Treating Victims of Mass Disaster and Terrorism
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The basic objective of this new series is to provide therapists with practical, evidence-based treatment guidance for the most common disorders seen in clinical practice – and to do so in a “reader-friendly” manner. Each book in the series is both a compact “how-to-do” reference on a particular disorder for use by professional clinicians in their daily work, as well as an ideal educational resource for students and for practice-oriented continuing education.

The most important feature of the books is that they are practical and “reader-friendly”: All are structured similarly and all provide a compact and easy-to-follow guide to all aspects that are relevant in real-life practice. Tables, boxed clinical “pearls”, marginal notes, and summary boxes assist orientation, while checklists provide tools for use in daily practice.
Treating Victims of Mass Disaster and Terrorism

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Preface

This book represents the integration of work conducted by a task force jointly sponsored by the Society of Clinical Psychology (Division 12 of the American Psychological Association, APA) and the North American Society for Psychotherapy Research (NASPR) with treatment tools for survivors of mass trauma events. Specifically, this book introduces the concept of using the principles of therapeutic changes identified by this Task Force as a framework for staged treatment for mass-trauma survivors. This approach is offered to the reader as one of many potential alternatives that are available for use in their efforts to address the needs of mass trauma survivors.

Given the preponderance of recent disasters, we find ourselves in a time when many mental health care professionals throughout the world are working to determine what approaches may be the most efficient and effective in assisting survivors. We acknowledge that there are many varied approaches available at this time and anticipate even more being available in the future. Our hope is that this program will serve as a contribution to these efforts, inspire additional ideas, and will be a foundation from which additional work can grow. We do not profess to have all the answers, but the following text offers to you some of our thoughts, intended to be of use to you in your disaster response efforts.
Acknowledgments

Work on this volume was supported in part by a Medical Reserve Corps (MRC) grant to James Breckenridge, PhD, in Palo Alto, CA. Larry E. Beutler, PhD, served as Director and Coinvestigator of the Palo Alto MRC Project and Jennifer Housley, MS, served as the Program Director. Our thanks are extended to Dr. Breckenridge for his ongoing support. The authors also wish to acknowledge and thank Josef Ruzek, PhD, for his valuable contributions to this book.

We would also like to acknowledge those professionals, both researchers and providers, who have dedicated so much of their resources and energy in growing the knowledge base of disaster response and trauma intervention. We are grateful to our peers at the Pacific Graduate School of Psychology who have provided us ongoing encouragement and support.

Consistent with our recommendations to thank those who have made our efforts possible (see Self-Care and Coping section), we would also like to extend our gratitude to our family and friends (specifically to Mr. Patrick Patterson, Ms. Mary Housley, and Ms. Jamie Beutler) for their encouragement, support, and tolerance of our work not only on this volume but also of our involvement in disaster response.

This effort is dedicated to the late Mr. Richard Housley for his everlasting love, knowledge, and wisdom that continue to bring light, comfort, and integrity to so many journeys. Mr. Housley recommended always doing “the best you can with what you’ve got at the time.” There may be few ways to better summarize our ongoing efforts in the disaster response field.
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1 Description

1.1 Terminology

Unlike many treatments that are described both in this series and under the heading of “empirically supported” or “research-based” treatments, the intervention described in this volume does not focus on individuals by diagnostic classification. The treatment of survivors and first responders who have been exposed to mass trauma, and particularly to terrorism is defined by the event, not by a specific form of psychopathological response. While most people who are exposed to mass trauma, including terrorist trauma, will experience acute stress disorder (ASD) during the immediate postevent process, as time goes on, a wide variety of responses occur, including a return to normal functioning.

People are surprisingly resilient, and a substantial majority of those exposed will not warrant a mental health diagnosis at all, beyond the immediate postevent period. Thus, to focus on a specific syndrome, like posttraumatic stress disorder (PTSD), is both to assume a degree of homogeneity of response that is not present following mass trauma and to miss the variety of problems presented. Moreover, basing a treatment on exclusionary consideration of a single diagnostic condition will fail adequately to address the needs of many, if not most, of those who are needy of services and whose postadjustment is characterized by such syndromes as major depression and chemical abuse/dependence, family disruption, and generalized anxiety. Thus, the treatment of survivors of terrorism and disasters must be broadly conceived and easily adaptable to a variety of patient conditions over a substantial period of time.

1.2 Definition

Because this book does not focus on a specific disorder, but rather on the broad range of psychological consequences that follow a terrorist-initiated event (or other mass casualty events), there is not a singular definition that can be provided of the disorder and problem to be treated. It is most efficient to characterize reactions to traumatic events through differing stress reactions (i.e., consequences). These consequences include those reactions normally associated with ASD and PTSD but also include other reactions. These other effects include any temporary or long-term, adverse psychological reactions that are stimulated by the trauma (e.g., use of negative coping in an effort to avoid memories or emotions through increased substance use, major depression, chemical dependence, etc.).
One of the most pervasive and consistent reactions to mass trauma is that of ASD, which is prevalent during the early, postevent period. But, for most people, this syndrome dissipates with time, even without specific treatment. ASD is but the nucleus of symptoms from which a variety of posttrauma reactions may evolve.

PTSD, depression, and chemical abuse are the diagnoses most often seen among postterror and posttrauma survivors, and generally are considered to be stress-induced (e.g., Galea, Ahern, Resnick et al., 2002; Galea, Vlahov, Resnick et al., 2003). A host of other, nonsyndromal, stress-related problems are likely to also manifest themselves in response to terrorist events, however, and many of these require or are likely to be responsive to treatment. These problems may range from specific symptoms of depression and chemical abuse to vague symptoms of anxiety and family disruption.

1.3 Epidemiology

Unfortunately, it is difficult to obtain accurate and reliable base rate data on minor and subclinical, stress-related conditions. The most accurate epidemiological picture of response to the specific case of a terrorist attack comes from mapping the incidence and prevalence rates observed among those who have been exposed to terrorism or other mass trauma onto the base-rates of stress-induced conditions of ASD, PTSD, major depression, and chemical abuse that existed previously in the observed population. The mental health impact of terrorist/mass trauma events can be estimated as the degree to which stress-induced conditions are increased above normative expectations, following a terrorist event. The best estimates of normative expectations for these comparisons are derived from three sources.

The Epidemiologic Catchment Area Study (Narrow et al., 2002; Regier et al., 1998; Robins, Locke, & Regier, 1991), conducted by the National Institute of Mental Health, extracted census-based samples at five sites between 1980 and 1985. Over 20,000 individuals over the age of 18 were surveyed. The National Comorbidity Study (NCS; Kessler et al., 1997) was initiated a few years later in response to a congressional mandate to identify the prevalence of mental health and substance abuse disorders which could then serve as the basis for establishing a national policy for the treatment of mental health and drug abuse disorders. A partial replication of this latter survey (NCS-R; Kessler, Chiu et al., 2005; Kessler, Demler et al., 2005) was conducted about 10 years later, between 2000 and 2003, to replicate the NCS study and to determine changes in incidence and prevalence rates of various disorders.

There are several important methodological differences in how these surveys were conducted. These differences, compounded with changes in the diagnostic system and the introduction of ASD in 1994, with the advent of DSM-IV, resulted in some significant disparities among the ECS and NCS surveys, particularly in estimates of lifetime rates of various disorders. Nonetheless, there is reasonable consistency among the reports on the 12-month incidence rates of trauma-induced disorders (ASD, PTSD, depression, chemical abuse). Supplemented by some specialized and continuing surveys of specific prob-
lems (e.g., the Household Survey on Drug Abuse by SAMHSA, 2002; surveys following the events of September 11, 2001), a reasonable estimate is possible of the impact of mass terrorism.

Combining the results of the initial ECA report (Regier et al., 1998 and the two NCS reports (Kessler et al., 1994; Kessler et al., 2005), the probable, 12-month prevalence rate of PTSD/ASD in the general population is about 8%. The risk rate for women is about twice that of men (10% versus 5%); among men, African-American males are at greatest risk. However, in all likelihood, the observed sex and ethnic differences are reflections of varying social roles, intensity of prior exposure to violence, and contexts rather than being reflections of inherent biological vulnerabilities (Galea, Vlahav, & Resnick, 2003).

Prevalence rates of depression are somewhat more variable in the demographic, normative surveys than are rates of PTSD/ASD in the normative samples. Lifetime prevalence rates of depression vary from 8% in the ECA survey to 19% in the NCS survey, with 12-month rates being somewhat more consistent and hovering near 10% (Beutler, Clarkin, & Bongar, 2000). Adding the prevalence rates of comorbid and non-comorbid chemical abuse, which hover around 10%, results in a general population baseline, 12-month risk of between 22% and 24%. This is the expected rate, within a nonterrorism-exposed population, of having the symptoms that are the most likely to be affected and exacerbated by a mass terror-initiated event.

Against this base rate, one can compare the prevalence rates of these same stress-induced disorders in the New York City area, following the terrorism-initiated events of 9/11/01. It is uncertain how generalizable the resulting estimates of terrorist impact are, however. It is likely that they are culture and region/country specific because of wide variations in the frequency of exposure and cultural beliefs about terrorism that characterizes the responses of survivors from different areas and cultures. For our purposes, we will compare the baseline rates observed in the three U.S. surveys to the rates of problems present among those people who were most directly exposed to mass terrorism on September 11, 2001.

Random surveys of residents of the New York City area following 9/11 have typically concluded that there has been an increase in mental health problems generally, in this region, especially among those most directly exposed to terrorism. However, actual demonstration that the post 9/11 prevalence is higher than the normative base rate expectations has been hard to come by, and estimates of actual incidence rates have varied widely among surveys. Population-based surveys have suggested slightly higher rates of PTSD-like symptoms than those surveys that have relied on less direct assessment methods (Galea, Ahern, Resnick et al., 2002). Nonetheless, it seems quite clear that symptoms of ASD during the first month following a mass trauma event affect most of the exposed population, and it is also clear that there is a high rate of general recovery even in untreated populations, over the following 6 months. Thus, somewhat surprisingly, diagnosable PTSD (which, by definition, can only be present after a month or more following the incident event) was not demonstrably different than the expected normative rates in the New York City area, within about six months of 9/11/2001. The data suggest that the greatest increases of stress-induced problems were in the areas of depression and chemical abuse, rather than in PTSD. Even here, however, it is uncertain how
large the increased risk actually might be. The most careful estimates suggest that over a six-month period, the overall risk of behavioral and emotional disorders was increased by about 10% (e.g., Galea, Vlahov, Resnick et al., 2003; Schuster, Stein et al., 2001; Vlahav, Galea, Resnick et al., 2002; Fairbrother, Stuber, Galea et al., 2003).

1.4 Course and Prognosis

Stress reactions are to be expected following a mass trauma event such as a terrorist attack. In fact, Friedman, Hamblen, Foa, and Charney (2004) report that one third of survivors of high impact disasters experience clinically significant distress, that those who express such symptoms in the early postdisaster time frame are at greatest risk for long-term impairment, and that delayed onset is rare. Nonetheless, there is a rapid recovery and relief of most of the early symptoms of distress. Thus, prognosis for recovery is good to excellent, even among untreated survivors. A substantial portion of victims do, however, have continuing and long-term problems. Predicting who will experience these is a continuing problem. There are a variety of predictors that have been investigated.

Proximity of exposure has been consistently related to the severity or subsequent symptoms. Proximity is defined as either by direct physical exposure or by being indirectly exposed through one’s relationships with survivors. However, even the influence of proximal exposure is moderated by the reported levels of previous exposure to trauma, one’s prior psychiatric status, and by availability of social support networks (Galea, Resnick, Ahern et al., 2002; Galea, Vlahov, Resnick et al., 2003). The role of multiple exposure to trauma is especially important, and among those who are repeatedly exposed, such as combat veterans, the prevalence rates of stress reactions are about double (+30%) that of those exposed to a single, major stressor (Kulka, Schlenger, & Fairbank, 1990).

High levels of acute stress reactions may also predict development of PTSD. Bryant (2003), in his review of studies testing the predictive power of an ASD diagnosis, reports that a portion of people who exhibit ASD within one month posttrauma develop PTSD. However, the majority of those who have ASD symptoms improve over the course of the intervening month and many who develop PTSD have not experienced a full complement of ASD symptoms. Thus, the presence of ASD symptoms immediately following the incident event may not be a reliable predictor of long-term problems (e.g., Friedman et al., 2004).

The presence of negative versus positive cognitions following traumatic events may also assist providers in determining who may be at risk of developing PTSD as positive cognitions may be associated with resilience (Friedman et al., 2004). Additional means by which to distinguish potentially resilient survivors may include the presence of accurate encoding, processing, and trauma memory retrieval early in the posttrauma period (Harvey, Bryant, & Dung, 1998; Moulds & Bryant 2002; Friedman et al., 2004). It is also important to note that avoidance, though one of the many symptoms associated with ASD
and PTSD, may actually serve an adaptive role in the early stages posttrauma (Ehlers & Steil, 1995; Friedman et al., 2004). Other factors likely to contribute to resiliency are high versus low cognitive ability and high versus low levels of social support (McNally, Bryant, & Ehlers, 2004).

Dissociative symptoms have also been investigated as predictors of PTSD. The results of these studies, however, are not entirely clear. For example, some researchers have found dissociation to have no additional predictive power beyond symptoms of reexperiencing, avoidance, and hyperarousal (Brewin, Andrews, Rose, & Kirk, 1999; Marshall & Schell, 2002; McNally et al., 2004) while others have found evidence that dissociation does carry predictive power (e.g., Murray et al., 2002; McNally et al., 2004).

Table 1 summarizes some of the current literature on predictors of long-term difficulties following an incident event. It is important to note that not all risk factors are consistent across studies and that the measures used, type of trauma experienced by participants, and populations sampled differ as well. This table illustrates the wide variety of risk factors associated with the development of PTSD and gives the provider a conceptual understanding of red flags that may alert them to whether referrals may be warranted. It is obviously premature to proclaim that at-risk individuals can be identified with great accuracy, but using this type of information as a general guide, the provider may be able to make better educated decisions regarding referral and treatment for individuals following a mass trauma event when using this proposed 3-stage program.

Collectively, one can conclude that with or without treatment, a large percentage of survivors do not qualify for an Axis I psychiatric diagnosis by the end of six-months following a traumatic incident (e.g., Galea, Resnick, Ahern et al., 2002; Galea, Vlahov, Resnick et al., 2003). Some, however, will continue to experience anxiety symptoms. There is also the possibility that some will have delayed reactions, even though these concerns have not been demonstrated to be warranted in extant research. While constant exploration of these possibilities is necessary, the pressing focus of any intervention must be to identify those who are at long-term risk as soon as possible, and to intervene with these individuals in the hope that by doing so we may reduce the rates of long-term effects among vulnerable groups.

The first problem facing those who seek to develop an effective treatment is identifying those who should be treated. Unfortunately, the relationship of prognostic predictors to treatment effectiveness is unclear, and the relationships between prognostic predictors and actual risk are weak. Without clearly being able to identify those who present a long-term risk, it may not be cost-effective to institute broad-ranging and inclusive treatment programs, especially early in the postevent period. Such programs result in treating many people who will recover without specific intervention. Additionally, such programs applied to everyone may come at the cost of actually slowing the natural course of recovery. For example, the most frequently used treatment during the initial period, immediately following a mass traumatic event, Critical Incident Stress Debriefing (CISD; Beutler et al., 2006; Gist & Lubin, 1999; Litz & Gray, 2004), has been shown to slow down the natural course of recovery among a substantial proportion (perhaps as many as 20%) of survivors (e.g., Litz et al., 2002; McNally et al., 2003; Rose et al., 1998; Rose et al., 1999).
<table>
<thead>
<tr>
<th>Identified predictor or risk factor</th>
<th>Reference</th>
</tr>
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<tbody>
<tr>
<td>Resource loss and depression act as superior predictors of psychological distress than a sense of</td>
<td>Kaiser, Sattler, &amp; Bellack (1996)</td>
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<tr>
<td>coherence and anxiety</td>
<td></td>
</tr>
<tr>
<td>In their review of 160 studies involving disaster victims, Friedman et al. determined that</td>
<td>Friedman, Hamblen, Foa, &amp; Charney (2004) (review article)</td>
</tr>
<tr>
<td>individual level risk factors for poor mental health outcome following disaster include: severity</td>
<td></td>
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<tr>
<td>of exposure, personal characteristics (such as female gender, low socioeconomic status, previous</td>
<td></td>
</tr>
<tr>
<td>psychiatric history, little previous disaster exposure), family context (including child caring</td>
<td></td>
</tr>
<tr>
<td>responsibilities for females, parental distress for children, significant distress by any family</td>
<td></td>
</tr>
<tr>
<td>member), and resource loss.</td>
<td></td>
</tr>
<tr>
<td>Predictors of PTSD are reported as Hispanic ethnicity, 2 or more prior stressors, occurrence of</td>
<td>Galea, Ahern, Resnick, Kilpatrick, Bucuvalas, Gold, &amp; Vlahov (2002)</td>
</tr>
<tr>
<td>panic attack during or immediately postterrorist attack, proximity to disaster location, and</td>
<td></td>
</tr>
<tr>
<td>event-caused loss of possessions.</td>
<td></td>
</tr>
<tr>
<td>Predictors of depression are reported as Hispanic ethnicity, 2 or more stressors, a panic attack,</td>
<td>Galea et al. (2002)</td>
</tr>
<tr>
<td>low level of social support, death of friend or relative, or loss of job resulting from terrorist</td>
<td></td>
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<tr>
<td>attacks.</td>
<td></td>
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<tr>
<td>Dysfunctional cognitions (e.g., world as completely dangerous place and viewing self as</td>
<td>Foa &amp; Cahill (2001); Friedman et al. (2004)</td>
</tr>
<tr>
<td>incompetent) proposed to mediate development and maintenance of PTSD</td>
<td></td>
</tr>
<tr>
<td>Negative cognitions about self and the world and self-blame correlated with measures of PTSD</td>
<td>Foa, Ehlers, Clark, Tolin, &amp; Orsillo (1999)</td>
</tr>
<tr>
<td>severity, depression, and general anxiety</td>
<td></td>
</tr>
<tr>
<td>Cognitive processing style (mental defeat, mental confusion, detachment), appraisal of assault</td>
<td>Dumore, Clark, &amp; Ehlers (2001); McNally et al. (2003)</td>
</tr>
<tr>
<td>sequelae (appraisal of symptoms, perceived negative responses of others, permanent change),</td>
<td></td>
</tr>
<tr>
<td>negative beliefs about self and world, and maladaptive control strategies (avoidance/safety</td>
<td></td>
</tr>
<tr>
<td>seeking) as variables predicting PTSD at both 6 and 9 month follow-ups postphysical or sexual</td>
<td></td>
</tr>
<tr>
<td>assault</td>
<td></td>
</tr>
<tr>
<td>Perceived threat as predictor of PTSD</td>
<td>Kilpatrick, Veronen, &amp; Resick (1982); Friedman et al. (2004)</td>
</tr>
<tr>
<td>Negative expectations about the impact (both immediate and long-term) of the traumatic event as</td>
<td>Bryant (2003); McNally et al. (2003); Friedman et al. (2004)</td>
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<tr>
<td>predictor of PTSD</td>
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Table 1 (continued)

<table>
<thead>
<tr>
<th>Identified predictor or risk factor</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sense of personal incompetence and loss of sanity as predictors of PTSD</td>
<td>Ehlers &amp; Clark (2000); McNally et al. (2003); Friedman et al., (2004)</td>
</tr>
<tr>
<td>Prior exposure to trauma</td>
<td>King, King, Foy, Keane, &amp; Fairbank (1999); Litz et al. (2002)</td>
</tr>
<tr>
<td>Indicates that risk of PTSD may be increased by effects of female gender, greater social, educational, and intellectual disadvantages, individual and family psychiatric history, previous adversity, and childhood abuse</td>
<td>Brewin, Andrews, &amp; Valentine (2000)</td>
</tr>
<tr>
<td>Posttrauma shame, guilt, and self-blame following the trauma as predictors of PTSD</td>
<td>Andrews, Brewin, Rose, &amp; Kirk (2000); McNally et al. (2003); Friedman et al. (2004)</td>
</tr>
<tr>
<td>Posttrauma (1 to 2 weeks and onward) symptom severity; depersonalization, emotional numbing, motor restlessness and a sense of reliving the trauma as measured within 1 month posttrauma as predictor of PTSD</td>
<td>Harvey &amp; Bryant (1998); McNally et al. (2003)</td>
</tr>
<tr>
<td>Peri-traumatic dissociation as predictor of PTSD</td>
<td>Ozer, Best, Lipsey, &amp; Weiss (2003); McNally et al. (2003)</td>
</tr>
<tr>
<td>Cognitive ability as predictor of PTSD</td>
<td>Vasterling, Brailey, &amp; Constans, (1997); McNally et al. (2003)</td>
</tr>
<tr>
<td>Peri-traumatic dissociation measured at 4 weeks posttrauma was a better predictor than persistent dissociation measured at 1-week posttrauma of PTSD at 6 months posttrauma</td>
<td>Murray, Ehlers, &amp; Mayou (2002); McNally et al. (2003)</td>
</tr>
<tr>
<td>Derealization and sense of time distortion at 1-week posttrauma as predictors of PTSD 6-months posttrauma</td>
<td>Shalev, Peri Canetti, &amp; Schrieber, (1996); McNally et al. (2003)</td>
</tr>
<tr>
<td>Potential for acute psychophysiological arousal as mediator of PTSD (long-term)</td>
<td>Bryant, Harvey, Guthrie, &amp; Moulds (2003)</td>
</tr>
</tbody>
</table>

The National Institute for Clinical Excellence (2005) recommends that “for individuals who have experienced a traumatic event, the systematic provision to that individual alone of brief, single-session interventions (often referred to as debriefing) that focus on the traumatic incident, should not be routine practice when delivering services” (p. 4).
1.5 Differential Diagnosis

For the purposes of this book, differential diagnosis is defined as the processes by which one can differentiate among those conditions that are initiated by a mass traumatic event and those that are preexistent or that are initiated by some other event.

The major conditions that are initiated by a mass traumatic event include ASD, PTSD, major depression, and chemical abuse or dependence. Additional, more transitory symptoms (e.g., sleep disorder, general anxiety, adjustment disorder, etc.) may also occur following trauma. These conditions may also occur from events other than a mass trauma, and in some cases may be observed in the absence of any identifiable traumatic event (e.g., major depression, chemical abuse). However, for the purposes of this treatment program, all symptoms and conditions within these spectra are considered to be available for treatment using this protocol, if they arise subsequent to a mass trauma, especially one that is initiated by a terrorist event. Symptoms and disorders that arise following a terrorist-initiated event but that did not exist prior to the event in the same form are defined by the nature of the event and the individual’s reaction to the event.

Though the approach taken by this volume does not seek to address specific disorders, it is helpful to review disorders that share symptoms similar to those seen following mass casualty events. The following descriptions are derived from the *DSM-IV-TR* (American Psychiatric Association, 2001). The descriptions provided are intended for informational purposes only and the reader is encouraged to consult the *DSM-IV-TR* for more detailed information regarding symptoms and diagnostic criteria.

1.5.1 Posttraumatic Stress Disorder (PTSD)

To diagnose someone with PTSD, the individual must have had exposure to a traumatic event that involved real or perceived threat to self or others. The individual must also experience one or more symptoms of reexperiencing, three or more symptoms of avoidance/numbing, and two or more symptoms of increased arousal. These symptoms, in addition to significant distress in life functioning, must be present for more than one month.

1.5.2 Acute Stress Disorder (ASD)

ASD shares a variety of symptoms with PTSD. The difference between ASD and PTSD is primarily a temporal one. For ASD, symptoms must be present for a minimum of 2 days and a maximum of 4 weeks and must occur within 1 month of the traumatic event. There are also less strict criteria as to the number of symptoms experienced within each category. The person must have experienced at least three symptoms of dissociation, one or more persistent reexperiencing symptoms, exhibit marked avoidance and increased arousal, and experience significant distress in life functioning. In addition, the client’s symptoms must not be due to substance use or a general medical condition.
1.5.3 Other Anxiety Disorders

As previously mentioned, a variety of anxiety-related symptoms are common following a significant traumatic event. To diagnose specific anxiety disorders additional criteria must be met. Anxiety disorders such as panic disorder with or without agoraphobia and generalized anxiety disorder share symptoms with those commonly seen after trauma exposure. For panic disorder with agoraphobia, the individual must experience recurrent unexpected panic attacks. These attacks must be followed by one month of one or more of either persistent concern about having more attacks, concern about the implication of an attack, and/or a significant behavioral change related to such attacks. In both cases, whether agoraphobia is present or not, the panic attacks must not be due to substance use or a general medical condition. For GAD, the individual must express excessive anxiety and worry for more days than not for at least six months. The worry must be difficult to control, and be associated with a range of other symptoms such as fatigue, irritability, difficulty concentration, tension, and sleep disturbance.

1.5.4 Major Depressive Disorder

A major depressive episode is characterized by the presence of five or more symptoms being present within a 2 week period where at least one of the symptoms is depressed mood or loss of interest or pleasure. These symptoms include depressed mood for the majority of the day, diminished pleasure in activities nearly everyday, significant weight change, sleep disturbances, psychomotor agitation or retardation, fatigue, feelings of worthlessness or guilt, and decreased ability to concentrate. These symptoms must not be due to substance use or a general medical condition. Major depressive disorder can be diagnosed as single episode or recurrent. To be considered as recurrent, there must be two or more depressive episodes that must be spaced out by at least 2 consecutive months.

1.5.5 Sleep Disorders

A variety of symptoms associated with sleep disorders are common among the reactions seen following traumatic events. These include dyssomnia’s such as primary insomnia. Primary hypersomnia, dyssomnia not otherwise specified, and parasomnia’s such as nightmare disorder, sleep terror disorder, and substance-induced sleep disorder. For dyssomnia’s, symptoms must be present for one month (or less if recurrent). To diagnose nightmare disorder, the nightmares must not be attributable to PTSD, substance or general medical condition. For sleep terror disorder, the individual must not be able to recall the dream in detail or respond to others’ efforts to comfort them during the terror, nor can the symptoms be due to substance use or a general medical condition.
1.5.6 Adjustment Disorders

Diagnoses of adjustment disorder requires a change in behavior or emotion that occurs within three months of the onset of an identifiable stressor. It is important to note that once the stressor is over, the symptoms must not continue for more than six months. A diagnosis of chronic adjustment disorder can be made if the symptoms last more than six months and are in response to a chronic stressor or one that has ongoing consequences.

1.5.7 Substance Related Disorders

Substance disorders include both substance dependence and substance abuse. Substance dependence requires that the individual exhibit a maladaptive pattern of abuse that leads to impairment. Within 12 months, three or more symptoms must be present. These symptoms include tolerance, withdrawal, taken in larger amounts or over longer time periods than intended, desire or failed efforts to reduce intake, spending a large portion of time attaining substance, decreased participation in social, job-related, or recreational activities, and continued use despite continued risk for physical or psychological harm to ensue. Substance abuse is characterized by recurrent and maladaptive substance use patterns that impair the individuals ability to meet expectations associated with work, school, or home activities, use that puts the individual or others at risk for harm (including legal problems), and continued use despite potential or actual problems.

1.6 Comorbidities

Aside from distinguishing among the foregoing disorders and conditions, the reaction to extreme trauma is complicated by the many coexistent or comorbid conditions that can occur. Any and all of the foregoing disorders may present as comorbid conditions. That is, they may coexist with one another. Major depressive disorder is a comorbid condition with anxiety conditions in over 60% of the cases (Beutler, Clarkin, & Bongar, 2000). Likewise, chemical dependence is a comorbid condition with anxiety and depressive disorders in a substantial percentage of cases (Galea et al., 2003). Moreover, personality disorders are a significant concern as comorbid conditions and tend to reduce the speed and magnitude of treatment gains (Castonguay & Beutler, 2006).

1.7 Diagnostic Procedures

Before discussing diagnostic procedures and screening/assessment, it is worth a moment to discuss the temporal aspects of early intervention care. Generally, intense emotional reactions immediately following a traumatic event are not to be considered pathological. Litz and Gray (2004) refer to this stage as the
Immediate impact phase in which immediate psychological and biological impacts of trauma are still present. The acute phase, as they term it, begins after the immediate impact phase and is a time in which survivors may be more apt to receive prevention interventions. For this program, we will address diagnostic procedures and interventions within three stages: (1) the acute support stage, (2) the intermediate support stage, and (3) the on-going treatment stage. Specific durations (number of days, weeks, months etc.) cannot be assigned to each stage given the uncertainties associated with mass trauma events, such as the need for repeated relocation for safety, repeated trauma (hurricane, flood, fire), and so forth, however, general temporal distinctions between stages are provided as a general guide.

The treatment program outlined here is designed to be flexible. And, while its implementation does not depend on the establishment of a formal diagnosis, particularly in the early period following an incident event, evaluation is a central and integrated part of the process. Evaluation is embedded in the procedure throughout and is closely related to treatment. It is somewhat awkward, therefore, to separate diagnostic and evaluation procedures into a section that is separate from treatment. But, for clarity and consistency across volumes, we have done so. In this section, we will describe the evaluation procedures that are used in each of the three stages, but in actual practice, these procedures are more a part of treatment itself than a separate activity.

In the immediate period following such exposure, virtually all “survivors” will have many of the symptoms of ASD, but few of these survivors will warrant or need treatment beyond the support and resources made available informally in the first hours after an attack. Differentiation of those who will need professional care from those who will not need such treatment is not possible at this point, and selecting a diagnosis beyond describing the symptoms of acute stress is virtually impossible. The assessment concentrates on identifying those who are likely to have continuing difficulties, regardless of the diagnostic form that these may take.

As time passes, many symptoms dissipate and change. There is no single diagnosis that is preeminent in defining one’s response. Though many clinicians assume that PTSD will be the defining condition, this is not the case, and it is not until 6–8 weeks after the incident event that one can begin to identify those individuals for whom a diagnostic formulation will be helpful in planning treatment. Only at this point, is the patient’s diagnosis relevant for treatment, and even then, a general rather than a specific diagnostic procedure is indicated. While a specific diagnosis will help orient treatment to fit each patient, the symptoms captured in the diagnosis are more helpful for defining how to assess and monitor change and improvement over time than to plan a truly discriminating treatment.

Many scholars (e.g., Litz et al., 2002) have raised the concern that specific explorations into traumatic experiences during the immediate posttrauma period may resensitize patients to the trauma itself, and may exacerbate anxiety rather than ameliorate it. Litz et al. suggest that the failure of CISD to produce better and faster rates of recovery among some victims, relative to those in no-treatment comparison groups, may be the result of resensitization resulting from prematurely raising strong emotions about the events. Moreover, since there are both multiple kinds of response to disaster and because most people
will respond positively without treatment, the assessment procedure should be less diagnostic than prognostic in focus. The basic task is to identify, as soon as possible, those who are at risk for having ongoing anxiety, depression, chemical abuse, or related conditions.

To avoid resensitizing the patient, the assessment procedure for identifying long-term risk must be as unobtrusive as possible and focused as much as possible on those factors that are known to be associated with prognosis. Moreover, because the risk of false positives (those predicted to be at risk but who are not) reduces with time, it should be implemented in stages over time, becoming increasingly honed to those who are most at risk. This staged approach permits the clinician to increasingly focus attention and the necessary resources on those who are not likely to respond resiliently. Thus, the treatment program described in this volume is presented in three stages, each of which is associated with a specific type of assessment, and each of which is embedded in a principle driven treatment protocol.

1.7.1 Introduction to Principle-Driven Treatment and Assessment

The unique aspect of the treatment program outlined in this volume is that it is based on a set of empirically derived principles of change rather than on a discrete therapeutic model. By building a treatment around empirically informed principles and using strategies that have been tested and researched in controlled research, the likelihood of treatment being demonstrably effective is increased.

The principles which guide treatment were identified by a joint task force of the Society of Clinical Psychology (Division 12 of the American Psychological Association) and the North American Society for Psychotherapy Research (Castonguay & Beutler, 2006a). Twenty-five task force members and 20 affiliated authors devoted three years to reviewing extant research and extracting a list of principles of therapeutic change that met the group’s criteria of efficacy. Subgroups worked on each of four problem areas: depressive spectrum disorders, anxiety disorders, chemical abuse disorders, and personality disorders. Within each problem area, they focused on each of three domains of variables: Qualities of the patient and therapist (participant variables), qualities of a beneficial relationship, and characteristics of effective treatments. In each case, they did so with an eye toward expressing relationships as “principles” that cut across theoretical models and specific techniques and focused on guiding strategies.

In the final analysis, the Joint Task Force (Castonguay & Beutler, 2006b) identified a total of 26 “common” and 35 “unique” principles. Common principles are those that cut across patient problems and disorders, while unique principles are those that appear to be relatively specific to the application of treatment to certain problems. In the current treatment, we relied on both common principles and unique principles that were specific to the treatment of anxiety disorders, depression, and chemical abuse. We have modified the principles slightly to clarify them and to make them more applicable to the experiences of mass trauma and terrorism.