit A. Posttraumatic sequelae in a community hit by an avalanche. J Trauma Stress 2002; 15 (6):479-85.

FM 21-11 First Aid for Soldiers document (1991) - The Department of the Army; Washington, DC, 4 December 1991. Complete document describing PFA components can be found at: http://www.vdh.state.va.us/EPR/pdf/PFA9-6-05Final.pdf

Foa EB, Dancu CV, Hembree EA, Jaycox LH, Meadows EA, Street GP. A comparison of exposure therapy, stress inoculation training, and their combination for reducing posttraumatic stress disorder in female assault victims. J Consult Clin Psychol 1999; 67 (2):194-200.

Foa EB, Davidson JRT, Frances A. The Expert Consensus Guideline Series: Treatment of Posttraumatic Stress Disorder. J Clin Psychiatry 1999a; 60 (Suppl 16).

Foa EB, Keane TM, Friedman MJ. Cohen J. (Eds.), Effective treatments for PTSD: Practice Guidelines from the International Society for Traumatic Stress Studies – 2nd Edition. New York: Guilford Press;2009.

Foa EB, Keane TM, Friedman MJ. Effective Treatments for PTSD: Practice Guidelines from the International Society for Traumatic Stress Studies. New York, NY: Guilford Press; 2000.

Foa EB, Meadows EA. Psychosocial treatments for posttraumatic stress disorder: a critical review. Annu Rev Psychol 1997; 48:449-80

Foa EB, Rauch SA. Cognitive changes during prolonged exposure versus prolonged exposure plus cognitive restructuring in female assault survivors with posttraumatic stress disorder. J Consult Clin Psychol 2004;72:879-84.

Foa EB, Riggs DS, Massie ED et al. The impact of fear activation and anger on the efficacy of exposure treatment for posttraumatic stress disorder. Behavior Therapy 1995; 26:487-99

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;59:715-23.

Foa EB, Zoellner LA, Feeny NC. An evaluation of three brief programs for facilitating recovery after assault. J Trauma Stress 2006;19:29-43.

Foa EB. Psychosocial treatment of posttraumatic stress disorder. J Clin Psychiatry 2000; 61 Suppl 5:43-8; discussion 9-51.

Folstein MF, Folstein SE, McHugh PR. "Mini-mental state". A practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res 1975; 12 (3):189-98.

Fontana A, Rosenheck R. Trauma, change in strength of religious faith, and mental health service use among veterans treated for PTSD. J Nerv Ment Dis 2004;192:579-84.

Forbes D, Phelps A, McHugh T. Treatment of combat-related nightmares using imagery rehearsal: a pilot study. J Trauma Stress 2001; 14 (2):433-42.

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;16:509-13.

Fossey MD, Hamner MB. Clonazepam-related sexual dysfunction in male veterans with PTSD. Anxiety 1994; 1 (5):233-6.

Foy DW, Card JJ. Combat-related post-traumatic stress disorder etiology: replicated findings in a national sample of Vietnam-era men. J Clin Psychol 1987;43:28-31.

Foy DW, Glynn SM, Schnurr PP, Jankowski MK, Wattenburg MS, Weiss DS. Group therapy. In EB Foa, TM Keane, MJ Friedman (Eds.), Effective treatments for PTSD: Practice Guidelines from the International Society for Traumatic Stress Studies. (pp. 155-175). Second Edition NY and London, Guilford Press:2009.

Foy DW, Ruzek JI, Glynn SM, Riney SJ, Gusman FD. Trauma focus group therapy for combat-related PTSD: an update. J Clin Psychol 2002;58:907-18.

Foy DW, Sipprelle RC, Rueger DB, Carroll EM. Etiology of posttraumatic stress disorder in Vietnam veterans: analysis of premilitary, military, and combat exposure influences. J Consult Clin Psychol 1984; 52 (1):79-87.

Freeman A, Datillo F, editors. Comprehensive casebook of cognitive therapy New York: Plenum Press; 1992.

Freeman A, Simon K, Beutler L et al., editors. Comprehensive handbook of cognitive therapy New York: Plenum Press; 1989.

Friedman MJ, Davidson JRT, Stein DJ. Psychopharmacotherapy for adults. In EB Foa, TM Keane, MJ Friedman (Eds.), Effective treatments for PTSD: Practice Guidelines from the International Society for Traumatic Stress Studies (pp. 245-268). Second Edition NY and London, Guilford Press:2009.

Friedman MJ, Marmar CR, Baker DG, Sikes CR, Farfel GM. Randomized, double-blind comparison of sertraline and placebo for posttraumatic stress disorder in a Department of Veterans Affairs setting. J Clin Psychiatry 2007;68:711-20.

Friedman MJ, Schnurr PP, McDonagh-Coyle A. Post-traumatic stress disorder in the military veteran. Psychiatr Clin North Am 1994;17:265-77.

Friedman MJ, Schnurr PP, Sengupta A, Holmes T, Ashcraft M. The Hawaii Vietnam Veterans Project: is minority status a risk factor for posttraumatic stress disorder? J Nerv Ment Dis 2004;192:42-50.

Frisman L, Ford J, Lin H, Mallon S, Chang R. Outcomes of trauma treatment using the TARGET model. J Groups Addict Recov 2008;3:285-303.

Frueh BC, Brady KL, de Arellano MA. Racial differences in combat-related PTSD: empirical findings and conceptual issues. Clin Psychol Rev 1998; 18 (3):287-305.

Frueh BC, Gold PB, de Arellano MA, Brady KL. A racial comparison of combat veterans evaluated for PTSD. J Pers Assess 1997; 68 (3):692-702.

Frueh BC, Monnier J, Yim E, Grubaugh AL, Hamner MB, Knapp RG. A randomized trial of telepsychiatry for post-traumatic stress disorder. J Telemed Telecare. 2007;13(3):142-7.

Frueh BC, Smith DW, Libet JM. Racial differences on psychological measures in combat veterans seeking treatment for PTSD. J Pers Assess 1996; 66 (1):41-53.

Fu SS, McFall M, Saxon AJ, Beckham JC, Carmody TP, Baker DG, Joseph AM. Post-traumatic stress disorder and smoking: a systematic review. Nicotine Tob Res 2007;9:1071-84.

Garfield DA, Fichtner CG, Leveroni C, Mahableshwarkar A. Open trial of nefazodone for combat veterans with posttraumatic stress disorder. J Trauma Stress 2001; 14 (3):453-60.

Gebhart RJ, Neeley FL. Primary care and PTSD. NCP Quarterly 1996; 6 (4):Fall.

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;66:207–14.

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 (9):390-4.

Gentilello LM, Rivara FP, Donovan DM, Jurkovich GJ, Daranciang E, Dunn CW, Villaveces A, Copass M, Ries RR.. Alcohol interventions in a trauma center as a means of reducing the risk of injury recurrence. Ann Surg 1999; 230 (4):473-80.

George MS, Ward HE Jr, Ninan PT, Pollack M, Nahas Z, Anderson B, Kose S, Howland RH, Goodman WK, Ballenger JC. A pilot study of vagus nerve stimulation (VNS) for treatment-resistant anxiety disorders. Brain Stimul. 2008 Apr;1(2):112-21.

Germain V, Marchand A, Bouchard S, Drouin MS, Guay S. Effectiveness of cognitive behavioural therapy administered by videoconference for posttraumatic stress disorder. Cogn Behav Ther 2009;38:42-53.

Gersons BP, Carlier IV, Lamberts RD, van der Kolk BA. Randomized clinical trial of brief eclectic psychotherapy for police officers with posttraumatic stress disorder. J Trauma Stress 2000;13:333-47.

Gidron YR, Gal R, Givati G, Lauden A. Snir Y, Benjamin J. Interactive effects of memory structuring and gender in preventing posttraumatic stress symptoms. Journal of Nervous & Mental Disease 2007;195:179-182

Gilbertson MW, Paulus LA, Williston SK, Gurvits TV, Lasko NB, Pitman RK, Orr SP. Neurocognitive function in monozygotic twins discordant for combat exposure: relationship to posttraumatic stress disorder. J Abnorm Psychol 2006;115:484-95.

Gill JM, Saligan L, Woods S, Page G. PTSD is associated with an excess of inflammatory immune activities. Perspect Psychiatr Care 2009;45:262-77.

Gillin JC, Smith-Vaniz A, Schnierow B, Rapaport MH, Kelsoe J, Raimo E, Marler MR, Goyette LM, Stein MB, Zisook S. An open-label, 12-week clinical and sleep EEG study of nefazodone in chronic combat-related posttraumatic stress disorder. J Clin Psychiatry 2001; 62 (10):789-96.

Gironda RJ, Clark ME, Massengale JP, Walker RL. Pain among veterans of Operations Enduring Freedom and Iraqi Freedom. Pain Med 2006;7:339-43.

Gist R, Lubin B, editors. Reponse to disaster: psychosocial, community, and ecological approaches Philadelphia, PA: Brunner/Mazel; 1999.

Glynn, S., Drebing, C., & Penk. W. Psychosocial rehabilitation. In EB Foa, TM Keane, MJ Friedman (Eds.), Effective treatments for PTSD: Practice Guidelines from the International Society for Traumatic Stress Studies. Second Edition NY and London, Guilford Press:2009.

Goldfinger SM, Schutt RK, Tolomiczenko GS, Seidman L, Penk WE, Turner W, Caplan B. Housing placement and subsequent days homeless among formerly homeless adults with mental illness. Psychiatr Serv. 1999 May;50(5):674-9.

Goldfinger SM, Schutt RK, Tolomiczenko GS. Housing persons who are homeless and mentally ill: independent living or evolving consumer households? In: WR Breakey; JW Thompson, editors, translator and editor Mentally ill and homeless special programs for special needs. Amsterdam: Harwood; 1997; p. 29-49.

Gould MS, Greenberg T, Velting DM, Shaffer D. Youth Suicide Risk and Preventive Interventions: A Review of the Past 10 Years. J Am Acad Child Adolesc Psychiatry 2003; 42 (4):386-405.

Green BL, Goodman LA, Krupnick JL, Corcoran CB, Petty RM, Stockton P, Stern NM. Outcomes of single versus multiple trauma exposure in a screening sample. J Trauma Stress 2000; 13 (2):271-86.

Gregurek R, Pavić L, Vuger-Kovacić H, Potrebica S, Bitar Z, Kovacić D, Danić S, Klain E. Increase of frequency of post-traumatic stress disorder in disabled war veterans during prolonged stay in a rehabilitation hospital. Croat Med J 2001; 42 (2):161-4.

Grinker R, Spiegel J. Men under stress. New York: McGraw-Hill; 1945.

Grossman P, Niemann L, Schmidt S, Walach H. Mindfulness-based stress reduction and health benefits. A meta-analysis. J Psychosom Res 2004;57:35-43.

Hahnemann, S (1996). Organon of Medicine. Cooper Publishing: Blaine, Washington.

Halford WK, Harrison C, Kalyansundaram et al. Preliminary results from a psychoeducational program to rehabilitate chronic patients. Psychiatr Serv 1995; 46 (11):1189-91.

Hamner MB, Brodrick PS, Labbate LA. Gabapentin in PTSD: a retrospective, clinical series of adjunctive therapy. Ann Clin Psychiatry 2001; 13 (3):141-6.

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 2003b;23:15-20.

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. International Clinical Psychopharmacology 2003a;18:1-8.

Harris JI, Erbes CR, Engdahl BE, Olson RH, Winskowski AM, McMahill J. Christian religious functioning and trauma outcomes. J Clin Psychol 2008;64:17-29.

Harvey AG, Bryant RA. Acute stress disorder after mild traumatic brain injury. J Nerv Ment Dis 1998a;186:333-7.

Harvey AG, Bryant RA. Predictors of acute stress following mild traumatic brain injury. Brain Inj 1998b;12:147-54.

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 (6):985-8.

Harvey AG, Bryant RA. Two-year prospective evaluation of the relationship between acute stress disorder and posttraumatic stress disorder following mild traumatic brain injury. Am J Psychiatry 2000; 157 (4):626-8.

Hassett AL, Radvanski DC, Vaschillo EG, Vaschillo B, Sigal LH, Karavidas MK, Buyske S, Lehrer PM. A pilot study of the efficacy of heart rate variability (HRV) biofeedback in patients with fibromyalgia. Appl Psychophysiol Biofeedback 2007;32:1-10.

Hawton K, Townsend E, Arensman E et al. Psychosocial versus pharmacological treatments for deliberate self harm. Cochrane Database Syst Rev 2000 (2):CD001764.

Hebert MA, Potegal M, Moore T, Evenson AR, Meyerhoff JL. Diazepam enhances conditioned defeat in hamsters (Mesocricetus auratus). Pharmacol Biochem Behav 1996;55:405-13.

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;317:1630-4.

Hembree EA, Foa EB. Posttraumatic stress disorder: psychological factors and psychosocial interventions. J Clin Psychiatry 2000; 61 Suppl 7:33-9.

Hendin H, Haas AP. Suicide and guilt as manifestations of PTSD in Vietnam ombat veterans. Am J Psychiatry 1991; 148 (5):586-91.

Heresco-Levy U, Kremer I, Javitt DC, Goichman R, Reshef A, Blanaru M, Cohen T. Pilot-controlled trial of D-cycloserine for the treatment of post-traumatic stress disorder. International Journal of Neuropsychopharmacology 2002;5:301-7.

Hertzberg MA, Butterfield MI, Feldman ME et al. A preliminary study of lamotrigine for the treatment of posttraumatic stress disorder. Biol Psychiatry 1999; 45 (9):1226-9.

Hertzberg MA, Feldman ME, Beckham JC et al. Open trial of nefazodone for combat-related posttraumatic stress disorder. J Clin Psychiatry 1998; 59 (9):460-4.

Hickling EJ, Blanchard EB. Post-traumatic stress disorder and motor vehicle accidents. J Anxiety Disord 1992;6:285–91.

Hickling EJ, Blanchard EB. The private practice psychologist and manual-based treatments: post-traumatic stress disorder secondary to motor vehicle accidents. Behav Res Ther 1997; 35 (3):191-203.

Hidalgo R, Hertzberg MA, Mellman T et al. Nefazodone in post-traumatic stress disorder: results from six open-label trials. Int Clin Psychopharmacol 1999; 14 (2):61-8.

Hirai M, Clum GA. An Internet-based self-change program for traumatic event related fear, distress, and maladaptive coping. J Trauma Stress 2005;18:631-6.

Hobbs M, Mayou R, Harrison B et al. A randomised controlled trial of psychological debriefing for victims of road traffic accidents. Bmj 1996; 313 (7070):1438-9.

Hoge CW, Auchterlonie JL, Milliken CS. Mental health problems, use of mental health services, and attrition from military service after returning from deployment to Iraq or Afghanistan. JAMA 2006;295:1023-32.

Hoge CW, Castro CA, Messer SC, McGurk D, Cotting DI, Koffman RL. Combat duty in Iraq and Afghanistan, mental health problems, and barriers to care. N Engl J Med 2004;351:13-22.

Hoge CW, Goldberg HM, Castro CA. Care of war veterans with mild traumatic brain injury--flawed perspectives. N Engl J Med. 2009;360(16):1588-91.

Hoge CW, Terhakopian A, Castro CA, Messer SC, Engel CC. Association of posttraumatic stress disorder with somatic symptoms, health care visits, and absenteeism among Iraq war veterans. Am J Psychiatry 2007;164:150-3.

Holbrook TL, Galarneau MR, Dye JL, Quinn K, Dougherty AL. Morphine use after combat injury in Iraq and post-traumatic stress disorder. N Engl J Med 2010;362:110-7.

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;51:287-92; discussion 92-3.

Hollifield M, Sinclair-Lian N, Warner TD, Hammerschlag R. Acupuncture for posttraumatic stress disorder: a randomized controlled pilot trial. J Nerv Ment Dis 2007;195:504-13.

Horowitz MJ. Stress response syndromes. 3rd ed. Northvale, NJ: Aronson; 1997.

Hutton HE, Treisman GJ, Hunt WR et al. HIV risk behaviors and their relationship to posttraumatic stress disorder among women prisoners. Psychiatr Serv 2001; 52 (4):508-13.

IOM (2007) - Institute of Medicine (IOM). Treatment of posttraumatic stress disorder: An assessment of the evidence. Washington, DC: The National Academies Press; 2008.

Ipser J, Seedat S, Stein DJ. Pharmacotherapy for post-traumatic stress disorder - a systematic review and meta-analysis. S Afr Med J 2006;96:1088-96.

Ironson G, Freund B, Strauss JL et al. Comparison of two treatments for traumatic stress: a community-based study of EMDR and prolonged exposure. J Clin Psychol 2002; 58 (1):113-28.

ISTSS (2009) - Effective Treatments for PTSD: Practice Guidelines from the International Society for Traumatic Stress Studies. Foa EB, Keane TM, Friedman MJ. Cohen J (Eds) 2009. Second Edition NY and London, Guilford Press:2009.

Iversen AC, Fear NT, Ehlers A, Hacker Hughes J, Hull L, Earnshaw M, Greenberg N, Rona R, Wessely S, Hotopf M. Risk factors for post-traumatic stress disorder among UK Armed Forces personnel. Psychol Med 2008;38:511-22.

Jacobsen LK, Southwick SM, Kosten TR. Substance use disorders in patients with posttraumatic stress disorder: a review of the literature. Am J Psychiatry 2001;158:1184-90.

Jakupcak M, Conybeare D, Phelps L, Hunt S, Holmes HA, Felker B, Klevens M, McFall ME. Anger, hostility, and aggression among Iraq and Afghanistan War veterans reporting PTSD and subthreshold PTSD. J Trauma Stress 2007;20:945-54.

Jakupcak M, Cook J, Imel Z, Fontana A, Rosenheck R, McFall M. Posttraumatic stress disorder as a risk factor for suicidal ideation in Iraq and Afghanistan War veterans. J Trauma Stress 2009;22:303-6.

Jensen JA. An investigation of eye movement desensitization and reprocessing (EMD/R) as a treatment for posttraumatic stress disorder (PTSD) symptoms in Vietnam combat veterans. Behavior Therapy.1994:25;311-25.

Jin P. Changes in heart rate, noradrenaline, cortisol and mood during Tai Chi. J Psychosom Res 1989;33:197-206.

Jiranek D. Use of hypnosis in pain management in post-traumatic stress disorder. Australian J Clinical and Experimental Hypnosis 1993; 21 (1):75-84.

Johnson M, Ostlund S, Fransson G, Kadesjo B, Gillberg C. Omega-3/omega-6 fatty acids for attention deficit hyperactivity disorder: a randomized placebo-controlled trial in children and adolescents. J Atten Disord 2009;12:394-401.

Kabat-Zinn J, Lipworth L, Burney R. The clinical use of mindfulness meditation for the self-regulation of chronic pain. J Behav Med 1985;8:163-90.

Kalra R,. Clark ME, Scholten J, Murphy JL, Clements KL. Managing pain among returning service members. Federal Practitioner 2008;25: 36–45.

Kardiner A. The traumatic neuroses of war. New York: Paul B. Hoeber; 1941.

Kaslow N, Thompson M, Meadows L et al. Risk factors for suicide attempts among African American women. Depress Anxiety 2000; 12 (1):13-20.

Kavanaugh DJ, Freese S, Andrade J et al. Effects of visuospatial tasks on desensitization to emotive memories. British Journal of Clinical Psychology 2001; 40:267-80.

Keane TM, Fairbank JA, Caddell JM et al. Implosive (flooding) therapy reduces symptoms of PTSD in Vietnam combat veterans. Behav Ther 1989; 20:245-60.

Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. Arch Gen Psychiatry 2005;62:593-602.

Kessler RC, Chiu WT, Demler O, Walters EE. Prevalence, severity, and comorbidity of twelve-month DSM-IV disorders in the National Comorbidity Survey Replication (NCS-R). Archives of General Psychiatry, 2005;62(6):617-27.

Kessler RC, Sonnega A, Bromet E et al. Posttraumatic stress disorder in the National Comorbidity Survey. Arch Gen Psychiatry 1995; 52 (12):1048-60.

Kilpatrick D, Tidwell R. Victims' rights and services in South Carolina: the dream, the law, the reality. Charleston, SC: Crime Victims Research and Treatment Center, Medical University of South Carolina; 1989.

Kilpatrick DG, Veronen LJ, Resick PA. Psychological sequelae to rape: assessment and treatment strategies. In: DM Dolays; RL Meredith, editors, translator and editor Behavioral medicine: assessment and treatment strategies. New York: Plenum Press; 1982; p. 473-97.

King DW, King LA, Foy DW et al. Posttraumatic stress disorder in a national sample of female and male Vietnam veterans: risk factors, war-zone stressors, and resilience-recovery variables. J Abnorm Psychol 1999; 108 (1):164-70.

King LA, King DW, Fairbank JA et al. Resilience-recovery factors in post-traumatic stress disorder among female and male Vietnam veterans: hardiness, postwar social support, and additional stressful life events. J Pers Soc Psychol 1998; 74 (2):420-34.

Kingsbury SJ. Brief hypnotic treatment of repetitive nightmares. Am J Clin Hypn 1993; 35 (3):161-9.

Kinzie JD, Leung P. Clonidine in Cambodian patients with posttraumatic stress disorder. J Nerv Ment Dis 1989; 177 (9):546-50.

Kirkwood G, Rampes H, Tuffrey V, Richardson J, Pilkington K. Yoga for anxiety: a systematic review of the research evidence. Br J Sports Med 2005;39:884-91.

Kirsch I, Capafons A, Cardena E et al. Clinical hypnosis and self-regulation therapy: an introduction. In: A Kirsc; E Capafons; E Cardena et al., editors, translator and editor Clinical hynosis and self-regulation therapy: a cognitive-behavioral perspective. Washington, DC: American Psychological Association; 1999.

Kirsch I, Lynn SJ. Dissociation theories of hypnosis. Psychol Bull 1998;123:100-15.

Kirsch I, Montgomery G, Sapirstein G. Hypnosis as an adjunct to cognitive-behavioral psychotherapy: a meta-analysis. J Consult Clin Psychol 1995; 63 (2):214-20.

Kirsch I. APA definition and description of hypnosis: defining hypnosis for the public. Contemporary Hypnosis 1994; 11:142-43.

Kirsch I. Hypnotic enhancement of cognitive-behavioral weight loss treatments--another meta-reanalysis. J Consult Clin Psychol 1996; 64 (3):517-9.

Knaevelsrud C, Maercker A. Internet-based treatment for PTSD reduces distress and facilitates the development of a strong therapeutic alliance: a randomized controlled clinical trial. BMC Psychiatry 2007;7:13.

Kobasa SC, Maddi, SR, & Kahn S. Hardiness and health : A prospective study. Journal of Personality and Social Psychology. 1982; 42, 168-177.

Koenen KC, De Vivo I, Rich-Edwards J, Smoller JW, Wright RJ, Purcell SM. Protocol for investigating genetic determinants of posttraumatic stress disorder in women from the Nurses' Health Study II. BMC Psychiatry 2009;9:29.

Koenen KC, Fu QJ, Ertel K, Lyons MJ, Eisen SA, True WR, Goldberg J, Tsuang MT. Common genetic liability to major depression and posttraumatic stress disorder in men. J Affect Disord 2008;105:109-15.

Koerner K, Linehan MM. Research on dialectical behavior therapy for patients with borderline personality disorder. Psychiatr Clin North Am 2000; 23 (1):151-67.

Koopman C, Classen C, Spiegel D. Predictors of posttraumatic stress symptoms among survivors of the Oakland/Berkeley, Calif., firestorm. Am J Psychiatry 1994; 151 (6):888-94.

Kornor H, Winje D, Ekeberg O, Weisaeth L, Kirkehei I, Johansen K, Steiro A. Early trauma-focused cognitive-behavioural therapy to prevent chronic post-traumatic stress disorder and related symptoms: A systematic review and meta-analysis. BMC Psychiatry 2008;8.

Kosten TR, Fontana A, Sernyak MJ, Rosenheck R. Benzodiazepine use in posttraumatic stress disorder among veterans with substance abuse. J Nerv Ment Dis 2000;188:454-9.

Kosten TR, Frank JB, Dan E et al. Pharmacotherapy for posttraumatic stress disorder using phenelzine or imipramine. J Nerv Ment Dis 1991; 179 (6):366-70.

Kosten TR, Krystal J. Biological mechanisms in posttraumatic stress disorder: relevance for substance abuse. In: M Galanter, editor, translator and editor Recent developments in alcoholism. Vol. 6. New York; 1988.

Kotler M, Iancu I, Efroni R, Amir M. Anger, impulsivity, social support, and suicide risk in patients with posttraumatic stress disorder. J Nerv Ment Dis 2001;189:162-7.

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 2001a;286:537-45.

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.

Krakow B, Johnston L, Melendrez D et al. An open-label trial of evidence-based cognitive behavior therapy for nightmares and insomnia in crime victims with PTSD. Am J Psychiatry 2001; 158 (12):2043-7.

Krakow B, Kellner R, Pathak D et al. Imagery rehearsal treatment for chronic nightmares. Behav Res Ther 1995; 33 (7):837-43.

Krakow B, Sandoval D, Schrader R et al. Treatment of chronic nightmares in adjudicated adolescent girls in a residential facility. J Adolesc Health 2001b; 29 (2):94-100.

Kramer TL, Lindy JD, Green BL et al. The comorbidity of post-traumatic stress disorder and suicidality in Vietnam veterans. Suicide Life Threat Behav 1994; 24 (1):58-67.

Kremen WS, Koenen KC, Boake C, Purcell S, Eisen SA, Franz CE, Tsuang MT, Lyons MJ. Pretrauma cognitive ability and risk for posttraumatic stress disorder: a twin study. Arch Gen Psychiatry 2007;64:361-8.

Krupnick JL, Green BL, Stockton P, Miranda J, Krause E, Mete M. Group interpersonal psychotherapy for low-income women with posttraumatic stress disorder. Psychother Res 2008;18:497-507.

Krystal JH, Rosenheck RA, Vessicchio JC, et al. Adjunctive risperidone treatment for antidepressant-resistant symptoms of chronic military service-related PTSD. JAMA 2011;306(5):493-502.

Kuiken D, Bears M, Miall D et al. Eye movement desensitization reprocessing facilitates attentional orienting. Imagination, Cognition, and Personality 2001-2002; 21 (1):3-20.

Kulka, R. A., Schlenger, W. E., Fairbank, J. A., Hough, R. L., Jordan, B. K., Marmar, C. R., et al. (1990). The National Vietnam Veterans Readjustment Study: Tables of findings and technical appendices. New York: Brunner/Mazel.

Laffaye C, Rosen CS, Schnurr PP, Friedman MJ. Does compensation status influence treatment participation and course of recovery from post-traumatic stress disorder? Mil Med 2007;172:1039-45.

Lagomasino I, Daly R, Stoudemire A. Medical assessment of patients presenting with psychiatric symptoms in the emergency setting. Psychiatr Clin North Am 1999; 22 (4):819-50, viii-ix.

Lang AJ, Aarons GA, Gearity J, Laffaye C, Satz L, Dresselhaus TR, Stein MB. Direct and indirect links between childhood maltreatment, posttraumatic stress disorder, and women's health. Behav Med 2008;33:125-35.

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.

Lange A, van de Ven J-P, Schrieken B. Interapy: Treatment of post-traumatic stress via the Internet. Cognitive Behaviour Therapy 2003;32:110-24.

Latimer EA, Lecomte T, Becker DR, Drake RE, Duclos I, Piat M, Lahaie N, St-Pierre MS, Therrien C, Xie H. Generalisability of the individual placement and support model of supported employment: results of a Canadian randomised controlled trial. Br J Psychiatry 2006;189:65-73.

Lee C, Gavriel H, Drummond P et al. Treatment of post-traumatic stress disorder: A comparison of stress inoculation training with prolonged exposure and eye movement desensitisation and reprocessing. Journal of Clinical Psychology 2002; 58:1071-89.

Lee E, editor Working with Asian Americans: a guide for clinicians New York: Guilford Press; 1997

Lee MS, Pittler MH, Ernst E. Effects of reiki in clinical practice: a systematic review of randomised clinical trials. Int J Clin Pract 2008;62:947-54.

Lehman AF. Vocational rehabilitation in schizophrenia. Schizophr Bull 1995; 21 (4):645-56.

Leskin GA, Westrup D. PTSD brief screen. Post traumatic stress disorder implications for primary care 1999.Department of Defense/EES, March 2002.

Levitt EE. Hypnosis in the treatment of obesity. In: SJ Lynn; JW Thue; I Kirsch, editors, translator and editor Handbook of clinical hypnosis. Washington D. C.: American Psychological Association; 1994; p. 533-53.

Lew HL, Otis JD, Tun C, Kerns RD, Clark ME, Cifu DX. Prevalence of chronic pain, posttraumatic stress disorder, and persistent postconcussive symptoms in OIF/OEF veterans: polytrauma clinical triad. J Rehabil Res Dev 2009;46:697-702.

Lin PY, Su KP. A meta-analytic review of double-blind, placebo-controlled trials of antidepressant efficacy of omega-3 fatty acids. J Clin Psychiatry 2007;68:1056-61.

Lindauer RJ, Gersons BP, van Meijel EP, Blom K, Carlier IV, Vrijlandt I, Olff M. Effects of brief eclectic psychotherapy in patients with posttraumatic stress disorder: randomized clinical trial. J Trauma Stress 2005;18:205-12.

Linde K, Berner MM, Kriston L. St John's wort for major depression. Cochrane Database Syst Rev 2008:CD000448.

Linde K, Jonas WB, Melchart D, Willich S (June 2001), "The methodological quality of randomized controlled trials of homeopathy, herbal medicines and acupuncture", Int J Epidemiol 30 (3): 526–31,

Lindley SE, Carlson EB, Hill K. A randomized, double-blind, placebo-controlled trial of augmentation topiramate for chronic combat-related posttraumatic stress disorder. J Clin Psychopharmacol 2007;27:677-81.

Lindy J. Psychoanalytic psychotherapy of post-traumatic stress disorder. In: B van der Kolk; AC McFarlane; L Weisaeth, editors, translator and editor Traumatic stress: the effects of overwhelming experiences on mind, body and society. New York: Guilford Press; 1996; p. 525-36.

Linehan MM, Heard HL, Armstrong HE. Naturalistic follow-up of a behavioral treatment for chronically parasuicidal borderline patients. Arch Gen Psychiatry 1993; 50 (12):971-4.

Litz B, Engel C, Bryant R, Papa A. A Randomized, Controlled Proof-of-Concept Trial of an Internet-Based, Therapist-Assisted Self-Management Treatment for Posttraumatic Stress Disorder. American Journal of Psychiatry 2007;164:1676-83.

Litz B. Bryant R. Early Cognitive-Behavioral Interventions for Adults. Chapter In EB Foa, TM Keane, MJ Friedman (Eds.), Effective treatments for PTSD: Practice Guidelines from the International Society for Traumatic Stress Studies. Second Edition NY and London, Guilford Press:2009.

Litz, B., Gray, M, Bryant, R. and Adler, A. Early Intervention for Trauma: Current Status and Future Directions: Clin. Psychol., May 1, 2002; 9(2): 112 -134.

Londborg PD, Hegel MT, Goldstein S et al. Sertraline treatment of posttraumatic stress disorder: results of 24 weeks of open-label continuation treatment. J Clin Psychiatry 2001; 62 (5):325-31.

Lovell K, Marks IM, Noshirvani H et al. Do cognitive and exposure treatments improve various PTSD symptoms differently? A randomized controlled trial. Behav and Cognitive Psychotherapy 2001; 29 (1):107-12.

Lubin H, Loris M, Burt J et al. Efficacy of Psychoeducational Group Therapy in reducing symptoms of posttraumatic stress disorder among multiply traumatized women. Am J Psychiatry 1998; 155 (9):1172-7.

Lysaker P, Bell M, Milstein R et al. Work capacity in schizophrenia. Hosp Community Psychiatry 1993; 44 (3):278-80.

MacGregor AJ, Corson KS, Larson GE, Shaffer RA, Dougherty AL, Galarneau MR, Raman R, Baker DG, Lindsay SP, Golomb BA. Injury-specific predictors of posttraumatic stress disorder. Injury 2009;40:1004-10.

Maguen, Shira; Lucenko, Barbara A; Reger, Mark A; Gahm, Gregory A; Litz, Brett T; Seal, Karen H; Knight, Sara J; Marmar, Charles R. The impact of reported direct and indirect killing on mental health symptoms in Iraq war veterans. Journal of Traumatic Stress 2010;23:1:86-90.

Manger TA, Motta RW. The impact of an exercise program on posttraumatic stress disorder, anxiety, and depression. Int J Emerg Ment Health 2005;7:49-57.

Mares AS, Kasprow WJ, Rosenheck RA. Outcomes of supported housing for homeless veterans with psychiatric and substance abuse problems. Ment Health Serv Res 2004;6:199-211.

Margoob MA, Ali Z, Andrade C. Efficacy of ECT in chronic, severe, antidepressant- and CBT-refractory PTSD: an open, prospective study. Brain Stimul 2010;3:28-35.

Marks I, Lovell K, Noshirvani H et al. Treatment of posttraumatic stress disorder by exposure and/or cognitive restructuring: a controlled study. Arch Gen Psychiatry 1998; 55 (4):317-25.

Marmar CR, Weiss DS, Schlenger WE et al. Peritraumatic dissociation and posttraumatic stress in male Vietnam theater veterans. Am J Psychiatry 1994; 151 (6):902-7.

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;158:1982-8.

Marshall RD, Lewis-Fernandez R, Blanco C, Simpson HB, Lin SH, Vermes D, Garcia W, Schneier F, Neria Y, Sanchez-Lacay A, Liebowitz MR. A controlled trial of paroxetine for chronic PTSD, dissociation, and interpersonal problems in mostly minority adults. Depress Anxiety 2007;24:77-84.

Marshall RD, Olfson M, Hellman F, Blanco C, Guardino M, Struening EL. Comorbidity, impairment, and suicidality in subthreshold PTSD. Am J Psychiatry 2001;158:1467-73.

Martenyi F, Brown EB, Caldwell CD. Failed efficacy of fluoxetine in the treatment of posttraumatic stress disorder: results of a fixed-dose, placebo-controlled study. J Clin Psychopharmacol 2007;27:166-70.

Martenyi F, Brown EB, Zhang H, Koke SC, Prakash A. Fluoxetine v. placebo in prevention of relapse in post-traumatic stress disorder. British Journal of Psychiatry 2002;181:315-20.

Martenyi F, Brown EB, Zhang H, Prakash A, Koke SC. Fluoxetine versus placebo in posttraumatic stress disorder. J Clin Psychiatry 2002;63:199-206.

Matar MA, Zohar J, Kaplan Z, Cohen H. Alprazolam treatment immediately after stress exposure interferes with the normal HPA-stress response and increases vulnerability to subsequent stress in an animal model of PTSD. Eur Neuropsychopharmacol 2009;19:283-95.

Maxfield L, Hyer L. The relationship between efficacy and methodology in studies investigating EMDR treatment of PTSD. J Clin Psychol 2002; 58 (1):23-41.

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; 176:589-93.

McCraty R, Atkinson M, Tomasino D, Stuppy WP. Analysis of twenty-four hour heart rate variability in patients with panic disorder. Biol Psychol 2001;56:131-50.

McDonagh A, Friedman M, McHugo G, Ford J, Sengupta A, Mueser K, Demment CC, Fournier D, Schnurr PP, Descamps M. Randomized trial of cognitive-behavioral therapy for chronic posttraumatic stress disorder in adult female survivors of childhood sexual abuse. J Consult Clin Psychol 2005;73:515-24.

McFall M, Atkins DC, Yoshimoto D, Thompson CE, Kanter E, Malte CA, Saxon AJ. Integrating tobacco cessation treatment into mental health care for patients with posttraumatic stress disorder. Am J Addict 2006;15:336-44.

McFall M, Saxon AJ, Thaneemit-Chen S, Smith MW, Joseph AM, Carmody TP, Beckham JC, Malte CA, Vertrees JE, Boardman KD, Lavori PW. Integrating smoking cessation into mental health care for post-traumatic stress disorder. Clin Trials 2007;4:178-89.

McFall M, Saxon AJ, Thompson CE, Yoshimoto D, Malte C, Straits-Troster K, Kanter E, Zhou XH, Dougherty CM, Steele B. Improving the rates of quitting smoking for veterans with posttraumatic stress disorder. Am J Psychiatry 2005;162:1311-9.

McFall ME, Wright PW, Donovan DM, Raskind M. Multidimensional assessment of anger in Vietnam veterans with posttraumatic stress disorder. Compr Psychiatry 1999;40:216-20.

McGhee LL, Maani CV, Garza TH, Desocio PA, Gaylord KM, Black IH.The Effect of Propranolol on Posttraumatic Stress Disorder in Burned Service Members. J Burn Care Res. 2008 Dec 3.

McHorney CA, Kosinski M, Ware JE, Jr. Comparisons of the costs and quality of norms for the SF-36 health survey collected by mail versus telephone interview: results from a national survey. Med Care 1994; 32 (6):551-67.

McHorney CA, Ware JE, Jr., Lu JF et al. The MOS 36-item Short-Form Health Survey (SF-36): III. Tests of data quality, scaling assumptions, and reliability across diverse patient groups. Med Care 1994; 32 (1):40-66.

McKay C, Johnsen M, Stein R. Employment outcomes in Massachusetts Clubhouses. Psychiatr Rehabil J 2005;29:25-33.

McLean SA, Clauw DJ, Abelson JL, Liberzon I. The development of persistent pain and psychological morbidity after motor vehicle collision : integrating the potential role of stress response systems into a biopsychosocial model. Psychosomatic Medicine.2005; 67:783–790.

McRae AL, Brady KT, Mellman TA, Sonne SC, Killeen TK, Timmerman MA, Bayles-Dazet W. Comparison of nefazodone and sertraline for the treatment of posttraumatic stress disorder. Depress Anxiety 2004;19:190-6.

Meisenhelder JB. Marcum, JP. Responses of Clergy to 9/11: Posttraumatic Stress, Coping, and Religious Outcomes. Journal for the Scientific Study of Religion.2004; 4: 547-554.

Mellman TA, Bustamante V, David D, Fins AI. Hypnotic medication in the aftermath of trauma. J Clin Psychiatry 2002;63:1183-4.

Mellman TA, Byers PM, Augenstein JS. Pilot evaluation of hypnotic medication during acute traumatic stress response. J Trauma Stress 1998;11:563-9.

Mellman TA, David D, Bustamante V et al. Predictors of post-traumatic stress disorder following severe injury. Depress Anxiety 2001; 14 (4):226-31.

Meltzer-Brody S, Connor KM, Churchill E et al. Symptom-specific effects of fluoxetine in post-traumatic stress disorder. Int Clin Psychopharmacol 2000; 15 (4):227-31.

Mental Health Advisory Team (MHAT) 6 Operation Enduring Freedom 2009. Afghanistan 6 November 2009 Office of the Command Surgeon US Forces Afghanistan (USFOR-A) and Office of The Surgeon General. United States Army Medical Command. Available at: www.armymedicine.army.mil

Mental Health Advisory Team (MHAT) VI. Operation Iraqi Freedom 07-09 8 May 2009 Office of the Surgeon Multi-National Corps-Iraq and. Office of The Surgeon General. United States Army Medical Command

Miller AL, Glinski J. Youth suicidal behavior: assessment and intervention. J Clin Psychol 2000; 56 (9):1131-52.

Miller MW, Wolf EJ, Martin E, Kaloupek DG, Keane TM. Structural equation modeling of associations among combat exposure, PTSD symptom factors, and Global Assessment of Functioning. J Rehabil Res Dev 2008;45:359-69.

Milliken CS, Auchterlonie JL, Hoge CW. Longitudinal assessment of mental health problems among active and reserve component soldiers returning from the Iraq war. JAMA 2007;298:2141-8.

Mischoulon D, Papakostas GI, Dording CM, Farabaugh AH, Sonawalla SB, Agoston AM, Smith J, Beaumont EC, Dahan LE, Alpert JE, Nierenberg AA, Fava M. A double-blind, randomized controlled trial of ethyl-eicosapentaenoate for major depressive disorder. J Clin Psychiatry 2009;70:1636-44.

Monnelly EP, Ciraulo DA, Knapp C, Keane T. Low-dose risperidone as adjunctive therapy for irritable aggression in posttraumatic stress disorder. J Clin Psychopharmacol 2003;23:193-6.

Monson CM, Price JL, Rodriguez BF, Ripley MP, Warner RA. Emotional deficits in military-related PTSD: an investigation of content and process disturbances. J Trauma Stress 2004;17:275-9.

Monson CM, Rodriguez BF, Warner R. Cognitive-behavioral therapy for PTSD in the real world: do interpersonal relationships make a real difference? J Clin Psychol 2005;61:751-61.

Monson CM, Schnurr PP, Resick PA, Friedman MJ, Young-Xu Y, Stevens SP. Cognitive processing therapy for veterans with military-related posttraumatic stress disorder. J Consult Clin Psychol 2006;74:898-907.

Morin CM, Hauri PJ, Espie CA, Spielman AJ, Buysse DJ, Bootzin RR. Nonpharmacologic treatment of chronic insomnia. An American Academy of Sleep Medicine review. Sleep 1999;22:1134-56.

Morland LA, Greene CJ, Rosen C, Mauldin PD, Frueh BC. Issues in the design of a randomized noninferiority clinical trial of telemental health psychotherapy for rural combat veterans with PTSD. Contemp Clin Trials 2009;30:513-22.

Morland LA, Greene CJ, Rosen CS, Foy D, Reilly P, Shore J, He Q, Frueh BC. Telemedicine for anger management therapy in a rural population of combat veterans with posttraumatic stress disorder: a randomized noninferiority trial. J Clin Psychiatry. 2010 Jul;71(7):855-63.

Morland LA, Pierce K, Wong MY. Telemedicine and coping skills groups for Pacific Island veterans with post-traumatic stress disorder: a pilot study. Telemed Telecare. 2004;10(5):286-9.

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;66:493-9.

Murray J, Ehlers A, Mayou RA. Dissociation and post-traumatic stress disorder: two prospective studies of road traffic accident survivors. Br J Psychiatry 2002; 180:363-8.

Muse M. Stress-related, posttraumatic chronic pain syndrome: Behavioral treatment approach. Pain 1986;25:389-94.

Najavits LM, Weiss RD, Shaw SR, Muenz LR. "Seeking safety": outcome of a new cognitive-behavioral psychotherapy for women with posttraumatic stress disorder and substance dependence. J Trauma Stress 1998;11:437-56.

Najavits LM, Weiss RD, Shaw SR. The link between substance abuse and posttraumatic stress disorder in women. A research review. Am J Addict 1997;6:273-83.

Najavits LM. Seeking safety: a manual for PTSD and substance abuse. New York: The Guilford Press; 2002.

Neria Y, Bromet EJ, Sievers S et al. Trauma exposure and posttraumatic stress disorder in psychosis: findings from a first-admission cohort. J Consult Clin Psychol 2002; 70 (1):246-51.

Neuner F, Onyut PL, Ertl V, Odenwald M, Schauer E, Elbert T. Treatment of posttraumatic stress disorder by trained lay counselors in an African refugee settlement: a randomized controlled trial. J Consult Clin Psychol 2008;76:686-94.

Neuner F, Schauer M, Klaschik C, Karunakara U, Elbert T. A comparison of narrative exposure therapy, supportive counseling, and psychoeducation for treating posttraumatic stress disorder in an african refugee settlement. J Consult Clin Psychol 2004;72:579-87.

Neylan T, Lenoci M, Samuelson K, Metzler T, Henn-Haase C, Hierholzer R, Lindley S, Otte C, Schoenfeld F, Yesavage J, Marmar C. No improvement of posttraumatic stress disorder symptoms with guanfacine treatment. American Journal of Psychiatry 2006;163:2186-8.

Neylan TC, Metzler TJ, Schoenfeld FB et al. Fluvoxamine and sleep disturbances in posttraumatic stress disorder. J Trauma Stress 2001; 14 (3):461-7.

NICE, 2005 - National Collaborating Centre for Mental Health. Post-traumatic stress disorder: the management of PTSD in adults and children in primary and secondary care. London (UK): National Institute for Clinical Excellence (NICE); 2005

NIMH, 2002 - National Institutes of Mental Health brochure "Post-Traumatic Stress Disorder: a Real Illness"Available at: http://www.nimh nih.gov/anxiety/ptsdri2.cfm, June 14, 2000.

Norman SB, Stein MB, Dimsdale JE, Hoyt DB. Pain in the aftermath of trauma is a risk factor for post-traumatic stress disorder. Psychol Med 2008;38:533-542.

Norris FH, Friedman MJ, Watson PJ, Byrne CM, Diaz E, Kaniasty K. 60,000 disaster victims speak: Part I. An empirical review of the empirical literature, 1981-2001. Psychiatry 2002;65:207-39.

Oldman J, Thomson L, Calsaferri K, Luke A, Bond GR. A case report of the conversion of sheltered employment to evidence-based supported employment in Canada. Psychiatr Serv 2005;56:1436-40.

Oquendo M, Brent DA, Birmaher B, et al. Post-traumatic stress disorder comorbid with major depression: factors mediating the association with suicidal behavior. Am J Psychiatry. 2005;162:560–566

O'Reilly R, Bishop J, Maddox K, Hutchinson L, Fisman M, Takhar J. Is telepsychiatry equivalent to face-to-face psychiatry? Results from a randomized controlled equivalence trial. Psychiatr Serv 2007;58:836-43.

Ortega AN, Rosenheck R. Posttraumatic stress disorder among Hispanic Vietnam veterans. Am J Psychiatry 2000; 157 (4):615-9.

Ospina MB, Bond K, Karkhaneh M, Tjosvold L, Vandermeer B, Liang Y, Bialy L, Hooton N, Buscemi N, Dryden DM, Klassen TP. Meditation practices for health: state of the research. Evid Rep Technol Assess (Full Rep) 2007:1-263. http://www.ahrq.gov/downloads/pub/evidence/pdf/meditation/medit.pdf

Osuch EA, Benson BE, Luckenbaugh DA, Geraci M, Post RM, McCann U. Repetitive TMS combined with exposure therapy for PTSD: A preliminary study. Journal of Anxiety Disorders 2009;23:54-9.

Otis JD, Keane TM, Kerns RD. An examination of the relationship between chronic pain and post-traumatic stress disorder. J Rehabil Res Dev 2003;40:397-405.

Ouimette P, Brown PJ. Trauma and Substance Abuse: Causes, Consequences, and Treatment of Comorbid Disorders. Washington, DC: American Psychological Association Press. 2002.

Ouimette PC, Brown PJ, Najavits LM. Course and treatment of patients with both substance use and posttraumatic stress disorders. Addict Behav 1998;23:785-95.

Ouimette PC, Moos RH, Finney JW. Two-year mental health service use and course of remission in patients with substance use and posttraumatic stress disorders. J Stud Alcohol 2000; 61 (2):247-53.

Ozer EJ, Best SR, Lipsey TL et al. Predictors of posttraumatic stress disorder and symptoms in adults: a meta-analysis. Psychological Bulletin 2003; 129 (1):52-73.

Padala PR, Madison J, Monnahan M, Marcil W, Price P, Ramaswamy S, Din AU, Wilson DR, Petty F. Risperidone monotherapy for post-traumatic stress disorder related to sexual assault and domestic abuse in women. International Clinical Psychopharmacology 2006;21:275-80.

Panasetis P, Bryant RA. Peritraumatic versus persistent dissociation in acute stress disorder. J Trauma Stress. 2003 Dec;16(6):563-6.

Paunovic N, Ost LG. Cognitive-behavior therapy vs exposure therapy in the treatment of PTSD in refugees. Behav Res Ther 2001; 39 (10):1183-97.

Pearlman LA, Saakvitne KW. Trauma and the therapist: countertransference and vicarious traumatization in psychotherapy with incest survivors. New York: Norton; 1995.

Pelletier JR, Nguyen M, Bradley K, Johnsen M, Mckay C. Integrating structured physical exercise into an ICCD certified clubhouse program: Results of a pilot study. Psychiatr Rehabil J 2005;29: 89-96.

Penk W, Flannery R. Psychosocial Rehabilitation. In EB Foa, TM Keane, MJ Friedman (Eds.), Effective treatments for PTSD: Practice Guidelines from the International Society for Traumatic Stress Studies. New York: Guilford Press; 2000

Penk WE, Ainspan ND. Community response to returning military. In SM Freeman, BA Moore, A Freeman (Editors). Living and surviving in harm's way: A psychological treatment handbook for pre- and post-deployment of military personnel. pp. 417-436. New York: Routledge; 2009

Penk WE, Robinowitz R, Black J et al. Ethnicity: post-traumatic stress disorder (PTSD) differences among black, white, and Hispanic veterans who differ in degrees of exposure to combat in Vietnam. J Clin Psychol 1989; 45 (5):729-35.

Petit L, Azad N, Byszewski A, Sarazan FF, Power B. Non-pharmacological management of primary and secondary insomnia among older people: review of assessment tools and treatments. Age Ageing. 2003 Jan;32(1):19-25.

Petty F, Brannan S, Casada J et al. Olanzapine treatment for post-traumatic stress disorder: an open-label study. Int Clin Psychopharmacol 2001; 16 (6):331-7.

Phillips CJ, Leardmann CA, Gumbs GR, Smith B. Risk factors for posttraumatic stress disorder among deployed US male marines. BMC Psychiatry 2010;10:52.

Pitman RK, Sanders KM, Zusman RM et al. Pilot study of secondary prevention of posttraumatic stress disorder with propranolol. Biol Psychiatry 2002; 51 (2):189-92.

Pittler MH, Ernst E. Kava extract for treating anxiety. Cochrane Database Syst Rev 2003:CD003383.

Power KG, McGoldrick T, Brown K et al. A controlled comparison of eye movement desensitization and reprocessing versus exposure plus cognitive restructuring, versus waiting list in the treatment of posttraumatic stress disorder. Journal of Clinical Psychology and Psychotherapy 2002; 9:299-318.

Prigerson HG, Slimack MJ. Gender differences in clinical correlates of suicidality among young adults. J Nerv Ment Dis 1999; 187 (1):23-31.

Prins A, Kimerling R, Cameron R et al. The primary care PTSD screen (PC-PTSD). In 15th Annual Meeting of the International Society for Tramatic Studies. Miami, FL; 1999.

Prins, A., Ouimette, P., Kimerling, R., Cameron, R. P., Hugelshofer, D. S., Shaw-Hegwer, J., Thrailkill, A., Gusman, F.D., Sheikh, JI. The primary care PTSD screen (PC–PTSD): development and operating characteristics. Primary Care Psychiatry 2003: *9;* 9-14.

Rand Corporation (2008) – see Tanielian et al., 2008.

Randall PK, Bremner JD, Krystal JH, Nagy LM, Heninger GR, Nicolaou AL, Charney DS. Effects of the benzodiazepine antagonist flumazenil in PTSD. Biol Psychiatry 1995;38:319-24.

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.

Raphael B, Newman L. Disaster Mental Health Response Handbook: An educational resource for mental health professionals involved in disaster management. Sydney: New South Wales Institute of Psychiatry and Centre for Mental Health. State Health Publication No: (CMH) 00145; 2000.

Raphael B. When disaster strikes: how individuals and communities cope with catastrophe. New York: Basic Books; 1986.

Raskind MA, Peskind ER, Hoff DJ, Hart KL, Holmes HA, Warren D, Shofer J, O'Connell J, Taylor F, Gross C, Rohde K, McFall ME. A Parallel Group Placebo Controlled Study of Prazosin for Trauma Nightmares and Sleep Disturbance in Combat Veterans with Post-Traumatic Stress Disorder. Biological Psychiatry 2007;61:928-34.

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;160:371-3.

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;63:565-8.

Rauch SA, Defever E, Favorite T, Duroe A, Garrity C, Martis B, Liberzon I. Prolonged exposure for PTSD in a Veterans Health Administration PTSD clinic. J Trauma Stress 2009;22:60-4.

Ready DJ, Thomas KR, Worley V, Backscheider AG, Harvey LA, Baltzell D, Rothbaum BO. A field test of group based exposure therapy with 102 veterans with war-related posttraumatic stress disorder. J Trauma Stress 2008;21:150-7.

Reger MA, Gahm GA. A meta-analysis of the effects of internet- and computer-based cognitive-behavioral treatments for anxiety. J Clin Psychol 2009;65:53-75.

Reich DB, Winternitz S, Hennen J, Watts T, Stanculescu C. A preliminary study of risperidone in the treatment of posttraumatic stress disorder related to childhood abuse in women. J Clin Psychiatry 2004;65:1601-6.

Reissig CJ, Strain EC, Griffiths RR. Caffeinated energy drinks--a growing problem. Drug Alcohol Depend 2009;99:1-10.

Reist C, Duffy JG, Fujimoto K, Cahill L. beta-Adrenergic blockade and emotional memory in PTSD. Int J Neuropsychopharmacol 2001;4:377-83.

Reist C, Kauffmann CD, Haier RJ et al. A controlled trial of desipramine in 18 men with posttraumatic stress disorder. Am J Psychiatry 1989; 146 (4):513-6.

Resick PA, Galovski TE, O'Brien Uhlmansiek M, Scher CD, Clum GA, Young-Xu Y. A randomized clinical trial to dismantle components of cognitive processing therapy for posttraumatic stress disorder in female victims of interpersonal violence. J Consult Clin Psychol 2008;76:243-58.

Resick PA, Nishith P, Weaver TL et al. 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 (4):867-79.

Resick PA, Nishith P. Two-year follow-up of a clinical trial comparing cognitive processing therapy and prolonged exposure for the treatment of PTSD. In, translator and editor Reaching undeserved trauma survivors through community-based programs: 17th Annual Meeting of the International Society for Traumatic Stress Studies; December 6-9, 2001; p. 54.

Resick PA, Schnicke MK. Cognitive processing therapy for sexual assault victims. J Consult Clin Psychol 1992;60:748-56.

Resnick H, Acierno R, Waldrop AE, King L, King D, Danielson C, Ruggiero KJ, Kilpatrick D. Randomized controlled evaluation of an early intervention to prevent post-rape psychopathology. Behaviour Research and Therapy 2007;45:2432-47.

Richards D. A field study of critical incident stress debriefing versus critical incident stress management. Journal of Mental Health 2001; 10 (3):351-62.

Risse SC, Whitters A, Burke J, et al: Severe withdrawal symptoms after discontinuation of alprazolam in eight patients with combatinduced posttraumatic stress disorder. Journal of Clinical Psychiatry 51:206–209, 1990

Rivers WHR. An address on the repression of war experience.Lancet, February 2, 1918.

Roberts NP, Kitchiner NJ, Kenardy J, Bisson J. Multiple session early psychological interventions for the prevention of post-traumatic stress disorder. Cochrane Database Syst Rev 2009(b)

Roberts NP, Kitchiner NJ, Kenardy J, Bisson JI. Systematic review and meta-analysis of multiple-session early interventions following traumatic events. Am J Psychiatry 2009(a);166:293-301.

Rog, DJ. The evidence on supported housing. Psychiatric Rehabilitation Journal. 2006 29, 334-344.

Rogers ES, Anthony W, Lyass A, Penk WE. A randomized clinical trial of vocational rehabilitation among persons with serious mental disorders. Rehabilitation Counseling Bulletin. 2006; 49: 143-156.

Rogers S, Silver SM, Goss J, Obenchain J, Willis A, Whitney RL. A single session, group study of exposure and Eye Movement Desensitization and Reprocessing in treating Posttraumatic Stress Disorder among Vietnam War veterans: preliminary data. J Anxiety Disord 1999;13:119-30.

Rollnick S, Heather N, Gold R, Hall W. Development of a short 'readiness to change' questionnaire for use in brief, opportunistic interventions among excessive drinkers. Br J Addict 1992; 87 (5):743-54.

Rose DA model for psychodynamic psychotherapy with the rape victim. Psychotherapy 1991; 28 (2):85-95.

Rose S, Bisson J, Wessely S. Psychological debriefing for preventing post traumatic stress disorder (PTSD) (Cochrane Review). The Cochrane Library 2002.

Rosen CS, DiLandro C, Corwin KN, Drescher KD, Cooney JH, Gusman F. Telephone monitoring and support for veterans with chronic posttraumatic stress disorder: a pilot study. Community Ment Health J. 2006 Oct;42(5):501-8.

Rosenberg PB, Mehndiratta RB, Mehndiratta YP, Wamer A, Rosse RB, Balish M. Repetitive transcranial magnetic stimulation treatment of comorbid posttraumatic stress disorder and major depression. J Neuropsychiatry Clin Neurosci 2002;14:270-6.

Rosenheck R, Fontana A. Race and outcome of treatment for veterans suffering from PTSD. J Trauma Stress 1996; 9 (2):343-51.

Roth S, Batson R. Naming the shadows: a new approach to individual and group psychotherapy for adult survivors of childhood incest. New York: Free Press; 1997.

Roth S, Friedman M. Childhood trauma remembered: a report on the scientific knowledge base and its applications: International Society for Traumatic Stress; 1997.

Rothbaum B. Psychosocial treatments for posttraumatic stress disorder. TEN 2001; 3 (10):59-63.

Rothbaum BO, Astin MC, Marsteller F. Prolonged Exposure versus Eye Movement Desensitization and Reprocessing (EMDR) for PTSD rape victims. J Trauma Stress 2005;18:607-16.

Rothbaum BO, Cahill SP, Foa EB, Davidson JR, Compton J, Connor KM, Astin MC, Hahn CG. Augmentation of sertraline with prolonged exposure in the treatment of posttraumatic stress disorder. J Trauma Stress 2006;19:625-38.

Rothbaum BO, Killeen TK, Davidson JR, Brady KT, Connor KM, Heekin MH. Placebo-controlled trial of risperidone augmentation for selective serotonin reuptake inhibitor-resistant civilian posttraumatic stress disorder. J Clin Psychiatry 2008;69:520-5.

Rothbaum BO, Meadows EA, Resick P et al. Chapter 4: Cognitive-Behavioral Therapy. In EB Foa, TM Keane, MJ Friedman (Eds.), Effective treatments for PTSD: Practice Guidelines from the International Society for Traumatic Stress Studies. (p.60-83);New York: The Guilford Press; 2000.

Rothbaum BO, Meadows EA, Resick P. Cognitive-behavioral treatment position paper summary for the ISTSS treatment guidelines committee. J Trauma Stress 2000b; 13:558-63.

Rotunda RJ, O'Farrell TJ, Murphy M, Babey SH. Behavioral couples therapy for comorbid substance use disorders and combat-related posttraumatic stress disorder among male veterans: an initial evaluation. Addict Behav 2008;33:180-7.

Ruff RL, Ruff SS, Wang XF. Headaches among Operation Iraqi Freedom/Operation Enduring Freedom veterans with mild traumatic brain injury associated with exposures to explosions. J Rehabil Res Dev 2008;45:941-52.

Ruff RL, Ruff SS, Wang XF. Improving sleep: initial headache treatment in OIF/OEF veterans with blast-induced mild traumatic brain injury. J REHABIL RES DEV 2009;46:1071-84.

S, Barber J, Thompson S. Manual-assisted cognitive-behaviour therapy (MACT): a randomized controlled trial of a brief intervention with bibliotherapy in the treatment of recurrent deliberate self-harm. Psychol Med 1999; 29 (1):19-25.

Safer DL, Telch CF, Agras WS. Dialectical behavior therapy for bulimia nervosa. Am J Psychiatry 2001; 158 (4):632-4.

Sanders SH, Harden RN, Vicente PJ. Evidence-based clinical practice guidelines for interdisciplinary rehabilitation of chronic nonmalignant pain syndrome patients. Pain Pract 2005;5:303-15.

Sarang P, Telles S. Effects of two yoga based relaxation techniques on heart rate variability (HRV). International Journal of Stress Management. 2006;13(4):460-475.

Sareen J, Cox BJ, Clara I, Asmundson GJ. The relationship between anxiety disorders and physical disorders in the U.S. National Comorbidity Survey. Depress Anxiety 2005;21:193-202.

Sayer NA, Chiros CE, Sigford B, Scott S, Clothier B, Pickett T, Lew HL. Characteristics and rehabilitation outcomes among patients with blast and other injuries sustained during the Global War on Terror. Arch Phys Med Rehabil 2008;89:163-70.

Saygin MZ, Sungur MZ, Sabol EU, Cetinkaya P. Nefazodone versus sertraline in treatment of posttraumatic stress disorder. Klinik Psikofarmakoloji Bulteni 2002;12:1-5.

Schelling G, Kilger E, Roozendaal B, de Quervain DJ, Briegel J, Dagge A, Rothenhausler HB, Krauseneck T, Nollert G, Kapfhammer HP. Stress doses of hydrocortisone, traumatic memories, and symptoms of posttraumatic stress disorder in patients after cardiac surgery: a randomized study. Biol Psychiatry 2004;55:627-33.

Schlenger WE, Kulka RA, Fairbank JA, Hough RL, Jordan BK, Marmar, CR. The prevalence of post-traumatic stress disorder in the Vietnam generation: A multimethod, multisource assessment of psychiatric disorder. Journal of Traumatic Stress 1992;5: 333-363.

Schnurr PP, Friedman MJ, Engel CC, Foa EB, Shea MT, Chow BK, Resick PA, Thurston V, Orsillo SM, Haug R, Turner C, Bernardy N. Cognitive behavioral therapy for posttraumatic stress disorder in women: a randomized controlled trial. Jama 2007;297:820-30.

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;60:481-9.

Schnurr PP, Green BL. Trauma and health: physical health consequences of exposure to extreme stress. Washington, DC: American Psychological Association, 2004.

Schnurr PP, Lunney CA, Sengupta A. Risk factors for the development versus maintenance of posttraumatic stress disorder. J Trauma Stress 2004;17:85-95.

Schnurr PP. Outcome of a randomized clinical trial of group therapy for PTSD. In, translator and editor Reaching undeserved trauma survivors through community-based programs: 17th Annual Meeting of the International Society for Traumatic Stress Studies; December 6-9, 2001; p. 61.

Scholes C, Turpin G, Mason S. A randomised controlled trial to assess the effectiveness of providing self-help information to people with symptoms of acute stress disorder following a traumatic injury. Behaviour Research and Therapy 2007;45:2527-36.

Schuster MA, Stein BD, Jaycox L et al. A national survey of stress reactions after the September 11, 2001, terrorist attacks. N Engl J Med 2001; 345 (20):1507-12.

Schutt RK, Garrett GR. Responding to the homeless: policy and practice. New York: Plenum; 1992.

Schutt RK., Rosenheck RE, Penk WE, Drebing CE, Seibyl CL. The social environment of transitional work and residences programs: Influences of health and functioning. Evaulation and Programs Planning 2005: 28; 291-300.

Schwartz AC, Bradley R, Penza KM, Sexton M, Jay D, Haggard PJ, Garlow SJ, Ressler KJ. Pain medication use among patients with posttraumatic stress disorder. Psychosomatics 2006;47:136-42.

Scott J, Williams J, Beck A. Cognitive therapy in clinical practice: an illustrative casebook. New York: Routledge; 1989.

Seal K. Bertenthal D. Miner CR. Sen S. Marmar C. Bringing the War Back Home: Mental Health Disorders Among 103 788 US Veterans Returning From Iraq and Afghanistan Seen at Department of Veterans Affairs Facilities. Arch Intern Med. 2007;167(5):476-482.

Seedat S, Stein DJ, Emsley RA. Open trial of citalopram in adults with post-traumatic stress disorder. Int J Neuropsychopharmacol 2000; 3 (2):135-40.

Seedat S, Stein DJ. Trauma and post-traumatic stress disorder in women: a review. Int Clin Psychopharmacol 2000; 15 Suppl 3:S25-33.

Seng JS. A conceptual framework for research on lifetime violence, posttraumatic stress, and childbearing. J Midwifery Womens Health 2002; 47 (5):337-46.

Servan-Schreiber D. Point: Eye movement desensitization and reprocessing: Is psychiatry missing the point? Psychiatric Times 2000; 17 (7).

Shakibaei F, Harandi AA, Gholamrezaei A, Samoei R, Salehi P. Hypnotherapy in management of pain and reexperiencing of trauma in burn patients. Int J Clin Exp Hypn 2008;56:185-97.

Shalev AY, Bloch M, Peri T et al. Alprazolam reduces response to loud tones in panic disorder but not in posttraumatic stress disorder. Biol Psychiatry 1998; 44 (1):64-8.

Shalev AY, Rogel-Fuchs Y. Auditory startle reflex in post-traumatic stress disorder patients treated with clonazepam. Isr J Psychiatry Relat Sci 1992; 29 (1):1-6.

Shapiro F. Efficacy of the eye movement desensitization procedure in the treatment of traumatic memories. Journal of Traumatic Stress 1989a; 2:199-223.

Shapiro F. Eye movement desensitization and reprocessing: Basic principles, protocols and procedures. 2nd Edition. New York: Guilford Press; 2001.

Shapiro F. Eye movement desensitization: A new treatment for post-traumatic stress disorder. Journal of Behavior Therapy and Experimental Psychiatry 1989b; 20:211-17.

Sharp TJ, Harvey AG. Chronic pain and posttraumatic stress disorder: mutual maintenance? Clin Psychol Rev 2001;21:857-77.

Sharp TJ. The prevalence of post-traumatic stress disorder in chronic pain patients. Curr Pain Headache Rep 2004;8:111-5.

Sharpley CF, Montgomery IM, Scalzo LA. Comparative efficacy of EMDR and alternative procedures in reducing the vividness of mental images. Scandinavian Journal of Behaviour Therapy 1996; 25:37-42.

Shea MT, McDevitt-Murphy M, Ready D J,Schnurr PP. Group therapy. In EB Foa, TM Keane, MJ Friedman (Eds.), Effective treatments for PTSD: Practice Guidelines from the International Society for Traumatic Stress Studies .(pp. 306-326). Second Edition NY and London, Guilford Press:2009.

Shengold L. Soul murder: the effects of childhood abuse and deprivation. New Haven, CT: Yale University Press; 1989.

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 (4):863-71.

Sherman JJ, Turk DC, Okifuji A: Prevalence and impact of posttraumatic stress disorder-like symptoms on patients with fibromyalgia syndrome. Clin J Pain 2000; 16:127–134.

Sherman JJ. Effects of psychotherapeutic treatments for PTSD: a meta-analysis of controlled clinical trials. J Trauma Stress 1998; 11 (3):413-35.

Sherman MD, Sautter F, Lyons JA, Manguno-Mire GM, Han X, Perry D, Sullivan G. Mental health needs of cohabiting partners of Vietnam veterans with combat-related PTSD. Psychiatr Serv 2005;56:1150-2.

Sherman, MD, Sherman DM. Finding my way: A teen's guide to living with a parent who has experienced trauma. Edina, Minnesota: Beaver Pond Press. 2005.

Shipherd JC, Keyes M, Jovanovic T, Ready DJ, Baltzell D, Worley V, Gordon-Brown V, Hayslett C, Duncan E. Veterans seeking treatment for posttraumatic stress disorder: what about comorbid chronic pain? J Rehabil Res Dev 2007;44:153-66.

Shord SS, Shah K, Lukose A. Drug-botanical interactions: a review of the laboratory, animal, and human data for 8 common botanicals. Integr Cancer Ther 2009;8:208-27.

Sijbrandij M, Olff M, Reitsma JB, Carlier IV, de Vries MH, Gersons BP. Treatment of acute posttraumatic stress disorder with brief cognitive behavioral therapy: a randomized controlled trial. Am J Psychiatry 2007;164:82-90.

Sijbrandij M, Olff M, Reitsma JB, Carlier IV, Gersons BP. Emotional or educational debriefing after psychological trauma. Randomised controlled trial. Br J Psychiatry 2006;189:150-5.

Simon NM, Connor KM, Lang AJ, Rauch S, Krulewicz S, LeBeau RT, Davidson JR, Stein MB, Otto MW, Foa EB, Pollack MH. Paroxetine CR augmentation for posttraumatic stress disorder refractory to prolonged exposure therapy. J Clin Psychiatry 2008;69:400-5.

Sinn N, Bryan J. Effect of supplementation with polyunsaturated fatty acids and micronutrients on learning and behavior problems associated with child ADHD. J Dev Behav Pediatr 2007;28:82-91.

Smith ML, Glass GV, Miller TI. The benefits of psychotherapy. Baltimore, MD: Johns Hopkins University Press; 1980.

Smith TC, Ryan MA, Wingard DL, Slymen DJ, Sallis JF, Kritz-Silverstein D. New onset and persistent symptoms of post-traumatic stress disorder self reported after deployment and combat exposures: prospective population based US military cohort study. BMJ 2008;336:366-71.

Smith TC, Wingard DL, Ryan MA, Kritz-Silverstein D, Slymen DJ, Sallis JF. Prior assault and posttraumatic stress disorder after combat deployment. Epidemiology 2008;19:505-12.

Smyth JM, Hockemeyer JR, Tulloch H. Expressive writing and post-traumatic stress disorder: effects on trauma symptoms, mood states, and cortisol reactivity. Br J Health Psychol 2008;13:85-93.

Spates RC., Koch E., Cusack K. Patago S and Waller S. Eye Moovement Desensitization and Reprocessing. Chapter In EB Foa, TM Keane, MJ Friedman (Eds.), Effective treatments for PTSD: Practice Guidelines from the International Society for Traumatic Stress Studies. Second Edition; NY and London, Guilford Press:2009.

Spiegel D, Classen C, Thurston E, Butler L. Trauma-foscused versus present-focused models of group thrapy or women sexually abused in childhood. In L. J. Koenig, L. S. Doll, A. O'Leary, & W. Pequegnat (Eds.), From childhood sexual abuse to adult sexual risk: Trauma, revictimization, and intervention (pp. 251-268). 2004. Washington, DC: American Psychological Association.

Spiegel D, Frischholz EJ, Fleiss JL et al. Predictors of smoking abstinence following a single-session restructuring intervention with self-hypnosis. Am J Psychiatry 1993; 150 (7):1090-7.

Spiegel D, Frischholz EJ, Maruffi B et al. Hypnotic responsitivity and the treatment of flying phobia. Am J Clin Hypn 1981; 23 (4):239-47.

Spiegel H, Spiegel D. Trance and treatment: clinical uses of hypnosis. Washington, D. C.: American Psychiatric Press; 1987.

Spitzer C, Barnow S, Völzke H, John U, Freyberger HJ, Grabe HJ: Trauma and posttraumatic stress disorder in the elderly: findings from a German community study. J Clin Psychiatry 2008, 69:693-700.

Stapleton JA, Taylor S, Asmundson GJ. Effects of three PTSD treatments on anger and guilt: exposure therapy, eye movement desensitization and reprocessing, and relaxation training. J Trauma Stress 2006;19:19-28.

Steil R, Ehlers A. Dysfunctional meaning of posttraumatic intrusions in chronic PTSD. Behav Res Ther 2000; 38 (6):537-58.

Stein DJ, van der Kolk BA, Austin C, Fayyad R, Clary C. Efficacy of sertraline in posttraumatic stress disorder secondary to interpersonal trauma or childhood abuse. Ann Clin Psychiatry 2006;18:243-9.

Stein DJ, Zungu-Dirwayi N, van der Linden GJ, Seedat S: Pharmacotherapy for posttraumatic stress disorder. Cochrane Database System Review 2000; 4:CD002795.

Stein MB, Kerridge C, Dimsdale JE, Hoyt DB. Pharmacotherapy to prevent PTSD: Results from a randomized controlled proof-of-concept trial in physically injured patients. J Trauma Stress 2007;20:923-32.

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 (10):1777-9.

Stein MB, McAllister TW. Exploring the convergence of posttraumatic stress disorder and mild traumatic brain injury. Am J Psychiatry 2009;166:768-76.

Stein MB, Walker JR, Hazen AL, Forde DR. Full and partial posttraumatic stress disorder: findings from a community survey. Am J Psychiatry 1997;154:1114-9.

Strauss JL, Calhoun PS, Marx CE, Stechuchak KM, Oddone EZ, Swartz MS, Butterfield MI. Comorbid posttraumatic stress disorder is associated with suicidality in male veterans with schizophrenia or schizoaffective disorder. Schizophr Res 2006;84:165-9.

Swanson JW, Swartz MS, Essock SM et al. The social-environmental context of violent behavior in persons treated for severe mental illness. Am J Public Health 2002; 92 (9):1523-31.

Tan G, Craine MH, Bair MJ, Garcia MK, Giordano J, Jensen MP, McDonald SM, Patterson D, Sherman RA, Williams W, Tsao JC. Efficacy of selected complementary and alternative medicine interventions for chronic pain. J Rehabil Res Dev 2007;44:195-222.

Tan G, Fink B, Dao TK, Hebert R, Farmer LS, Sanders A, Pastorek N, Gevirtz R. Associations among pain, PTSD, mTBI, and heart rate variability in veterans of Operation Enduring and Iraqi Freedom: a pilot study. Pain Med 2009;10:1237-45.

Tanielian, T. & Jaycox, L. (Eds.). (2008). Invisible Wounds of War: Psychological and Cognitive Injuries, Their Consequences, and Services to Assist Recovery. Santa Monica, CA: RAND Corporation. http://veterans.rand.org.

Tarrier N, Pilgrim H, Sommerfield C et al. A randomized trial of cognitive therapy and imaginal exposure in the treatment of chronic posttraumatic stress disorder. J Consult Clin Psychol 1999; 67 (1):13-8.

Taubman-Ben-Ari O, Rabinowitz J, Feldman D et al. Post-traumatic stress disorder in primary-care settings: prevalence and physicians' detection. Psychol Med 2001; 31 (3):555-60.

Taylor FB, Lowe K, Thompson C, McFall MM, Peskind ER, Kanter ED, Allison N, Williams J, Martin P, Raskind MA. Daytime prazosin reduces psychological distress to trauma specific cues in civilian trauma posttraumatic stress disorder. Biol Psychiatry 2006;59:577-81.

Taylor FB, Martin P, Thompson C, Williams J, Mellman TA, Gross C, Peskind ER, Raskind MA. Prazosin effects on objective sleep measures and clinical symptoms in civilian trauma posttraumatic stress disorder: a placebo-controlled study. Biol Psychiatry 2008;63:629-32.

Taylor S, Thordarson DS, Maxfield L, Fedoroff IC, Lovell K, Ogrodniczuk J. Comparative efficacy, speed, and adverse effects of three PTSD treatments: exposure therapy, EMDR, and relaxation training. J Consult Clin Psychol 2003;71:330-8.

Telch CF, Agras WS, Linehan MM. Dialectical behavior therapy for binge eating disorder. J Consult Clin Psychol 2001; 69 (6):1061-5.

Terhakopian A, Sinaii N, Engel CC, Schnurr PP, Hoge CW. Estimating population prevalence of posttraumatic stress disorder: an example using the PTSD checklist. J Trauma Stress 2008;21:290-300.

Thomas JL, Wilk JE, Riviere LA, McGurk D, Castro CA, Hoge CW. Prevalence of mental health problems and functional impairment among active component and National Guard soldiers 3 and 12 months following combat in Iraq. Arch Gen Psychiatry 2010;67:614-23.

Trent CR, Jr., Rushlau MG, Munley PH et al. An ethnocultural study of posttraumatic stress disorder in African-American and white American Vietnam War veterans. Psychol Rep 2000; 87 (2):585-92.

Tsemberis S, Eisenberg RF. Pathways to housing: supported housing for street-dwelling homeless individuals with psychiatric disabilities. Psychiatr Serv 2000;51:487-93.

Tucker P, Potter-Kimball R, Wyatt DB, Parker DE, Burgin C, Jones DE, Masters BK. Can physiologic assessment and side effects tease out differences in PTSD trials? A double-blind comparison of citalopram, sertraline, and placebo. Psychopharmacol Bull 2003;37:135-49.

Tucker P, Trautman RP, Wyatt DB, Thompson J, Wu SC, Capece JA, Rosenthal NR. Efficacy and safety of topiramate monotherapy in civilian posttraumatic stress disorder: a randomized, double-blind, placebo-controlled study. J Clin Psychiatry 2007;68:201-6.

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;62:860-8.

Tuerk PW, Yoder M, Ruggiero KJ, Gros DF, Acierno R. A pilot study of prolonged exposure therapy for posttraumatic stress disorder delivered via telehealth technology. J Trauma Stress. 2010 Feb;23(1):116-23.

Turk DC, Okifuji A, Starz TW, Sinclaire JD. Effects of type of symptom onset on psychological distress and disability in fibromyalgia syndrome patients. Pain 1996;68: 678-81.

Turpin G, Downs M, Mason S. Effectiveness of providing self-help information following acute traumatic injury: randomised controlled trial. Br J Psychiatry 2005;187:76-82.

Ulbricht C, Chao W, Costa D, Rusie-Seamon E, Weissner W, Woods J. Clinical evidence of herb-drug interactions: a systematic review by the natural standard research collaboration. Curr Drug Metab 2008;9:1063-120.

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;54:947-9.

van den Hout M, Muris P, Salemink E et al. Autobiographical memories become less vivid and emotional after eye movements. British Journal of Clinical Psychology 2001; 40:121-30.

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;55:517-22.

van der Kolk BA, Spinazzola J, Blaustein ME, et al. A randomized clinical trial of eye movement desensitization and reprocessing (EMDR), fluoxetine, and pill placebo in the treatment of posttraumatic stress disorder: treatment effects and long-term maintenance. J Clin Psychiatry 2007;68(1):37-46.

van Emmerik AA, Kamphuis JH, Emmelkamp PM. Treating acute stress disorder and posttraumatic stress disorder with cognitive behavioral therapy or structured writing therapy: a randomized controlled trial. Psychother Psychosom 2008;77:93-100.

van Emmerik AA, Kamphuis JH, Hulsbosch AM, Emmelkamp PM. Single session debriefing after psychological trauma: a meta-analysis. Lancet 2002;360:766-71.

Van Etten M, Taylor S. Comparative efficacy of treatments for post-traumatic stress disorder: a meta-analysis. Clinical Psychology and Psychotherapy 1998; 5:126-44.

van Praag HM. Moving ahead yet falling behind. A critical appraisal of some trends in contemporary depression research. Neuropsychobiology 1989; 22 (4):181-93

Vaughan K, Armstrong MS, Gold R, O'Connor N, Jenneke W, Tarrier N. A trial of eye movement desensitization compared to image habituation training and applied muscle relaxation in post-traumatic stress disorder. J Behav Ther Exp Psychiatry 1994;25:283-91.

Verheul R, Van Den Bosch LM, Koeter MW et al. Dialectical behaviour therapy for women with borderline personality disorder: 12-month, randomised clinical trial in The Netherlands. Br J Psychiatry 2003; 182:135-40.

Vieweg WV, Julius DA, Fernandez A, Tassone DM, Narla SN, Pandurangi AK. Posttraumatic stress disorder in male military veterans with comorbid overweight and obesity: psychotropic, antihypertensive, and metabolic medications. Prim Care Companion J Clin Psychiatry 2006;8:25-31.

Viola J, Ditzler T, Batzer W, Harazin J, Adams D, Lettich L, Berigan T. Pharmacological management of post-traumatic stress disorder: clinical summary of a five-year retrospective study, 1990-1995. Mil Med 1997;162:616-9.

Wald J, Taylor S. Responses to interoceptive exposure in people with posttraumatic stress disorder (PTSD): a preliminary analysis of induced anxiety reactions and trauma memories and their relationship to anxiety sensitivity and PTSD symptom severity. Cogn Behav Ther 2008;37:90-100.

Walker RL, Clark ME. The “Postdeployment Multi-Symptom Disorder”: An Emerging Syndrome in Need of a New Treatment Paradigm. Psychological Services, 2010.7(3):136-47.

Wallis D. Reduction of trauma symptoms following group therapy. Aust Nz J Psychiatry 2002;36:67-74.

Wang C, Bannuru R, Ramel J, Kupelnick B, Scott T, Schmid CH. Tai Chi on psychological well-being: systematic review and meta-analysis. BMC Complement Altern Med 2010;10:23.

Warner MD, Dorn MR, Peabody CA. Survey on the usefulness of trazodone in patients with PTSD with insomnia or nightmares. Pharmacopsychiatry 2001; 34 (4):128-31.

Watson CG, Tuorila JR, Vickers KS et al. The efficacies of three relaxation regimens in the treatment of PTSD in Vietnam War veterans. J Clin Psychol 1997; 53 (8):917-23.

Watts B. Electroconvulsive therapy for comorbid major depressive disorder and posttraumatic stress disorder. J ECT 2007;23:93-5.

Weine S, Kulauzovic Y, Klebic A, Besic S, Mujagic A, Muzurovic J, Spahovic D, Sclove S, Pavkovic I, Feetham S, Rolland J. Evaluating a multiple-family group access intervention for refugees with PTSD. J Marital Fam Ther 2008;34:149-64.

Weisberg RB, Bruce SE, Machan JT et al. Nonpsychiatric illness among primary care patients with trauma histories and posttraumatic stress disorder. Psychiatr Serv 2002; 53 (7):848-54.

Wells BG, Chu CC, Johnson R et al. Buspirone in the treatment of posttraumatic stress disorder. Pharmacotherapy 1991; 11 (4):340-3.

White House Commission (2002) - Gordon JS. The White House Commission on Complementary and Alternative Medicine Policy: final report and next steps. Altern Ther Health Med 2002;8:28-31.

WHO (1992) - The ICD-10 Classification of Mental and Behavioural Disorders: F43.0 Acute Stress Reaction. Geneva: World Health Organization.

Williams ER, Shepherd SM. Medical clearance of psychiatric patients. Emerg Med Clin North Am 2000; 18 (2):185-98, vii.

Wilson DL, Silver SM, Covi WG, Foster S. Eye movement desensitization and reprocessing: effectiveness and autonomic correlates. J Behav Ther Exp Psychiatry 1996;27:219-29.

Wilson JP, Lindy JD, editors. Countertransference in the treatment of PTSD New York: Guilford Press; 1994.

Wilson S. and Argyropoulos S. Antidepressants and sleep: A qualitative review of the literature. Drugs 2005; 65:927-47.

Witvliet, C.V.O., Phillips, K.A., Feldman, M.E., & Beckham, J.C. (2004). Posttraumatic mental and physical health correlates of forgiveness and religious coping in military veterans. Journal of Traumatic Stress, 17(3), 269-273.

Wolkove N, Elkholy O, Baltzan M, Palayew M. Sleep and aging: 2. Management of sleep disorders in older people. CMAJ 2007;176:1449-54.

Yehuda R, McFarlane AC, Shalev AY. Predicting the development of posttraumatic stress disorder from the acute response to a traumatic event. Biol Psychiatry 1998; 44 (12):1305-13.

Zach S, Raviv S, Inbar R. The benefits of a graduated training program for security officers on physical performance in stressful situations. *International Journal of Stress Management* 2007; 14 (4): 350–369

Zatzick DF, Galea S. An epidemiological approach to the development of early trauma focused intervention. J Trauma Stress 2007;20:401-412.

Zisook S, Chentsova-Dutton YE, Smith-Vaniz A et al. Nefazodone in patients with treatment-refractory posttraumatic stress disorder. J Clin Psychiatry 2000; 61 (3):203-8.

Zivin K, Kim HM, McCarthy JF et al. Suicide mortality among individuals receiving treatment for depression in the Veterans Affairs health system: associations with patient and treatment setting characteristics. Am. J. Public Health 97(12), 2193–2198 (2007).

Zlotnick C, Najavits LM, Rohsenow DJ, Johnson DM. A cognitive-behavioral treatment for incarcerated women with substance abuse disorder and posttraumatic stress disorder: findings from a pilot study. J Subst Abuse Treat 2003;25:99-105.

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;10:425-36.

Zohar J, Amital D, Miodownik C, Kotler M, Bleich A, Lane RM, Austin C. Double-blind placebo-controlled pilot study of sertraline in military veterans with posttraumatic stress disorder. J Clin Psychopharmacol 2002;22:190-5.

Zohar J, Fostick L, Cohen A, Bleich A, Dolfin D, Weissman Z, Doron M, Kaplan Z, Klein E, Shalev AY; Israeli Consortium on PTSD. Risk factors for the development of posttraumatic stress disorder following combat trauma: a semiprospective study. J Clin Psychiatry. 2009 Dec;70(12):1629-35

Zucker TL, Samuelson KW, Muench F, Greenberg MA, Gevirtz RN. The effects of respiratory sinus arrhythmia biofeedback on heart rate variability and posttraumatic stress disorder symptoms: a pilot study. Appl Psychophysiol Biofeedback 2009;34:135-43.

Zygmont M, Prigerson HG, Houck PR et al. A post hoc comparison of paroxetine and nortriptyline for symptoms of traumatic grief. J Clin Psychiatry 1998; 59 (5):241-5.