

Treating Survivors in the Acute Aftermath of Traumatic Events

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Outline

Several factors make the treatment of survivors, in the acute aftermath of traumatic events, extremely difficult to describe and discuss. At such time the survivor's concrete needs may be very urgent, secondary stressors may still be operating, expressions of distress are volatile and highly reactive to external realities and symptoms expressed may not reflect psychopathology. Importantly, normal healing processes are already operating, and significant assistance is provided by natural supporters and healers (e.g., relatives, community leaders) and should not be interfered with. Professional helpers are often enduring significant stress themselves and do not operate in their usual environment. The adequacy of both the medical and psychological treatment models must, therefore, be questioned. An alternative model may be considered, which favors knowledge of pathogenic processes over symptom recognition. The treatment of early survivors requires therapeutic flexibility. The more one is professionally prepared to handle novelty and uncertainty, the better one's therapeutic impact may be. Helpers' unavoidable distress should be managed during the intervention, such that they remain effective and don't harm themselves.

Introduction

Current knowledge about post-traumatic stress disorder (PTSD) has sensitized the

public and the mental health community to the damaging potential of exposure to traumatic events. From a clinical point of view it is important to note that most trauma survivors who develop prolonged stress disorders express symptoms of distress at the early aftermath of traumatization (Rothbaum, Foa, Riggs, Murdock, & et al, 1992). Moreover, most instances of recovery from early and distressful responses to traumatic events occur within the following year (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995); (Shalev, Freedman, Peri, Brandes, & Sahar, 1997). The early aftermath of traumatization offers, therefore, a window of opportunity during which individuals at risk for developing chronic stress disorders can be identified and treated (Solomon & Benbenishty, 1986); (Bryant, Harvey, Dang, Sackville, & Basten, 1998); (Foa, Hearst Ikeda, & Perry, 1995).

The **optimal time** for such interventions, however, is unclear. On one hand, the very early days that follow traumatic events may constitute a 'critical' or 'sensitive' period, during which neuronal plasticity is enhanced (Shalev, 1999), and indelible aversive learning occurs (Shalev, Rogel Fuchs, & Pitman, 1992). On the other hand, most trauma survivors do not present to treatment before having endured weeks of suffering, possibly because they, and others around them, see the initial distress, and the associated symptoms as a normal response. It is also unclear whether the very early and short interventions, such as on-site debriefing, have any prolonged effect (Bisson & Deahl, 1994; Raphael, Meldrum, & McFarlane, 1996). Studies of early treatment of combat soldiers, however, point to a more positive outcome (Solomon & Benbenishty, 1986){Solomon, Gerrity, et al. 1992 #985}.

Beyond their optimal timing, the **content and the techniques** of immediate

interventions must be examined. Interventions conducted weeks or months following trauma have received some attention in the literature, and involve treatment techniques that resemble those used in prolonged mental disorders (e.g., CBT). Whether or not the earlier, acute interventions should be conducted according to the same principles is unclear. What is clear, however, is that the physical and mental condition of survivors, at the end of the impact phase of a trauma, are very different from those seen days or few weeks later. Most are mid way between enduring stress and re-appraising its consequences (Lazarus, 1984). Helpers and therapists who approach the survivors at this stage are also 'in the field,' and not in their usual working environment. Some are experiencing, along with their clients, a major life event. As such, these circumstances dictate a modified approach.

There are several ways in which a 'therapy' during the acute phase may be different. **First**, a *conceptual re-framing* is needed: at this phase one may still be handling the trauma, rather than treating a post-traumatic condition. Psychological rescue (or first aid) may, therefore, be a proper term for some interventions. **Second**, along with symptoms, current sources of stress should be in the forefront of the clinical evaluation. Relocation, separation or continuous threat (such as during political repression) are powerful modulators of behavior, which can not be ignored when the totality of the individual is considered. Help at this stage may consist of mitigating the effect of concurrent stressors. **Third**, the complexity of events and responses should be noted: The mental and physical conditions that follow a traumatic events are extremely complex, and the resulting behavior is unstable and rapidly changing (Shalev in JTS, Solomon, Riess on therapeutic

flexibility). The perception of the event may vary from one individual to the other (Shalev at JTS). For better or worse, individuals may be suggestible and unusually reactive: they may be very responsive to the emotional tone of helpers, but also reactive to real or fantasized realities, such as rumors. **Fourth**, expression of distress are often appropriate at this stage, and one should be very careful not to classify them as 'symptoms' in the sense of being indicative of a mental disorder. Medicalising (or pathologizing) an early response is often the fruit of profound misunderstanding of the role of pain and anxiety as signals to the body, to the psyche and to others. An essential diagnostic element, at this stage, is, therefore, not so much the intensity, but rather the appropriateness and the 'productiveness' of the early response. **Fifth**, during rescue efforts professionals and non-professionals may have similar roles (e.g., soothing, comforting, orienting, reassuring etc...). Non professional, however, are available in larger numbers and include the survivor's natural supporters (e.g., relatives, peers) and other community members. These supporters may also be overwhelmed and distressed, and in many instances the professional helper's role is to support and guide the supporters: a nurse, a family member, a disaster area manager etc. **Finally**, sharing another human being's grief is a powerful emotional response, which has a unique healing power. Sharing may also be painful. Sharing by therapists, therefore, is both desirable and unavoidable. The degree to which professional helpers are being induced to share emotions, are able to sustain such experiences, and receive adequate preparation and support in so doing may have important effects on their efficacy as helpers, and on their own well being.

The above-mentioned particularities of the early response to traumatic events

lead to asking another important question: Who, if at all, should be treated by mental health specialists? On the one hand, the very frequent occurrence of traumatic events (Breslau & Davis, 1992) defies any effort to provide specialized care for all. This is especially true for underdeveloped countries, and major disaster areas. Moreover, as mentioned above, in most cases the responses are self-limited. Yet, given the risk of developing chronic stress disorders providing such help may make the difference between recovery and life-long illness.

This dilemma has been approached in two systematic ways: The first was to provide specialized treatment to those identified as being ill (e.g., soldiers who ceased to function during combat because of stress responses) (Kormos, 1978, Solomon, 1993). The second consisted of covering all those exposed by providing some of professional intervention, recently in the form of debriefing.

Far from being solved, however, the question of whether or not to intervene, or what kind of intervention to provide, emerges again and again, in each individual case. Deciding what to do and how much, is the essence of clinical wisdom. This chapter proposes to help making such decisions by pointing to the following ideas:

The dichotomous choice between treatment and no-treatment should be replaced by the notion of 'depth of treatment'.

The early and urgent needs of all should be addressed (yet, not necessarily by psychological interventions)

Trauma survivors should be considered as being at risk for developing traumatic stress disorders.

Specific risk factors should be evaluated, for each case, on the basis of the existing literature

The survivors' progress towards recovery should be followed and clinical decisions made on the basis of longitudinal observations (instead of cross-sectional examination).

Treatment should be provided in a context of continuity of care.

This chapter delineates the implementation of these ideas. It starts by discussing the nature of traumatic events. It subsequently outlines a framework for assessing trauma survivors. It then points to the general rules of early intervention and to some intervention techniques. It ends by advising clinician about termination of treatment or continuity of care. In order to remain practical, the chapter avoids extended discussion of each point, sending the reader to the relevant literature.

The traumatic event

Formal definition

The DSM IV (1994, p.431) defines traumatic events as including an element of threat ("...actual or threatened death or serious injury, or a threat to the physical integrity of self or others") and a typical response ("intense fear helplessness or horror"). DSM IV definition sets an entry criterion for considering an event as traumatic in the context of making a diagnosis of PTSD. It should not be read, however, as a good-enough descriptor of traumatic events: It is non-specific (i.e., applies to wide variety of events, from car accidents to being incarcerated in a concentration camp). Importantly, DSM IV definition does not address the mechanisms of mental traumatization.

Understanding the mechanisms of mental traumatization is extremely important, particularly when one comes to evaluate and assist the recent survivor. Phenomenology alone is not enough at this stage: specific syndromes are not yet formed, there is great variability in the expression of distress, and one is better

served by knowing the principles than by identifying sets of symptoms. Moreover, events are never 'traumatic' just because they meet a threshold criterion. Extreme events become traumatic when they include one or several pathogenic elements, or when the individual exposed is, for some reason, vulnerable to their effect.

This section outlines some of the traumatizing elements of events, the salient responses and the ways in which people cope with traumatic stressors (and with their own responses). A discussion of vulnerability and risk factors for developing traumatic stress disorders is beyond the scope of this chapter.

Trauma is more than fear and threat

Initially believed to mainly consist of threat and fear response, extreme events may, in fact, traumatize people in many different ways. For some, fear and threat are indeed the essence of a trauma. For others, the traumatic event includes a major element of loss. Exposure to grotesque and disfigured human bodies may be the sole traumatizing element of some other events (McCarroll et al., 1995)(15). Dehumanization, degradation and humiliation are at the core of other experience, such as rape, racially motivated trauma, or torture. Forced separation, relocation, are other independent elements. For example, in a study of acute stress disorder among prisoners of a concentration camp, (Kozaric Kovacic et al.,) the prisoners rated lack of information about their families as most stressful during their captivity.

Several concrete elements of traumatic events are known to increase the risk for post-traumatic stress disorders. These include a threat to one's life and body integrity; severe physical harm or injury; receipt of intentional injury or harm; exposure to the grotesque; witnessing or learning of violence to loved ones; and

causing the death or severe harm to another (Green, 1995).

The current inclusion of all such experiences under the title of 'psychological trauma' implies that all of them are similar at some level (e.g., biologically). This, however, has not been proven convincingly. Indeed, specific ways in which one is traumatized may have a prolonged effect. A study of chronic PTSD, for example, suggests that exposure therapy, a method derived from a 'threat and learning' model, may not be effective for survivors who have endured mental defeat, alienation, or permanent change (Ehlers et al., 1998)

It follows that the clinical assessment of the recent survivor should clarify, first, what had been particularly traumatizing for an individual within the traumatic event. This is important because helpers may tend to assume a traumatizing element of an event by putting themselves (or their theory) in the place of the survivor. For example a-priori assuming that threat was the major element in the case of a survivor of bombing or shooting incident: further inquiry may reveal other major sources of distress, such as having been separated from one's child during the evacuation or having failed to rescue significant others. Hence, in approaching the recent trauma survivor, who is emerged in his or her particular narrative, understanding individual experience (as opposed to imposing one's own template) is the key for creating a therapeutic report.

Traumatic events can also be described by their psychological dimensions. Both psychologically and biologically the severity of traumatic events is related to their being intense, inescapable, uncontrollable and unexpected (Foa et al., 1992). Traumatic events can also be defined as those exceeding the person's coping resources (Lazarus and Folkman, 1984) or breaking his or her protective

defenses (Freud, 1920/1955). In the assessment section, below shall make clear how these very abstract construct may be used to guide one's practice.

The biological dimension

Construed under the general umbrella of stress- and learned conditioning theories, the biological interpretation of mental traumatization have to explain the link between the early biological response to extreme events and the subsequent development of mental disorders. PTSD was originally explained as an exaggeration of a normal learning response, related to fear conditioning (Pitman, 1988). The intensity of the initial adrenergic response was believed to foster emotional (and amygdala – mediated) learning, at the expenses of rational or declarative, hippocampus mediated learning (e.g., Metcalfe and Jacobs, 1996.). Accordingly, initial hypersecretion of the stress hormone epinephrine could be involved in an exaggeration and a consolidation of fear-related memories of the traumatic event (Cahill et al., 1994), McGaugh, 1990). Supporting evidence for the above theory can be found in a recent study, in which heart rate levels upon admission to an emergency room following trauma were linked with the subsequent occurrence PTSD (Shalev et al., 1998)

Recently, the belief in a normal initial response has been challenged (Yehuda & McFarlane, 1995). **Abnormally low cortisol levels** following trauma were reported, shortly after trauma in individuals who were at higher risk for developing PTSD (Resnick et al., ; Yehuda et al.,). A combination of increased adrenergic activation and low plasma levels of cortisol (Yehuda et al., 1990) had been shown to synergetically increase emotional learning (e.g., Munch et al., 1984).

An alternative to the role of early biological responses argues that prolonged stress disorders result from

factors that follow the initial exposure. PTSD, accordingly, follows a 'progressive temporal sensitization' (Antelman,), which may be linked to the presence of persistent reminders of the traumatic event or to other stressors. Abnormal responses to startle, for example, do not develop in individuals with PTSD until one or four months following exposure (Shalev et al., in press). Recent prospective studies further suggest that depressive symptoms, during the weeks that follow trauma, are potent predictors of PTSD, explaining the occurrence of the disorder above and beyond prediction made from early PTSD- and anxiety symptoms (Freedman et al., 1990).

Possibly, a mixture of early stress responses and delayed activation of other biological co-factors is the best explanatory model for PTSD. Importantly both theories point to the causal role of early distress in PTSD, hence the focus on reducing distress by all possible means.

Human responses to traumatic stressors

Learning by fear

Understanding the impact of threat and alarm on the brain comes from two theoretical bodies: stress theory and classical conditioning theory. Stress theory predicts that a threat would be responded to by specific innate or previously acquired defenses. Learned conditioning theory further predicts that stress would be associated with learning, particularly with of learning avoidance and emotional memories.

The intensity of the threat (Resnick et al., 1995), its perception by the individual and the quality of the immediate bio-psychological response are, therefore, important predictors of subsequent psychopathology . The degree of control over events and over one's reaction is another important bio-

psychological modulator of the effect of stress on the brain ((Prince CR & Anisman H, 1990). Physiological stress (e.g., bleeding or dehydration) may further enhance the hormonal stress response.

Impacted grief

Beyond threat and fear, traumatic events often cause real and symbolic damage in the form of injury, separation or death of significant others, destruction of social networks etc.... Common to all of the above is an element of **loss**. Loss is an independent and rather neglected dimension of mental traumatization (Hobfoll & Jackson, 1991). Yet, far from being a secondary mechanism, loss and subsequent mental processing may be central to the development of PTSD. For example, loss of social network, due to relocation, was found to predict higher levels of PTSD symptoms 3-4 years following an earthquake (Bland et al., 1988-1994). Indeed, the core PTSD symptoms of intrusive recollections, numbing and detachment have been derived from earlier descriptions of unexpected loss (Lindemann E, 1994). The combined effect of loss and threat may similarly explain the frequent co-occurrence of PTSD and depression. Finally, responses to loss (e.g., of territory, offspring or partners) may trigger independent neuro-biological mechanisms of weaning and yearning, which may come to complement those related to threat.

Collapse of structures and defenses

A third traumatizing element of extreme events is the **collapse of defensive mental structures**. A frequent clinical expression of a breakdown of one's defenses is "I could not believe that this was happening" or "I felt paralyzed, unable to thinking and act". Breaking down, either during the event (e.g., surrender to pain or to a threat) or during the immediate aftermath maybe extremely damaging to individuals in

that previous defenses (or 'coping mechanisms', or 'cognitive schemata') are "shattered" (Janoff Bulman, 1989) and have to be rebuilt. This category of responses may be seen in exposure to human cruelty, forced degradation, trauma motivated by racism, human right violations and other events to which one can not be mentally prepared. Again, being overwhelmed by grotesque events may be independent from experiencing a threat or a loss yet may leave individuals puzzled, restless and traumatized.

Isolation; break down of social bonds

A most striking description of mental traumatization is that of an army officer who fought in the 1982 Lebanon war and developed PTSD. Few days into the war, while advancing with his men on one of the main roads, they met an evacuation convoy, carrying casualties. By curiosity he stopped and jumped into one of the vehicles to discover the disfigured body of a deadly wounded close friend. He describes his experience in the following hours as follows: "From that point on nothing mattered any more. I continued to sit by my driver, as I did before, but was totally cut from others. I was completely alone, detached from my own soldiers who suddenly became total strangers to me." Dasberg (1976) described loneliness and social isolation as a core traumatic experience in combat stress reaction casualties of the 1956 Sinai campaign. Indeed, a piercing experience of many PTSD patients is the alienation from others, often expressed as "No one can ever understand what I have been through" (or what I experience now).

Complementing these retrospective descriptions is the view that it is most difficult, if not impossible to recover from trauma on one's own. As with serious physical injury, psychological wounds require the help of others to heal. The prime element of such help is to firstly break the wall of mental isolation, which often follows exposure

to extreme stressors. Hence the importance of the **quality of the initial contact** established with survivors. It is equally important not to let such walls be built again: Many trauma survivors carry the experience of good initial intent and subsequent ‘betrayal.’ Hence the importance of continuity of care.

Closing the narrative.

Finally, it is during the short period that follows trauma that a stable narrative of the traumatic events and of one’s own responses is formed and consolidated (Shalev et al., *milit. Medicine*, 1998), and these may shape the way in which the event will be remembered. Long-term memories of one’s personal experience can often be confounded by what others have said and observed and with the larger social appraisal of the event (e.g., a failure, disaster, heroic act etc...). Appraisal of one’s current symptoms may predict PTSD above and beyond the effect of symptom severity (Ehlers et al., 1998). The resulting mixture of personal and narrated facts is then consolidated into the ‘authentic’ and ‘accurate’ memory of the event. One is especially vulnerable to the effect of such interference during the immediate post-event period (Loftus, 1993).

The social context

The social context of a traumatic event has major effects on the expression and the course of the immediate responses. Concrete and adverse social factors include the above-mentioned relocation, family disruption, and dissolution of communities. Other factors include community leadership and appraisal of the event by the society. Societies tend to assign a value tag to being exposed to traumatic events and to one’s behavior during exposure (e.g., merit, virtue, and honor versus shame, cowardice or

dishonor). These tags may confer a decisive meaning to the event, to which the survivor himself or herself may adhere. Importantly **the social perception of trauma survivors is often polarized, going from glorification to defamation.** All too often it is the victim who is blamed for the victimizing event, (e.g., a young female blamed for having provoked a rape). **Extreme social tags are often counter-productive.** Both a recognized hero and defamed coward may find it difficult to access and work-through their traumatic experience. Those who intervene in the immediate aftermath of extreme events should, therefore, facilitate the expression of individual experiences and go beyond socially assigned value tags. One should let a hero cry out his or her fear, and a rape victim tell how wisely he or she managed to escape death.

Coping with traumatic stress

During the days that follow trauma survivors go from a period of being under stress to a period of reappraisal and reevaluation. Typical for the ‘traumatic’ period is the use of extreme defenses, such as over control of emotions or dissociation and a focus of **surviving trauma**. The second period is characterized by intrusive recollections of the traumatic event and has for main psychological task the **assimilation** of events and their consequences. Both periods can be extremely painful, hence the need to cope effectively during each of them. Following is a short description of the way in which coping can be assessed.

Coping can be defined as an effort to reduce the effect of environmental demands on physiological and psychological responses, i.e., ‘**effort to increase the gap between stress and distress**’ (Pearlin and Schooler, 1978). Coping mechanisms have been authoritatively discussed (e.g., Lazarus

and Folkman, 1984), and most such descriptions address the myriad of specific ways in which people react to adversity. Individuals may, indeed, differ significantly in their preferred ways of coping: Some are action prone, others are more reflective and analytical. Some would express emotions while others may hide them.

Studies of coping in trauma survivors have addressed the relative efficacy of specific ways of coping in cohorts of survivors (Solomon et al., 1988). From a clinical point of view, however, what is ultimately important is the degree to which coping efforts are successful. In other words, at the immediate aftermath of traumatization **the specific way by which the person copes with a stressor is often less important than the extent to which coping has been successful.**

According to Pearlin and Schooler (1978), successful coping must protect four vital functions: (a) the ability to **continue task-oriented activity**, (b) the ability to **regulate emotion**, (c) the ability to **sustain positive self value** and (d) the capacity to **maintain and enjoy rewarding interpersonal contacts**. Importantly, effective coping may be seen despite extreme misery and vice versa, poor coping may follow events that objectively appear to be quite mild.

Symptoms expressed following trauma.

General considerations

Symptoms of distress as an effective human behavior.

In their march towards recovery, trauma survivors express common responses that may enhance communication with others (e.g., by telling their story time and again); recruit support (e.g., by expressing a 'cry for help'), and effectively initiate a process of learning and reappraisal (by going back to memories of the traumatic event and

associating them with one's past experiences). The same expressions, however, may, in some cases, prevent communication (e.g., when telling the story if fearfully avoided or truncated) decrease the helping response of others and consolidate the link between traumatic memories and negative emotions. In other words the **effectiveness of expressed behavior** is most important (see assessment of coping below). This section outlines the three common patterns of the early responses to trauma.

Symptoms are complex and unstable

People react to traumatic events in many different ways. The intensity of fear response may vary from fight or flight to freezing and surrender. Bodily responses, such as increased heart rate are seen and may predict subsequent PTSD, (Shalev et al., 1998). Extreme psychological responses, such as dissociation disorientation and confusion may also be seen and require specific management (Marmar et al., 1994). Some trauma survivors look sad, exhausted and depressed. Physical injury, and distress of physiological origins (e.g., dehydration, hypothermia) are also seen and may be predictive of PTSD (Blanchard EB et al., 215-1934).

A study of combat stress reaction (CSR) in Israel found that symptoms expressed shortly after combat were "**polymorphous and labile**" (Yitzhaki et al., 1991). They included a mixture of exhaustion, stupefaction, sadness, anxiety, agitation and blunted affect. These symptoms varied rapidly with time and in response to external reality. Another longitudinal observation found that initial heroic response in some survivors, often fostered by exposure to the media or by the need to be there for others (Shalev, Schreiber and Galai, 1993). To the author of this chapter, such behavior suggests that the survivor is still 'within the trauma' and is using

extreme defenses to cope with the event. These initial responses, however, do not last and are quickly replaced by the unavoidable circle of intrusive and painful recollections of the traumatic event.

Symptoms are normally expressed, yet some are alarming

Some symptoms observed immediately following trauma are 'normal' in the sense of affecting most survivors, being socially acceptable, psychologically effective, and self limited. Yet other symptoms may be announcing trouble. Among these are symptoms of dissociation which, particularly when they repeat themselves following evacuation, should be seen as very alarming (Shalev et al., 1996); (Marmar et al., 1992-1994), (Eriksson & Lundin, 1996). Other symptoms may be predictive by their sheer intensity, yet studies have shown that the intensity of initial symptoms is not a specific predictor of PTSD (i.e., most people who express intense symptoms will still recover, (Shalev et al., 1997)). The opposite, however, is very true, i.e. survivors who do not express high degree of distress following traumatic events are more likely not to develop post-traumatic disorders. In other words, lack of significant distress has more predictive power than the presence of such symptoms.

Tolerance and communicability of symptoms

As mentioned above, a short period of shock and/or heroic defenses is regularly followed by repeated recollections of the traumatic event. Within this general pattern, some survivors are extremely disturbed while others are not. The intensity of intrusive recollections, for example, may be such that they are fearfully avoided, experienced as a torment, seriously interfere with sleep, curtail conversations about the traumatic

event, create a wall of silence and increase the survivors isolation and loneliness. In other cases, however, survivors use the intrusive recollections to repeatedly tell others about the traumatic event, and thereby recruit sympathy and help. Such redundant re-telling of the story is so frequent that it may be useful to educate primary care givers, such as nurses and family members, to be tolerant to and acceptant of hearing the same story again and again. A closer observation of the 'effective' re-telling of the story shows that its content changes with time, the narrative becomes richer, includes other elements and takes a reflective tone (e.g., "When I think about it now, I could have done worse"). Nightmares are often changing as well, from mere repetition of one instance of the traumatic event to more remote renditions of the event, associated with past events and with the person's total life experience. Such individuals may be on their way to recovery.

Specific syndromes

Acute Stress Disorder

DSM IV proposes a diagnostic category of acute stress disorder (ASD) with symptoms of PTSD (reexperiencing, avoidance and hyperarousal) occurring, along with dissociative symptoms within one month of the traumatic event. Symptoms of ASD may occur at any time (including during the traumatic event), should last for at least two days, cause clinically significant distress, significantly interfere with the individual's functioning or impair the individual's ability to pursue necessary tasks.

The presence of full or partial ASD may be associated with an increased risk of developing PTSD (Bryant & Harvey, 1995; Classen et al., 1990-1998; Classen et al., 1990-1998; North et al., 1982-1994). ASD has been linked with prior mental disorders (Barton et al., 1996).

However, many survivors without initial ASR develop PTSD as well. Specifically, a subset of ASR symptoms (numbing, depersonalization, a sense of reliving the trauma and motor restlessness) has been found to be strongly predictive of PTSD, while other symptoms, including most dissociative symptoms did not (Harvey AG & Bryant RA, 507-1998). Clearly, the presence of ASR signals a higher risk for developing PTSD, yet the validity of the currently-defined syndrome has been questioned.

Symptom criteria for Acute Stress Disorder

- 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 reexperienced in at least one of the following ways:
- recurrent images, thoughts, dreams, illusions, flashback episodes, or a sense of reliving the experience; or distress on exposure to reminders of the traumatic event.
- D. Marked avoidance of stimuli that arouse recollections of the trauma (e.g., thoughts, feelings, conversations, activities, places, people).

- E. Marked symptoms of anxiety or increased arousal (e.g., difficulty sleeping, irritability, poor concentration, hypervigilance, exaggerated startle response, motor restlessness),
- F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or impairs the individual’s ability to pursue some necessary task, such as obtaining necessary assistance or mobilizing personal resources by telling family members about the traumatic experience.
- G. The disturbance lasts for a minimum of 2 days and a maximum of 4 weeks and occurs within 4 weeks of the traumatic event.

Dissociation and depression

Symptoms of depression and dissociation have been in recent trauma survivors. Among injured survivors, dissociation during the traumatic event (peri-traumatic dissociation) was found to be significantly associated with the subsequent development of PTSD (Shalev et al., 1996). Holen ((Holen, 1991)) found that dissociation during the North Sea Oil Rig Disaster was significantly associated with the short-term psychological outcome of this event. Bremner et al.((Bremner et al., 1992)) found that Vietnam veterans with PTSD had experienced levels of dissociative symptoms during combat that are higher than those reported by veterans without PTSD. Koopman et al. (Koopman et al., 1994)) found that early dissociative symptoms, in survivors of the Oakland/Berkeley firestorm, predicted PTSD symptoms seven months later. Finally, Marmar et al (Marmar et al., 1994) have shown that peri-traumatic dissociation, in Vietnam veterans, contribute to current PTSD over and above the contribution of combat

exposure. Special emphasis should be given, therefore, to evaluating dissociative symptoms in the recent survivors.

Depression is often associated with chronic PTSD ((Kessler et al., 1995)), and might be an independent consequence of traumatic stress. Recent studies have shown that major depression occurs as early as one month following traumatization and that depressive symptoms one week following trauma and one month later predict chronic PTSD above and beyond prediction made from PTSD and dissociation symptoms (Freedman et al., 1999). While these data suggest that persistent depression in the weeks that follow trauma may require specific treatment intervention, not enough is known about the short term and long term effect of treating depression as such in the immediate aftermath of traumatic events.

Assessment and Evaluation

Given the problematic nature of assessing early symptom, this chapter offers two alternatives: First, to assess the **evolution of symptoms**. Second, to assess the degree to which **symptoms are tolerated** by the survivor and the degree to which the **intefere with normal functions** or tasks. Beyond assessing symptoms, it is necessary to go back and evaluate risk factors related to the traumatic event, loss and damage incurred, presence of secondary stressors, quality, intensity and development of the early responses and the availability of healing resources . A short overview of each of these domains follows.

The traumatic event

The assessment of the traumatic event should tell us what happened, what has made an impact on the survivor. This information is easily obtained: trauma survivors tend to narrate their experience, often repeatedly, provided

that confidence and safety are established. The survivor's story, however, may be incomplete, mixed up, or redundant. One should listen to it and appraise its **content and structure** without confronting the subject with inconsistencies or interpretations. The story told includes concrete description, subjective appraisal and emotional responses. As such it optimally represents the **psychological reality of the event** and the survivor's experience. Keeping a record of the initial narrative has tremendous value as testimony and for later ascertainment of facts and deeds.

Telling the story can be **stressful**, and is rarely without strong emotion. Telling the detail of the story to a helper is also **binding**, in that it creates an emotional bond between the narrator and the listener. Importantly, telling the story is an interaction, and good listeners are often those who respond emotionally while listening. Being in an interaction with survivors can be overwhelming for helpers, and requires peer support and opportunity to ventilate and share emotions.

Loss and damage suffered

Traumatic events are often associated with real and symbolic losses. Among the former are loss of life, injury, loss of property, relocation and loss of social network. Symbolic losses include loss of previously held beliefs and cognitive schemata, loss of one's identity, honor, peace of mind. These have been authoritatively described elsewhere (Janoff Bulman, 1989; Foa et al., 1989; McCann & Pearlman, 1992).

Traumatized survivors often describe a loss of continuity with their previous life, such as 'not being the same person any more'.

Facing losses may be the most distressing element of the immediate post-impact period of traumatic events. Importantly, recovery from loss involves

grieving and re-adaptation, that is, new learning about self and others. Pharmacological agents that interfere with learning (e.g., benzodiazepines) may prevent such learning, hence preliminary data on the negative effect of administering such drugs continuously to trauma survivors (Gelpin et al., 1996)

Secondary stressors

Traumatic events do not have a clear end point. Pain, uncertainty and series of surgical procedure may follow traumatic injury. This is especially true in burn victims (Taal & Faber, 288-1990). Rape may be followed by aggressive police interrogation. Disasters are often the prelude of prolonged relocation, separation and estrangement. Some survivors, therefore, are in the midst of continuous traumatization when helping efforts begin. We refer to these newly emerging adversities as secondary stressors.

Physiological stressors may often go undetected in the recent survivor. Classical pitfalls include internal bleeding (e.g., in badly beaten rape victim) mistaken for panic anxiety. Dehydration explained as mental confusion, or, vice versa, agitation taken to be a chemical intoxication (Ohbu S et al., 587-1993). Pain is a major secondary stressor, which had been linked with PTSD (Schreiber & Galai Gat,). Medical examination must, therefore, precede the psychological assessment when the physical conditions of the trauma can have caused a physical damage.

Countless psychological stressors may follow trauma, including bewilderment and disorientation, uncertainty about self and significant others, missing family members, exposure to disfigured bodies or to other people's agony during evacuation etc... Helpers may systematize the quest for such stressors by asking the following simple questions: Is the survivor secure and out

of danger? Does he or she have enough control of what is happening now? Are there major uncertainties in or around the patient's condition? Are negative events (or news) still expected? Does the patient have clear enough information about self and significant others? Has adequate human attention and warmth been given to the patient? Has trust been established between patient and helpers? Can the current conditions humiliate or dishonor the patient?

Assessment of Secondary Stressors

Is the survivor secure and out of danger?

Does he or she have enough control of what is happening?

Are there major uncertainties in the present?

Are negative events (or news) still expected?

Does the survivor have clear enough information about self and significant others?

Has adequate human attention and warmth been given to the survivor?

Has trust been established between survivors and helpers?

Can the current conditions humiliate or dishonor the survivors?

Evaluating coping efficacy

The assessment of coping efficacy takes the inquiry from the purely medical arena to the more general way of evaluating human performance and emotion. It will often be found that such line of inquiry provides clinically relevant information. Four dimensions of successful coping have been outlined above. Failure to cope may, accordingly, results in one or more of the following: Impaired task performance (e.g., work, care of children, getting help!) Poorly modulated emotions (e.g., fear, sadness, anger). Negative self-perception (e.g., self-accusation, self - devaluation).

Inability to enjoy rewarding interaction with others (including inability to be helped).

The clinician may wish to assess coping behavior during the traumatic event or current coping efficacy. The following questions may be of help in the assessment of global coping efficacy.

Assessment of coping efficacy

Can the survivor continue task-oriented activity? How well organized, goal directed and effective is such activity?

Is the survivor overwhelmed by strong emotions most of the time? Can emotions be modulated when such modulation is required?

Is the survivor inappropriately blaming himself or herself? Does the survivor generalize such accusations to his or her personality or self.

How isolated, alienated or withdrawn is the survivor. Does he seek the company of others or rather avoid it?

Interventions

Few individuals can pull themselves out of traumatic occurrences by their own will and power. The general case is that trauma survivors need help and support from others, and sometimes from those professionally trained to help. The latter often refer to their way of helping others as being a 'treatment.' In order to be effective, however, such 'treatment interventions' must firstly meet the survivor's needs. Those who provide treatment must be tolerant of symptomatic behavior and respect the person's ability to self-regulate and monitor his or her environment. Importantly, the survivor must be able to properly utilize and enjoy what is offered. Stress responses may reduce such capacity, yet social or cultural mismatch between helpers and survivors may also reduce the usefulness of

'treatment.' Interventions should, therefore be tuned to needs, capacities, and desires of survivors.

Generic goals of early interventions

The main goals of early interventions are the following: First, to reduce psychobiological distress, which seems to be strongly related to subsequent psychopathology. Within this first goal, reducing the effect of secondary stressors is often the precondition for conducting other interventions. Second, to treat specific symptoms when they interfere with normal healing processes, i.e., sharing and assimilating the traumatic event. Third, because situations of extreme stress are followed by natural healing processes, the best that therapy can do is to assist the normal healing, by supporting the survivor and his or her immediate helpers, by seeing that such helpers are available, that families are evacuated together etc.... In terms of assessment and evaluation, the main goal of the assessment, at this stage is to follow progress. Assessment of global coping efficacy functions may be a useful tool to follow such progress.

Interventions in the different phases of the acute response

Peri-traumatic period

The goal of interventions, during this stage, is to protect the survivors from further exposure to stress, contain the immediate physiological and psychological responses and increase controllability of the event and of subsequent rescue efforts.

Mental health professionals do not conduct most interventions at this stage. Yet, some psychological principles apply. Firstly, to maintain human contact with survivors throughout rescue efforts. Within human contacts, one should remember that survivors might have difficulties to verbally express their experience, whereas other bodily and emotional channels are open for

communication. Helpers should be tuned and responsive to the survivor's attempts to gain a degree of comfort and dignity during the event (e.g., by covering his or her body, avoiding intrusive looks of others and of the media).

Second, one should not spare efforts to soothe and comfort a recent survivor. Soothing bodily contacts with the recently rescued are often of great help, yet gender and social boundaries must be respected. Bringing in natural helpers (e.g., relatives) and helping them by advice and orientation may be of great help.

In order to increase controllability and reduce the unexpected one should re-orient the survivor within the rescuing environment. Rescuer should clearly identify themselves and their role. They should continuously inform the survivors about steps to be taken (e.g., evacuation to a hospital) medication given (e.g., morphine) and other information. Whilst genuine information (including admitting lack of information) must be the rule, breaking bad news may not be the rescuer's primary goal. A link between survivors from the same family should be established and, if possible, survivors should be evacuated along with those to whom they are close.

Finally, at this phase, those who participate in rescue effort are also at risk for developing stress responses. Excessive self exposure (i.e., being unable to disengage from work), irritability, inability to relax, difficulties communicating with others are warning signs of burn-out. A perception of having failed (e.g., by not preventing death or injury) are particularly poignant. Monitoring rescuers exposure, securing and ordering resting periods, relieving overburdened workers and conducting debriefing may help reduce the effect of traumatic stressors on rescuers.

Immediate early responses

Shortly after exposure, the traumatic event ceases to be concrete event and starts to become a psychological event. As such, it has to be metabolized and assimilated, that is, become part of the survivor's inner network of meanings and experiences. Assimilating a traumatic event is often very painful, and involves repeated recall and reassessment of the traumatic event, and progressive assimilation. Progressive sensitization may also occur, in which aversive responses increase and are generalized to other psychological and social domains. The task of early interventions is, therefore, to facilitate psychological recovery and disable progressive sensitization.

Several mediators of recovery are well known at this point. These include verbalizing and sharing the individual story with others; being able to endure and express painful emotions and oscillating between periods of extreme anguish and relative rest. Continuous distress, turning one's back to others, and being unable to think about the trauma (but rather experience it) are symptoms of bad processing.

A professional therapist is not needed when proper processing of the traumatic event takes place. Hence the first role of a therapist is to assess the strengths and the weakness of the survivor's immediate supporters.

One should allow for specific recovery styles to develop in individuals and families (one may talk and another may be silent). In some cases it is important to educate and explain saliency of symptoms to survivors and their helpers. Importantly one is to follow success or failure of recovery style

Emergence of specific symptoms

An interesting observation shows that burn victims as well as other injured trauma survivors (Baur et al., 1998;

Shalev, Schreiber and Galai, 1993) become more symptomatic as they prepare to leave the hospital. Fully expressed phobic responses, major depression and acute PTSD may, indeed, characterize the period of re-entry to life, possibly because they start to interfere with normal tasks. At this point in time dedicated interventions are called for, including specific therapies (see below) and pharmacotherapy. One should be aware, however, that recovery is still the most frequent outcome, and that one is not yet treating a chronic post-traumatic stress disorder.

Specific techniques

Despite the rich history of attempts to treat the immediate survivor, evidence regarding the long-term effect of early interventions is missing. The following are, therefore, descriptions of methods used, in the past, to treat the immediate response to traumatization. Importantly, lack of empirical data, in this area, should not be confounded with negative results. Indeed the field is lacking prospective evaluations of trauma survivors, which could definitely tell us what is the best to do.

Crisis interventions and stress management

Stress theory suggests that the more distressed an individual is, the less capable he is to mentally disengage from the situation, reflect, imagine, and create solutions. Moreover, there is tendency to repeat sterile attempts to solve a problem, without changing them. Crisis interventions attempt to stop the vicious circle of catastrophic appraisal and extreme distress. They also address the perception, by those in crisis, that their reaction is abnormal or that they have totally lost their inner strength.

In the recent survivor crisis interventions address those elements of the short - term response to trauma that may not work effectively because of excessive distress. The situation of

crisis is perceived as one in which the individual is caught in an emotional and cognitive trap, hence a 'crisis.' Crisis situations are emotionally overwhelming and cognitively inescapable. The survivor, accordingly, does not engage in effective salutary efforts, and can not perceive a solution to the situation, often despite adequate resources.

The combination of extreme distress and blindness to solutions is intolerable to most individuals, and may result in unexpected behavior (e.g., suicide; life-threatening bravery). Statements like 'all is lost,' 'there is no way out' and 'I can't tolerate it for another minute' are typical expressions of crises.

Crisis interventions starts by appraising with the individual (or the group) what, in a given situation, creates intolerable distress. It is often found that one specific element of the whole situation (e.g., lost communication with a family member) is most distressful. The second step is to recognize, legitimize yet smoothly challenge the perceived totality of the situation. Once this is clarified, one may address efforts already made to solve the salient problem, and assess other ways or other resources. It is often found that once extreme emotions subside, individuals may find better solutions than expected. Solutions may include alternative plan of action, effective help-seeking, or postponing efforts to find a solution and engaging in alternative goals (e.g., "meanwhile I also have to take care of my children"). Moving subjects from a stage of disarray to a stage of effective coping signals the success of crisis intervention.

Treatment of combat stress reaction (CSR) within the military

Combat stress disorder (CSR) and its treatment have been authoritatively discussed elsewhere (e.g., Solomon, 1993). Importantly, the goals assigned to the treatment of combat soldiers has

never been purely medical, and often included other considerations, such as reducing manpower loss due to psychological reactions. Within this dual goal, a strategy of frontline treatment has often been used. Otherwise known as the PIE model (PIE for proximity, immediacy and expectations), this treatment approach insisted on treating CSR casualties as soon as possible, as near as possible to the frontline, and with an expectation of recovery and return to duty. The content of PIE interventions varied, going from a minimalist approach (known as the ‘chicken soup’ treatment’) in which protection, shelter and respite were the main elements, to more active supportive group therapies.

Several controversial elements of the PIE model may still be in the background of one’s mind, when it comes to treat the acutely traumatized. First, one may be tempted to assume that in and of itself, an early return to ‘duty’ or to full performance is salutary. Accordingly, a traumatized police officer may be immediately sent to another stressful task, as part of his or her ‘therapy.’ It is important to stress that the effectiveness of such practice has not been confirmed by studies of CSR. Quite to the contrary, the extensive ‘return to duty’ policy, employed by US psychiatrists in Vietnam (e.g., Bourne, 1978) does not seem to have prevented the occurrence of PTSD. The Israeli experience, in contrast, seems to show that the implementation of the PIE model was a success (Solomon & Benbenishty, 1986). It is important to remember, however, that all Israeli wars were short (e.g., three weeks of fighting during the 1982 Lebanon war). Those who returned to their units, therefore did not have to face heavy combat.

Another problematic concept is the so called ‘labeling’ theory, according to which making a diagnosis or otherwise telling survivors that they have a problem (e.g., by sending them back to a

treatment facility) may be pathogenic. Ignoring an existing problem, however, may not be better. Between refusal to recognize that one is ill and over-dramatizing one’s malady, clinicians must find a balanced path. Indeed what seems to work in the frontline treatment is a natural selection process by which those who recover within the time allocated to staying in a frontline facility may go back to their previous role, while those with persistent reactions are evacuated to the rear. In general, therefore, it may be wise to support the survivors in the immediate aftermath of traumatization and follow his or her progress before making a final assessment of severity.

Brief cognitive interventions

Cognitive behavioral therapy (CBT) is an effective treatment for prolonged PTSD (see Foa, this volume). In the more recently traumatized, CBT has been evaluated in two controlled trials: Foa, Hearst-Ikeda and Perry (Foa et al., 1995) evaluated a brief prevention program comprised of 4 sessions for female victims of sexual and non-sexual assault. The intervention group was matched with a group of victims seen previously. At two months follow-up the intervention group had a 10% rate of PTSD compared to 70% in the control group. Bryant et al., 1998 compared five sessions of CBT with sessions of supportive counseling in ASD (Bryant et al., 1998). Seventeen percent of CBT patients and 67 percent of supportive counseling patient had PTSD at six months. Echarburua et al (Echeburua et al.,) similarly showed that cognitive restructuring and specific coping skills training was superior to relaxation training in reducing PTSD symptoms (but not other symptoms) at 12 months. CBT-derived interventions, therefore, seem to be useful for acute stress disorder, i.e., at the stage of early syndrome formation. The current

literature, however, provides no comparison between CBT and other active approaches (e.g., pharmacotherapy, intensive interpersonal psychotherapy). Hence, it would be inappropriate at this stage to say that **only** CBT works.

Debriefing

Debriefing has been developed as semi-structured group intervention designed to alleviate initial distress and prevent the development of mental disorders following exposure to traumatic events [Mitchell 1983; Raphael, 1986; and Dyregrov, 1989]. Debriefing usually consists of one session, during which the participants of an event share and learn from their experiences.

Among debriefing protocols, Mitchell's Critical Incident Stress Debriefing (CISD), and a more recent version, Dyregrov's (1989) Psychological Debriefing, have been largely implemented. CISD sessions include seven consecutive stages: Introduction: where the purpose of the session and its rules are described. A Fact phase: which consists of describing the traumatic event. A thought phase addresses the appraisal of the event. A reaction phase explores the participants' emotion during and after the event. A Symptom phase discusses the normal nature of symptoms. A teaching phase prepares for future developments, and outlines way of coping with further consequences of the traumatic events. A re-entry or disengagement phase offers a general discussion of the session and practical conclusions. The length of the sessions may vary. The same series of consecutive steps has been used in individual cases as well.

Debriefing sessions were expected to accomplish a lot. Among others they include reviewing the facts, sharing emotions, validating individual experiences, learning coping skills, evaluating current symptoms, and

preparing for future experience. The long-term goal of debriefing, which is the prevention of stress disorders, is also very ambitious. As shown in the table below, however, controlled follow-up studies have not shown that debriefing has such capacity ((Bisson et al., 1997), (Hobbs et al., 1438-1996), (Bisson & Deahl, 1994), (Deahl et al., 1994) Lee et al., 1996). Notwithstanding, the same studies show that most survivors perceived debriefing sessions as beneficial and satisfying. Moreover, debriefing has been shown to significantly reduce concurrent distress and enhance group cohesion (Shalev et al., 1998)). It is, therefore, too early to conclude that debriefing is of no use, mostly because no study has evaluated debriefing in a context of continuous care. What one may conclude, however, is that single, one-session interventions are not enough to stop the causation of stress disorders

Author	Type of Trauma (n)	Time	Method	Outcome measure	Results
Bisson et al 1997	Burn Trauma (46/57)	2-19 days	Individual 'debriefing' /no interventions	IES, HADS, CAPS	Negative effect at 13 months <ul style="list-style-type: none"> • Satisfaction in 52% of survivors
Hobbs et al 1996	MVA victims (52/54)	<2 days	Individual 'debriefing' / Standard Care	IES, BSI at four months	Worse outcome following interventions
Lee et al., 1996	Miscarriage (21/19)	2 Weeks	Individual 'debriefing' /no intervention	IES & HADS at four months	No sig. effect by psychometrics <ul style="list-style-type: none"> • 'helpful' by clients' judgement
Deahl et al., 1994	Body handlers (20/40)	Misc.	Group debriefing	IES, GHQ-28 at nine months	No effect

Pharmacological interventions

Very little is known about the pharmacotherapy of recent trauma survivors. As with other forms of therapy, pharmacotherapy has two distinct targets: control of current distress and prevention of subsequent stress disorders. The two targets are often complementary, but may also be contradictory (see discussion of benzodiazepines, below).

Current empirical research assigns two specific targets for early pharmacological treatment: hyperarousal (including sleep disturbances) and depression. As discussed above, hyperarousal in the days and weeks following trauma predicts subsequent PTSD. Depressive symptoms, one week after trauma, were also found to predict PTSD at four months and one year (Freedman, 1999). The role of anxiolytics and anti depressants is therefore discussed first

Anxiolytics and Sedatives

Anxiolytics (mostly benzodiazepines) can reduce anxiety and improve sleep in recent trauma survivors. Their use, however, should be monitored: A study of prolonged treatment by high potency benzodiazepines in recent trauma survivors (2 to 18 days following trauma) has shown that these drugs were associated with higher incidence of PTSD at six months (Gelpin et al., 1996). On the other hand Mellman et al., have administered a benzodiazepine hypnotic for five nights to recent (between one and three weeks) trauma survivors and found an improvement in sleep and PTSD symptoms (Mellman et al., 563-1998). The use of sedatives, therefore, should have specific target (e.g., sleep, control of panic attacks), and should be time limited

Anti depressants

As shown by recent studies, chronic PTSD symptoms may be reduced by

antidepressants (see relevant chapter in this volume). Moreover, the effect of antidepressants may be greater in individuals with recent PTSD (van der Kolk et al., 1994). Given the above-mentioned association between early depression and PTSD (Freedman et al., 1999), it is not unreasonable to use antidepressants in the treatment of post-traumatic depression. Among antidepressants, one may prefer those that have been shown to affect chronic PTSD. Importantly, patients should be followed and the effect of antidepressants monitored. Treatment may be started during the first weeks following trauma. When depression is not present, however, the rationale for using antidepressants is not very strong, yet these drugs have been shown to affect core PTSD symptoms in patients with prolonged disorders. Clearly, antidepressant treatment should be reserved to the period in which specific syndromes are expressed, and is not justified, currently, as treatment of the immediate response.

Future and hypothetical approaches

Current theories of causation assign several additional and yet hypothetical targets. First, PTSD may result from neuronal kindling (Post et al., 285-1995), hence the hypothetical usefulness of anti kindling drugs, such as valproate, in the early aftermath of traumatization. Second, PTSD has been associated with sympathetic activation during and immediately following trauma (Shalev et al., 1998). Sympathetic activation has also been associated with enhanced recall of negative emotions. Hypothetically, therefore, drugs that reduce sympathetic activation, such as propranolol, have been proposed as immediate preventive treatment in recent trauma survivors. The administration of

propranolol should start as soon as possible following trauma, i.e., upon admission to an emergency room. Third, PTSD has been associated with a cascade of biological events related to the activation of corticotropin release hormone (CRH) receptors in central brain nuclei. Modulators of CRH activity may become available, and used in the future for the prevention of PTSD. Finally, chronic PTSD has been associated with cortisol-related damage to hippocampal cells. Several compounds were found to protect the hippocampus from cortisol-related cellular damage, and these drugs may be of use in PTSD as well.

Conclusion

Early interventions following trauma are not easy to conduct. Controlled prospective studies of such interventions are even more difficult, and raise both practical and ethical problems. Consequently, the uncertainty about the long-term effect of early interventions is likely to remain. Lessons from the recent and not so recent past do not show that early interventions can prevent PTSD. Such is the case of the Vietnam and the Lebanon wars, in both of which the management of combat stress responses was seen as important service to combatants. Such is also the case of most studies of debriefing, as cited above.

There are multiple reasons for such unfortunate results, first among which is, probably, the complex etiology of PTSD, which encompasses biological endowment, acquired vulnerability, intensity of traumatization and recovery factors. Within such complex etiology, the relative contribution of early and short interventions is necessarily small. Other reasons to believe that the task of preventing stress disorders is, indeed,

tremendous have been outlined in this chapter. These include the plasticity of the early reaction, the admixture of normal and abnormal behavior, the difficulties to identify subjects at risk and the proper difficulties of conducting interventions in early aftermath of disastrous events.

Yet, knowledge cumulated so far leads to narrowing the above mentioned uncertainties. First, convincing success of some interventions has been documented recently, mainly through the use of cognitive behavioral techniques. This is an important step forward which should be followed. Central to pursuing this venue is the question of timing of the intervention. Studies of CBT have been conducted in the weeks following traumatic events, and not during the first few days. Possibly other treatment methods may be useful at that stage, such as treatment for depression, which has been shown to strongly predict PTSD.

If effective treatment can be administered during the early post-traumatic period, then it is mandatory to deliver it to symptomatic survivors. What the immediate contact can provide the survivor with, is an open door (or address) for continuous treatment and the ability to identify oneself as being in need for treatment. Interventions during the acute aftermath of traumatization, therefore, should end by advising survivors about possible sources of help, and about self-diagnosis. Additionally, survivors should be systematically evaluated at the end of treatment, such that those who are abnormally symptomatic will have knowledge of their situation. What are 'abnormal symptoms' is obviously a matter of clinical judgement. The coping model proposed above, or indeed any measure

of the interference of symptoms with expected performance may facilitate such judgement.

As to the earlier stages of the immediate response to traumatic events, the following sequence suggests a general framework. At first provide concrete help, food, warmth and shelter. Once out of concrete danger, immediate treatment is mainly by soothing and reducing states of extreme emotion and increasing controllability. At a third stage survivors have to be assisted in the painful and repetitive re-appraisal of the trauma. The appropriate level of clinical observation and decision making, at this stage, may be the salient symptom (e.g., pain, insomnia). Treating specific syndromes comes at the following stage, when syndromes can be reliably diagnosed and followed. Acute PTSD, depression, and possibly other anxiety disorders are the main targets for treatment at this stage.

Finally, one should look forward to further discovery in the area of early responses and their treatment. Studies of chemical agents (e.g., propranolol) are now on their way. Attention given to traumatization may lead to trials of additional immediate therapies. Better understanding of predictors and risk factors for chronic disorders may enable more valid diagnostic routines in the future. Yet, preventing stress disorders is only one goal of early interventions. Providing immediate relief is not a lesser task. Possibly the two should not be confounded, and one should help those who suffer at the level of their immediate human and clinical needs, without pretending, yet while hoping to prevent chronic stress disorders.

References

- Antelman, S. M. (1988) Time-dependent sensitization as the cornerstone for a new approach to pharmacotherapy: drugs as foreign/stressful stimuli. *Drug Development Research*, 14: 1-30. NT Review Article: 176 Refs..
- Barton, K. A., Blanchard, E. B., & Hickling, E. J. (1996). Antecedents and consequences of acute stress disorder among motor vehicle accident victims. *Behav.Res.Ther.*, 34, 805-813.
- Baur, K. M., Hardy, P. E., & Van Dorsten, B. (1998). Posttraumatic stress disorder in burn populations: a critical review of the literature. *J-Burn-Care-Rehabil*, 19, 230-240.
- Bisson, J. I., & Deahl, M. P. (1994). Psychological Debriefing and Prevention of Post-Traumatic Stress - More Research is Needed. *British Journal of Psychiatry*, 165: 717-720.
- Bisson, J. I., Jenkins, P. L., Alexander, J., & Bannister, C. (1997). Randomised controlled trial of psychological debriefing for victims of acute burn trauma. *Br.J.Psychiatry*, 171, 78-81.
- Blanchard EB, Hickling EJ, Forneris CA, Taylor AE, Buckley TC, Loos WR, & Jaccard J. (1997). Prediction of remission of acute posttraumatic stress disorder in motor vehicle accident victims. *J-Trauma-Stress*, 10: 215-234.
- Bland, S. H., O'Leary, E. S., Farinaro, E., Jossa, F., Krogh, V., Violanti, J. M., & Trevisan, M. (1997). Social network disturbances and psychological distress following earthquake evacuation. *J-Nerv-Ment-Dis*, 185: 188-194.
- Borrell J, de Kloet ER, Bohus B (1984). Corticosterone decreases the efficacy of adrenaline to affect passive avoidance retention of adrenalectomized rats. *Life Sciences* 34:99-103
- Bourne, P.G. (1978) Military psychiatry and the Viet Nam war in perspective. *In The Psychology and Physiology of Stress*. Bourne P.G, Editor: pp. 219-236, Academic Press, New York, NY
- Bremner, J. D., Southwick, S., Brett, E., Fontana, A., Rosenheck, R., & Charney, D. S. (1992). Dissociation and posttraumatic stress disorder in Vietnam combat veterans. *Am.J.Psychiatry*, 149, 328-332.
- Breslau, N., & Davis, G. C. (1992). Posttraumatic stress disorder in an urban population of young adults: risk factors for chronicity. *Am.J.Psychiatry*, 149, 671-675.
- Bryant, R. A., & Harvey, A. G. (1998). Relationship between acute stress disorder and posttraumatic stress disorder following mild traumatic brain injury. *Am-J-Psychiatry*, 155: 625-629
- Bryant, R. A., Harvey, A. G., Dang, S. T., Sackville, T., & Basten, C. (1998). Treatment of acute stress disorder: a comparison of cognitive-behavioral therapy and supportive counseling. *Journal of Consulting and Clinical Psychology*, 66(5), 862-866.

- Bunn, T., & Clarke, A., (1979) Crisis Intervention: An Experimental Study of the Effects of a Brief Period of Counselling on the Anxiety of Relatives of Seriously Injured or Ill Hospital Patients. *British Journal of Medical Psychology* 52, 191-195.
- Cahill, L., Prins, B., Weber, M., & McGaugh, J. L. (1994). Beta-Adrenergic activation and memory for emotional events. *Nature*, 371, 702-703.
- Classen, C., Koopman, C., Hales, R., & Spiegel, D. (1998). Acute stress disorder as a predictor of posttraumatic stress symptoms. *Am-J-Psychiatry*, 155: 620-624
- Dasberg H. Belonging and loneliness in relation to mental breakdown in battle. *Israel Annals Psychiatry Relat Sciences* 1976;14: 307-321.
- Deahl, M. P., Gillham, A. B., Thomas, J., Searle, M. M., & Srinivasan, M. (1994). Psychological sequelae following the Gulf War. Factors associated with subsequent morbidity and the effectiveness of psychological debriefing. *Br.J.Psychiatry*, 165, 60-65.
- Dyregrov, A., (1989) Caring for Helpers in Disaster Situations: Psychological Debriefing. *Disaster Management* 2, 25-30.
- Echeburua, E., De Corral, P., Sarasua, B., & Zubizarreta, I. (1996). Treatment of acute posttraumatic stress disorder in rape victims: an experimental study. *Journal of Anxiety Disorders* 10: 185-199
- Ehlers, A., Clark, D. M., Dunmore, E., Jaycox, L., Meadows, E., & Foa, E. B. (1998). Predicting response to exposure treatment in PTSD: the role of mental defeat and alienation. *J-Trauma-Stress*, 11:457-471.
- Ehlers, A., Mayou, R. A., & Bryant, B. (1998). Psychological predictors of chronic posttraumatic stress disorder after motor vehicle accidents. *J-Abnorm-Psychol*, 107:508-519.
- Eriksson, N. G., & Lundin, T. (1996). Early traumatic stress reactions among Swedish survivors of the m/s Estonia disaster. *Br.J.Psychiatry*, 169, 713-716.
- Foa, E. B., Hearst Ikeda, D., & Perry, K. J. (1995). Evaluation of a brief cognitive-behavioral program for the prevention of chronic PTSD in recent assault victims. *J.Consult.Clin.Psychol.*, 63, 948-955.
- Foa, E. B., Steketee, G., & Rothbaum, B. O. (1989). Behavioral/cognitive conceptualizations of post-traumatic stress disorder. *Behav Therapy*, 20:155-176.
- Foa, E. B., Zinbarg, R., & Olasov Rothbaum, B. (1992). Uncontrollability and unpredictability in post-traumatic stress disorder: an animal model. *Psychological Bulletin* 112:218-238
- Freedman SA, Peri T, Brandes D, Shalev AY. Predictors of chronic PTSD - A prospective study. *Br. J Psychiatry*, 174:, 353-359, 1999
- Gelpin, E., Bonne, O., Peri, T., & Brandes, D. (1996). Treatment of

- recent trauma survivors with benzodiazepines: A prospective study. *J Clin Psychiatry*, 57:390-394
- Green, B. L. (1995). Defining trauma: terminology and generic stressors dimensions. *J.of.Applied.Social.Psychology*. 20, 1632-1642
- Harvey AG, & Bryant RA. (507-1998). The relationship between acute stress disorder and posttraumatic stress disorder: a prospective evaluation of motor vehicle accident survivors. *J-Consult-Clin-Psychol*, 66: 507-512.
- Hobbs, M., Mayou, R., Harrison, B., & Worlock, P. (1996). A randomised controlled trial of psychological debriefing for victims of road traffic accidents. 313:1438-1439
- Hobfoll, S. E., & Jackson, A. P. (1991). Conservation of resources in community intervention. *Am-J-Community-Psychol*, 19, 111-21.
- Holen A: the North Sea Oil Rig Disaster, in *International Handbook of Traumatic Stress Syndromes*. Edited by Wilson JP, Raphael B. New York Plenum, 1993
- Janoff Bulman, R. (1989). Assumptive worlds and the stress of traumatic events: Applications of the schema construct. *Social Cognition*, -136.
- Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C. B. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Arch.Gen.Psychiatry*, 52, 1048-1060.
- Koopman, C., Classen, C., & Spiegel, D. (1994). Predictors of posttraumatic stress symptoms among survivors of the Oakland/Berkeley, Calif., firestorm. *Am.J.Psychiatry*, 151, 888-894.
- Kormos HR: The nature of combat stress. in *Combat stress disorders among Vietnam veterans* Edited by C. Figley, New York, Brunner Mazel, 1978, pp. 3-22
- Kozaric Kovacic, D., Folnegovic Smalc, V., & Marusic, A (1998) Acute post-traumatic stress disorder in prisoners of war released from detention camps. *Drustvena Istrazivanja* 7: 485-497 (1998)..
- Lazarus, R. S., Folkman, S. (1984) *Stress, Appraisal and Coping*. Chapter 2: Cognitive Appraisal Processes. Springer, New York
- Lee, C., Slade, P., & Lygo, V., (1996) The Influence of Psychological Debriefing on Emotional Adaption in Women following Early Miscarriage: A Preliminary Study *British Journal of Medical Psychology* 69, 47-58.
- Lindemann E. (1994). Symptomatology and management of acute grief. 1944 [classical article]. *Am-J-Psychiatry*, 151, 155-60.
- Loftus, E. F. (1993). The Reality of Repressed Memories. *American.Psychologist*., 48, 518-537.
- Marmar, C. R., Weiss, D. S., Schlenger, W. E., Fairbank, J. A., Jordan, B. K., & Kulka, R. A. (1994). Peritraumatic dissociation and

- posttraumatic stress in male Vietnam theater. *Am.J.Psychiatry*, 151, 902-907.
- Marmar, C.R., Weiss, D.S., Schlenger, W.E., Fairbank, J.A., Jordan, K., Kulka, R.A., Hough, R.L. (1994) Peritraumatic Dissociation and Posttraumatic Stress in Male Vietnam Theater Veterans. *American Journal of Psychiatry*, 151, 902-907.
- McCann, I. L., & Pearlman, L. A. (1992). Constructivist self-development theory: a theoretical framework for assessing and treating traumatized college students. *J.Am.Coll.Health*, 40, 189-196.
- McCarroll, J. E., Ursano, R. J., & Fullerton, C. S. (1995). Symptoms of PTSD following recovery of war dead: 13-15-month follow-up. *Am.J.Psychiatry*, 152, 939-941.
- McGaugh, J. L., 1990. Significance and remembrance: the role of neuromodulatory systems. *Psychological Science*. 1:15-25.
- Mellman, T. A., Byers, P. M., & Augenstein, J. S. (1998). Pilot evaluation of hypnotic medication during acute traumatic stress response. *J-Trauma-Stress*, 11: 563-569
- Metcalfe J. and W. J. Jacobs, 1996. A "hot-system/cool-system" view of memory under stress. *PTSD Research Quarterly*. 7:1-3.
- Mitchell, J.T., (1983) When Disaster Strikes.... *Journal of Emergency Medical Services* 8, 36-39.
- Munch A, Guyere PM, Holbrock MJ. (1984) Physiological functions of glucocorticoids in stress and their relations to pharmacological actions. *Endocr Rev*, 93:9783-9799.
- North CS, Smith EM, & Spitznagel EL. (1994) Posttraumatic stress disorder in survivors of a mass shooting. *Am-J-Psychiatry*, 151: 82-88.
- Ohbu S, Yamashina A, Takasu N, Yamaguchi T, Murai T, Nakano K, Matsui Y, Mikami R, Sakurai K, & Hinohara S. (1993). Sarin poisoning on Tokyo subway. *South-Med-J*, 90:587-593.
- Pearlin, L.I., Schooler, C. (1978). The structure of coping. *Journal of Health and Social Behavior*, 22, 337-356.
- Pitman, R. K. (1988). Post-traumatic stress disorder, conditioning, and network theory. *Psychiatric Annals*, 18, 182-189.
- Post, R. M., Weiss, S. R., Smith, M., Li, H., & McCann, U. (1997). Kindling versus quenching. Implications for the evolution and treatment of posttraumatic stress disorder. *Ann-N-Y-Acad-Sci*, 821: 285-95
- Prince CR, & Anisman H. (1990). Situation specific effects of stressor controllability on plasma corticosterone changes in mice. *Pharmacol-Biochem-Behav*, 37, 613-621.
- Raphael, B. (1986) When Disaster Strikes. Basic Books, New York pp. 222-244.

- Raphael, B., Meldrum, L., & McFarlane, A. C. (1996). Does debriefing after psychological trauma work? Time for randomised controlled trials. *Accid. Emerg. Nurs.*, 4, 65-67.
- Raphael, B., Meldrum, L., & McFarlane, A.C., (1995) Does Debriefing after Psychological Trauma Work? *BMJ* 310:1479-1480.
- Resnick, H. S., Yehuda, R., Pitman, R. K., & Foy, D. W. (1995). Effect of previous trauma on acute plasma cortisol level following rape. *Am.J.Psychiatry*, 152, 1675-1677.
- Rothbaum BO, Foa EB: Subtypes of posttraumatic stress disorder and duration of symptoms, In *Posttraumatic Stress Disorder, DSM IV and Beyond*. Edited by JRT Davidson and EB Foa, Washington, D.C., American Psychiatric Press, 1993, pp. 23-35.
- Schreiber, S., & Galai Gat, T. (1993) Uncontrolled pain following physical injury as the core-trauma in post-traumatic stress disorder. *Pain* 54: 107-110
- Shalev AY, Pitman RK, Orr SP, Peri T, Brandes D (in press) Auditory Startle in Trauma Survivors with PTSD: A prospective study. *Am J of Psychiatry*
- Shalev AY, Schreiber S, & Galai T. Early psychiatric responses to traumatic injury. *Journal of Traumatic Stress* 6:441-450, 1993
- Shalev AY. (1999) Psychophysiological risk-factors for PTSD. in Yehuda R (editor) *Risk Factors for PTSD*, American Psychiatric Press, Washington D.C.
- Shalev, A. Y., Freedman, S., Peri, T., Brandes, D., & Sahar, T. (1997). Predicting PTSD in trauma survivors: prospective evaluation of self-report and clinician-administered instruments. *Br.J.Psychiatry*, 170, 558-564.
- Shalev, A. Y., Peri, T., Canetti, L., & Schreiber, S. (1996). Predictors of PTSD in injured trauma survivors: a prospective study. *Am.J.Psychiatry*, 153, 219-225.
- Shalev, A. Y., Peri, T., Rogel Fuchs, Y., Ursano, R. J., & Marlowe, D. (1998). Historical group debriefing after combat exposure. *Mil Med*, 163(7), 494-8.
- Shalev, A. Y., Rogel Fuchs, Y., & Pitman, R. K. (1992). Conditioned fear and psychological trauma. *Biol.Psychiatry*, 31, 863-865.
- Shalev, A. Y., Sahar, T., Freedman, S., Peri, T., Glick, N., Brandes, D., Orr, S. P., & Pitman, R. K. (1998). A prospective study of heart rate response following trauma and the subsequent development of posttraumatic stress disorder. *Arch-Gen-Psychiatry*, 55(553-559).
- Solomon Z. (1993) *Combat Stress Reaction* Plenum, New York
- Solomon, Z., & Benbenishty, R. (1986). The role of proximity, immediacy, and expectancy in frontline treatment of combat stress reaction among Israelis in the Lebanon War. *American Journal of Psychiatry*, 143, 613-617.

- Solomon, Z., Mikulincer, M., & Avitzur, E. (1988). Coping, locus of control, social support, and combat-related posttraumatic stress disorder: A prospective study. *Journal of Personality and Social Psychology*, 55, 279-285.
- Taal, L. A., & Faber, A. W. (1997). Burn injuries, pain and distress: exploring the role of stress symptomatology. *Burns*, 23: 288-290.
- van der Kolk, B. A., Dreyfuss, D., Michaels, M., Shera, D., Berkowitz, R., Fislser, R., & Saxe, G. (1994). Fluoxetine in Posttraumatic Stress Disorder. *Journal of Clinical Psychiatry*, 55, 517-522.
- Yehuda, R., & McFarlane, A. C. (1995). Conflict between current knowledge about posttraumatic stress disorder and its original conceptual basis. *Am.J.Psychiatry*, 152, 1705-1713.
- Yehuda, R., McFarlane, A. C., & Shalev, A. Y. (1998) Predicting the development of posttraumatic stress disorder from the acute response to a traumatic event. *Biological Psychiatry* 44: 1305-1313
- Yehuda, R., Southwick, S. M., Nussbaum, G., Wahby, V., Giller, E. L. J., & Mason, J. W. (1990). Low urinary cortisol excretion in patients with posttraumatic stress disorder. *J.Nerv.Ment.Dis.*, 178, 366-369.
- Yitzhaki, T., Solomon, Z., & Kotler, M. (1991). The clinical picture of acute combat stress reaction among Israeli soldiers in the 1982 Lebanon war. *Military Medicine*, 156, 193-197.