

**POSTTRAUMATIC STRESS DISORDER, ITS SEQUELAE AND COGNITIVE
BEHAVIOURAL THERAPY AS AN APPROPRIATE THERAPY**

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STATEMENT

I, the undersigned, hereby declare that the work contained in this assignment is my own original work, and that I have not previously in its entirety or in part submitted it at any university for a degree.

ABSTRACT

Posttraumatic stress Disorder (PTSD) has been researched and written about since its recognition as a disorder in the DSM-III in 1980. This study reviews the literature on PTSD and cognitive behavioural therapies, designed for its treatment. It covers the historical aspects of how the syndrome came to be identified, defined and recognized as a discrete disorder. The neurobiological features of the disorder, its cognitive processes, particularly those of dissociation and traumatic memory are dealt with. Cognitive behavioural therapies are reviewed, these include: relaxation; hypnosis; exposure; eye movement desensitization reprocessing; anxiety management training; cognitive therapy and combination therapies. Further areas for research are suggested based on the lacunae that have yet to be explored with regard to cognitive behavioral therapy treatments.

OPSOMMING

Posttraumatiesestressversteuring is nagevors en bespreek vandat dit in 1980 in die DSM-III as 'n versteuring herken is. Hierdie studie gee 'n oorsig van die literatuur oor PTSV en kognitiewe gedragsterapeutiese, ontwerp om dit te behandel. Dit gee 'n historiese oorsig van hoe dit geïdentifiseer, beskryf en gedefinieër is as 'n spesifieke gedragsversteuring. Die neurobiologiese implikasies van die versteuring, die kognitiewe kenmerke, veral die van dissosiasie en traumatiese geheue word ondersoek. Kognitiewe gedragsterapie word beskryf en sluit die volgende in: ontspanning; blootstelling, oogbewegingdesensiteringherprosering; angsbeheeropleiding; kognitiewe terapie en kombinasie terapie. Verdere areas vir navorsing word voorgestel en is gebaseer op die gebreke wat bestaan in kognitiewe gedragsterapie.

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POSTTRAUMATIC STRESS DISORDER, ITS SEQUELAE AND COGNITIVE BEHAVIOURAL THERAPY AS AN APPROPRIATE THERAPY

Introduction

This study focuses on Posttraumatic Stress Disorder (PTSD), its sequelae and on Cognitive Behavioural Therapy (CBT) as an appropriate therapy for its treatment. Treating PTSD with CBT is the most well-researched form of dealing with the syndrome. Since it has been the subject of the greatest number of empirical studies of PTSD treatments, it seems fitting that a literature study should cover how the disorder has been dealt with using this approach.

How PTSD came to be recognized as a discrete disorder, its official recognition by the American Psychiatric Association and its definition are discussed. The neurobiology of PTSD, its cognitive processes and traumatic memories are dealt with. The various Cognitive Behavioural treatments: relaxation, hypnosis, exposure, eye movement desensitization reprocessing, anxiety management training, cognitive therapy and combination therapies are explored.

Etiology of nosology of PTSD

The conceptualization of Posttraumatic Stress Disorder has been problematic since its inception. The historical origins of how the syndrome came to be recognized as a discrete disorder illustrate the difficulty of definition. Instances of trauma in literature date back to Shakespeare's time. One only needs to think of King Lear's loss at the death of Cordelia to appreciate this. According to Joseph, Williams & Yule (1997), trauma has featured in literature dating back to the 1600s. As reported by Daly, cited in Joseph et al. (1997), Samuel Pepys recorded his own reactions to the great fire of London in 1666. In Victorian times clinical explanations for psychological reactions that seemed out of scale with actual accidents arose from mass transport disasters. The physical

consequences of trauma were the focus of interest at that time. Damage to the spine and central nervous system, for example, were considered the cause of “railway spine” (Erichsen, 1866; Trimble, 1981 cited in Joseph et al., 1997).

Shell shock observed in soldiers from World War I was the first real explanation given for reactions to stress on the battlefield. Shell shock was initially conceived solely in physical terms, namely, as that which caused damage to the physical system. Gradually it came to be realized that some people developed similar symptoms without having experienced a shell explode near to them. By World War II concepts such as post-trauma syndrome were named by Kardiner (1941) and war neurosis was a term coined by Grinkel & Spiegel (1943). Kardiner realized that the syndrome included “feelings of irritability”, “outbursts of aggression, exaggerated startle response, and fixations on the traumatic event”, (Yule, Williams & Joseph, quoted in Yule, 1999, p.2).

Mental health professionals began to recognise similar clusters of symptoms among civilians exposed to acute stressors. It was the advent of the Vietnam War that saw the recognition of the syndrome of Post-Traumatic Stress Disorder (PTSD) according to Figley, (1978) cited in Yule et al. (1997). Long term effects on personality and adjustment were considered as a result of exposure to the horrors of war. Horowitz’s work (1976-1979) on trauma-related reactions saw the traumatized individual as suffering from intrusive and emotionally disturbing memories. He noted that avoidant strategies were used to ward off distressing thoughts, images and feelings. Avoidance or denial was seen as a normal defence against overwhelming emotions. In the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 1980) a syndrome of PTSD was recognized that postulated that, following particularly traumatic events, intrusive re-experiencing of the trauma would occur, along with avoidant behaviours, and a group of symptoms of increased physiological arousal.

The triad of symptoms recognized as PTSD - re-experiencing, numbing and avoidance, and hyperarousal, have come to be recognized as part of the syndrome as defined in the DSM-IV-TR, the latest diagnostic manual of the American Psychiatric Association, (American Psychiatric Association, 2000).

The official recognition by the APA of the syndrome of PTSD occurred in 1980. The definition of the disorder was revised in the DSM-III-R in 1987 and in the DSM-IV in 1994. The tenth revision of the International Classification of Diseases (ICD 10) defined PTSD along similar lines to the DSM. By 1990, PTSD "had been defined and recognized internationally" (Yule, 1999, p.5).

DSM-IV-TR definition

According to the DSM-IV-TR the diagnostic criteria for Posttraumatic Stress Disorder are as follows:

A. The person has been exposed to a traumatic event in which both of the following were present:

- (1) The person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others
- (2) The person's response involved intense fear, helplessness, or horror. **Note:** In children, this may be expressed instead by disorganized or agitated behavior

B. The traumatic event is persistently experienced in one (or more) of the following ways:

- (1) Recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions. **Note:** In young

children, repetitive play may occur in which themes or aspects of the trauma are expressed.

- (2) Recurrent distressing dreams of the event. **Note:** In children, there may be frightening dreams without recognizable content.
 - (3) Acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated). **Note:** in young children, trauma-specific reenactment may occur.
 - (4) Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event
 - (5) Physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event
- C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:
- (1) Efforts to avoid thoughts, feelings, or conversations associated with the trauma
 - (2) Efforts to avoid activities, places, or people that arouse recollections of the trauma
 - (3) Inability to recall an important aspect of the trauma
 - (4) Markedly diminished interest or participation in significant activities
 - (5) Feeling of detachment or estrangement from others
 - (6) Restricted range of affect (e.g., unable to have loving feelings)
 - (7) Sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span)
- D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:

- (1) Difficulty falling or staying asleep
- (2) Irritability or outbursts of anger
- (3) difficulty concentrating
- (4) hypervigilance
- (5) exaggerated startle reponse

E. Duration of the disturbance (symptoms in Criteria B, C, and D) is more than 1 month.

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

Specify if:

Acute: if duration of symptoms is less than 3 months

Chronic: if duration of symptoms is 3 months or more

Specify if:

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

Issues in defining PTSD :

(i) Normal versus abnormal reaction

In the literature pertaining to PTSD, there has been a conflict between seeing victims as suffering from a normal reaction to an abnormal event and those who characterise PTSD as a psychiatric illness. Historically, according to Yehuda & McFarlane (1995), Freud's initial theory of neurosis was that hysteria had traumatic origins, but he differentiated this from neuroses that were the result of developmental fixation. Similarly, Janet [van der Kolk & van der Hart (1989), cited in Yehuda & McFarlane (1995)] observed dissociation following exposure to

trauma and reinforced the idea that exposure to trauma might have consequences for mental health. The existence of PTSD by the time that DSM III definition occurred, raised the issue of how to conceptualise the decompensation that is manifested in individuals following exposure to trauma. The question revolved around whether trauma survivors were 'psychologically damaged' by their traumatic experiences, or whether their responses were an adaptation to frightening environmental events (Yehuda & McFarlane, 1995).

Scientific evidence about the effects of trauma was important in the rationale for PTSD. Early biological studies of stress contributed to early conceptualisations of PTSD. According to Yehuda and McFarlane (1995), many recent findings disagree with the notion that traumatic events are the primary cause of symptoms and challenge the idea of PTSD as a typical stress response. Emerging data support the validity of this view. Among those who are exposed to severe and prolonged trauma, there are a substantial number of individuals who do not develop PTSD or other psychiatric illnesses.

The theory of diathesis stress suggests that some individuals are more likely, because of a general predisposition to mental illness, to develop PTSD when faced with a traumatic event. Horowitz's model has postulated that the symptoms of PTSD are "a continuation of the normal acute traumatic phenomena" and of "the failure of restitution of this process" (Yehuda & McFarlane, 1995, p1708). The severity of symptoms is dependant on the magnitude of the trauma. Other studies have questioned Horowitz's theory, as shown below.

(ii) Previous history of traumatization

A range of factors other than the severity of trauma may account for the nature of acute traumatic reactions. These may include history of prior trauma. In a study undertaken by Resnick and colleagues [Resnick, Kilpatrick, Best & Kramer (1992) and Resnick, Yeshiva, Pitman & Foy (1995), cited in Yehuda &

McFarlane (1995)], women with a prior history of rape were found to be three times more likely to develop PTSD than those raped for the first time. Lower cortisol levels were recorded in those with a previous rape history. Since cortisol levels are affected by trauma, lowered levels represent a risk for the development of PTSD. These studies question the idea that PTSD is a continuation of the normal stress response. The data suggest that the emergence of chronic symptoms may be predicted by particular biological and psychological features of acute response to trauma (Yehuda & McFarlane, 1995).

(iii) Defining traumatic events

Not only has the conceptualization of PTSD as a discrete disorder been a point of contention, but what constitutes a traumatic event has been difficult to define. Traumatic events range from earthquakes, floods, fires, to war and individual trauma such as rape and hijacking. There has been a lack of agreement about what constitutes a disaster. A number of typologies and classification systems have been suggested, but further conceptualizing and classification is needed, according to Baum et al. (1993). Part of the problem in identifying PTSD following disasters is that various studies have focused on disasters which are quite different in nature and severity. Difference exists between a repeated traumatisation and an acute, discrete one. Many natural disasters such as earthquakes or gas leakages may occur over a few days or weeks and, therefore, become lasting processes. The precise point at which the posttrauma process begins, is not always obvious. This represents a methodological issue which relates to posttrauma adaptation. Similarly there are various war experiences, ranging from combat to loss of a buddy, to medical personnel stress, to witnessing and participating in atrocities which will affect postwar outcomes differentially.

(iv) Severity of trauma

The severity of a trauma may also influence the development of PTSD, according to Foa and Meadows (1998). Studies of Vietnam veterans found that the “severity of posttrauma pathology was predicted by the degree of exposure to combat”, (Foy et al. 1984; Pitman et al. 1989 quoted in Foa & Meadows, 1998). Perception of threat to an individual’s life during rape predicted severity of resulting post-trauma pathology, (Girelli et al. 1986; Kilpatrick et al. 1989, cited in Foa & Meadows, 1998). Therefore, it is clear that it is also the nature of the traumatic event that has an effect on the risk of developing PTSD.

(v) Other experiences

Individual experiences during and after a trauma may influence a person’s reactions to trauma. The extent of dissociation, during and after a trauma, has been associated with posttrauma distress and PTSD, according to Foa and Meadows (1998). Trauma victims with early peaking PTSD symptoms (that is, within 2 weeks after the trauma) show better recovery 3 months later, than those whose critical PTSD symptoms occur 2 to 6 weeks after the trauma (Foa and Meadows, 1998).

In summary, experiencing a traumatic event is a necessary but not essential condition for developing PTSD. Other factors, such as trauma history, pretrauma functioning, the responses of the traumatized individual and of significant others after the traumatic event, also play an important role. How the individual responds cognitively to the trauma, including the meaning that is ascribed to it and the consequent changes in beliefs about the self and world, are involved in the development of PTSD.

Neurobiology of PTSD

Establishing PTSD as a distinct biological entity, was controversial in the 1980s because it was unclear that what was being described by this diagnosis differed from what was being captured by other mood and anxiety disorders. Biological studies have helped to clarify that PTSD is a particular type of stress response characterized by “a progressive sensitization of biological systems” (Yehuda & McFarlane, 1995, p.1710).

As referred to by Young (1995), the biological effects of ordinary stress responses are fleeting, unlike those of PTSD which are characterized by enduring neurophysiological changes. When a traumatic event occurs, large amounts of noradrenalin (NOR) are released into the neuronal synapses and this leads to “long-term augmentation’ or “potentiation” of the locus coeruleus pathways leading to the limbic system and to changes in the “synaptic structures.....in the temporal-amygdaloid complex concerned with agonistic behaviour”, according to van der Kolk and his associates (1985) , cited in Young (1995). According to Mason et al. 1990 and Watson 1988, cited in Young (1995), it is also responsible for neuronal changes in the sympathetic system. In effect, van der Kolk is saying that the body remembers the trauma somatically.

According to DeBellis et al., (1999) cited in Cohen, Deblinger, Mannarino & Steer’s study (2004), abused children who suffer from PTSD symptoms have smaller total brain and corpus callosum volumes as well as lower IQs than matched controls. Those suffering from these differences were also younger in age at the onset of abuse and had a longer duration of PTSD symptoms.

Experiencing a trauma is like exposing an animal to an inescapable shock. When an individual is exposed to overwhelming stressors, they are unable to predict the onset or termination of the stressors and are unable to fight against them or

to flee from them. Inescapable shock stimulates the release of large amounts of NOR in the central nervous system. This NOR eventually becomes depleted because the demand for it exceeds the supply and this results in feelings of “chronic helplessness”, “diminished motivation, decline in occupational functioning and global constriction” in the form of numbing and anhedonia, as well as extreme sensitivity to transient stimulation (Young, 1995, pp. 277-278).

Behaviourally, when the autonomic system becomes hypersensitive, the person becomes hyper-reactive. This is evidenced in startle responses, intrusive images and memories, nightmares and an overreaction to conditioned stimuli as well as outbursts of anger, (Young, 1995).

The release of large amounts of NOR results in a form of hypermnesia in which the details of the event “become locked into the person’s memory” (Pitman et al., 1987, cited in Young, 1995). A single neuron in the locus coeruleus can branch both into the limbic system and into the neocortex where long term memories are stored. This combination of hypermnesia and hyperreactivity explains the flashbacks and the vivid nightmares that occur after the aversive event.

One of the most characteristic features of PTSD is that memories of traumatic experiences are stored indelibly for decades and are easily reawakened by all kinds of stimuli and stressors. Some of the acute neurobiologic responses to trauma may facilitate the encoding of traumatic memories. Simple sensory phenomena, in those suffering from PTSD, such as specific smells, sounds and visions, related to the traumatic event, persistently produce a resurfacing of traumatic memories and flashbacks. The regions of the brain that mediate this process include the amygdala, locus coeruleus, hippocampus and sensory cortex. The amygdala is the most important in the conditioning and extinction of sensory and cognitive associations to the original trauma and to the subsequent activation of traumatic memories. The amygdala has extensive connections to sensory systems in the cortex. Many memories associated with traumatic events

are most likely stored in the cortical region (Charney, Deutch, Krystal, Southwick & Davis, 1993, cited in Young, 1995).

Changes occur in the synaptic pathways in the locus coeruleus and hypothalamus which adversely affect the cerebral cortex's inhibitory control over the parts of the brain that are concerned with the expression of aggression and sleep cycles. These changes, together with autonomic hypersensitivity, account for the explosive anger and difficulties falling asleep and staying asleep in PTSD. When PTSD victims are exposed to stimuli that resemble their traumatic experiences endorphins are released. At the same time that arousal occurs and endorphins are released, temporary relief may be provided, as a protective measure.

What is notable in traumatic stress responses is that psychological and physiological aspects cannot be separated. According to Shalev (2003), perceived threat triggers intense bodily reactions that form the individual's mental traces of the aversive event. The subjective quality of these traces are then used to interpret subsequent adversity. This cycle of bio-psychosocial events can reduce with time. Intervention in the form of shelter, information, orientation, warmth and hope can improve the bio-psychological consequences.

Animal and human studies have isolated the specific brain areas and circuits involved in anxiety and fear which characterize PTSD. Fear, an emotion that has evolved to deal with danger, causes an automatic, protective response in many parts of the body. The fear response is the result of abnormal activation of the amygdala, deep inside the brain. Specific hormones involved in response to stress become raised. Higher levels of natural opiates are produced and these can temporarily mask pain. Even after the danger has passed, people with PTSD continue to produce those higher levels and this may lead to the blunting of emotions associated with the syndrome.

Neurophysiologically, adrenaline and noradrenaline levels increase to higher than normal, while cortisol levels lower, (National Institutes of Mental Health (NIMH), 2001). One of the functions of the neurotransmitter, noradrenaline, is to activate the hippocampus, the part of the brain that deals with organizing and storing long-term memory. The high levels of noradrenaline explains why emotionally arousing events are better remembered than other situations. Under the extreme stress of trauma, noradrenaline may act for longer and more intensely on the hippocampus and would explain the formation of abnormally strong memories in the form of flashbacks or intrusions. Cortisol limits noradrenaline activation and, since cortisol levels are lowered during trauma, these lowered levels may represent a risk factor for developing PTSD (Yehuda & McFarlane, 1995). The distinctiveness of biological changes following exposure to trauma support the notion of PTSD as a discrete disorder.

Cognitive processes of PTSD

In looking at the cognitive processes in PTSD, the phenomenon of dissociation needs to be considered. Acute dissociation is “a critical component of long-term post-traumatic reactions”, according to McFarlane (2003, p 50).

Dissociation consists of many components including depersonalization, derealization, amnesia and time-distortion. Reported dissociative symptoms range from disorganization and slowness in thinking and decision-making, as seen in dam disaster victims, (Titchener and Kapp, 1976, cited in McFarlane, 2003), as well as to constricted affect in combat soldiers, (Feinstein, 1989, cited in McFarlane, 2003). According to Berah et al., 1984, cited in McFarlane (2003), shock and bewilderment were reported after a bush fire disaster.

Derealization occurs when the individual perceives the external environment as unreal, while depersonalization is a state in which “individuals have a sense of observing themselves from the outside”, as detached from their own bodies (McFarlane, 2003, p.52). Earthquake and tornado survivors tend to experience

depersonalization, (Cardena and Spiegel, 1993, cited in McFarlane, 2003). Other dissociative symptoms include numbing and a narrowing of awareness of the environment. Theoretically, Janet (1911) and van der Kolk et al. (1996) according to McFarlane (2003) consider dissociation as a primary coping mechanism “that minimizes the disruptive effects of traumatic events by decreasing the individual’s awareness” of what is happening and what has happened. This change in awareness is associated with “memory impairment, emotional detachment, derealization and depersonalization” (McFarlane, 2003, p.52). Dissociative reactions may hinder the linking of cognitive reappraisals that occur after trauma with fear structures that are lodged “within memory representations of trauma” (McFarlane, 2003, p. 52). As a result, memory becomes fragmented.

Trauma can lead to extremes of retention and forgetting, frightening experiences can be remembered vividly or they may resist integration completely. Often individuals report a combination of both modes of processing memory. Some aspects of a traumatic event or events seem to become fixed in the mind in the form of post-traumatic nightmares in which subjects complain that they see the same traumatic scenes over and over again, without changes, up to over a fifteen year period, (van der Kolk, Blitz, Burr & Hartmann, 1984, cited in van der Kolk & Fisler, 1995).

Emotional and perceptual elements tend to be more prominent in traumatic memories, giving rise to “the notion that traumatic memories may be encoded differently to memory for ordinary events”, perhaps because of alterations in attentional focusing and perhaps because extreme emotional arousal interferes with memory functions in the hippocampal area of the brain (Christianson et al, 1992, cited and quoted in van der Kolk & Fisler, 1995, p. 4).

Traumatic memories

A wide variety of memory functions can be affected by trauma. Difficulties include (i) traumatic amnesia, (ii) global memory impairment, (iii) dissociative processes and the (iv) sensorimotor organization of traumatic memories.

Elliot and Briere (unpublished at the time), cited in van der Kolk & Fisler (1995), undertook a study of 485 subjects in which they reported significant degrees of trauma-related amnesia after almost every form of traumatic experience. The highest rates were reflected in “childhood sexual abuse, witnessing domestic violence as a child, and combat exposure” (Elliot & Briere, unpublished, cited in van der Kolk & Fisler, 1995, p. 5). The “younger the age at the time of the trauma and the more prolonged the traumatic event, the greater the likelihood of significant amnesia”, (Briere & Conte, 1993; Herman & Shatzow, 1987; van der Kolk, Roth, Pelcovitz & Mandel, 1993, cited in van der Kolk & Fisler, 1995 p. 5).

Traumatic amnesia

Amnesia can last for hours, weeks, or years. Recall is set in action by exposure to sensory or affective stimuli that are similar to sensory or affective elements associated with the trauma. Activation of one aspect of the memory system facilitates the recall of associated memories, according to Collins & Loftus, 1975; Leichtman, Ceci & Ornstein, 1992, cited in van der Kolk and Fisler (1995). Affect seems crucial for the retrieval of information along the associative pathways. The affective power of any particular experience is important in determining what cognitive schemes will be activated. Many people with traumatic histories can function quite well until feelings related to their traumatic memories are stirred up.

Global memory impairment

The mechanism by which memory becomes impaired in trauma is not properly understood. More research is needed to explore the observation that adults who are severely traumatized as children suffer from “generalized impairment of memories for both cultural and autobiographical events” (Cole & Putnam, 1992, cited in van der Kolk & Fisler, 1995). Lack of autobiographical memory, dissociation and a lack of meaning schemes of victimization, helplessness and betrayal, is likely to make these individuals open to suggestion and to explanations for their traumatic affects that may not bear relationship to the realities of their lives, (van der Kolk & Fisler, 1995).

Trauma and dissociation

Experiencing dissociation at the moment of trauma is the most important long term predictor of PTSD (Holen, 1993; Marmar et al., 1994; Spiegel, 1991, cited in van der Kolk & Fisler, 1995).

In dissociation, “elements of the traumatic experience are not integrated into a whole, but are stored in memory as isolated fragments” and “as sensory perceptions, affective states or as behavioral enactments”, (Nemiah, 1998; van der Kolk & van der Hart, 1989, 1991, quoted in van der Kolk & Fisler, 1995, p.6). Lack of integrating traumatic memories leads to the development of PTSD. Information processing and information storage into explicit memory seems to be interfered with by intense arousal. The person is often left in a state of “speechless terror” when they cannot describe what has happened (van der Kolk, 1987, cited in van der Kolk & Fisler, 1995). Although an individual may not be able to give a narrative account of events from explicit memory, there may be no interference with implicit memory; they may know the feeling of a stimulus and be aware of associated perceptions, but they may not be able to give reasons for why they feel or behave in a particular way.

Sensory-motor organization of traumatic experience

According to van der Kolk & Fisler (1995, p.8), numerous authors, such as Janet (1889), van der Kolk & van der Hart (1991), Kardiner (1941) and Terr (1993), “have observed that trauma is organized in memory on sensori-motor and affective levels”. Memories of trauma seem to be predominantly experienced as fragments of the sensory aspects of the event, that is, as “visual images, olfactory, auditory, or kinesthetic sensations”, or as “intense waves of feeling” of elements of the original traumatic event (quoted in van der Kolk & Fisler, 1995, p.8). Patients claim that their perceptions are precise reenactments of sensations at the time of the trauma.

Van der Kolk and Fisler's 1995 study for examining traumatic and non-traumatic memories in individuals with PTSD to record “whether, and how memories of traumatic experiences are retrieved differently from memories of personally significant, non-traumatic events”, established that all subjects, regardless of the age at which the trauma first occurred, reported that they “initially ‘remembered’ the trauma in the form of somatosensory or emotional flashback experiences”. Of the subjects, 89% were able to narrate a story about what happened to them, while 11% were unable to recount a coherent narrative, with a beginning, middle and end, about the trauma, even though they they had outside confirmation of what they had experienced.

It was established in the study undertaken by van der Kolk & Fisler (1995) that there are critical differences between the ways in which people experience traumatic memories compared with other significant events. The traumatic memory is dissociated and is initially stored as sensory fragments “without a coherent semantic component” (van der Kolk & Fisler, 1995, p. 12). It is only with time, according to this study, that individuals come to develop a narrative of their trauma.

In participants in van der Kolk & Fisler's study, who had been abused as children, the trauma was initially "remembered" in the form of somatosensory flashback experiences - as "visual, olfactory, affective, auditory and kinesthetic... modalities". Initially these sensory modalities do not occur together. As the trauma comes into consciousness, more sensory modalities come into awareness. As the participants became aware of more of the aspects making up their traumatic experience, they were able to construct a narrative explaining what had happened to them. By contrast, when people have had non-traumatic experiences, the sensory elements of the experience are not registered separately in consciousness, but are incorporated automatically in the personal narrative. This sensory aspect of trauma means that traumatic experiences continue to return as "sensory perceptions and affective states" (van der Kolk & Fisler, 1995). Although a narrative may be constructed subsequently, the intrusions related to the trauma continue as sensations. Learning to put the traumatic experience into words, therefore, does not abolish the occurrence of flashbacks. A more sensory kind of therapeutic processing, therefore, may be more appropriate in the treatment of trauma. The failure of information processing "on a symbolic level", by which it can be "categorized and integrated with other experiences", form the "core of the pathology of PTSD", according to van der Kolk & Ducey, cited in van der Kolk & Fisler (1995).

Traumatic memories, therefore, constitute emotional and sensory states, with little verbal representation. It was hypothesized that, under extreme stress, "the hippocampal memory categorization system fails" and leaves memories to be stored as "affective and perceptual states" (van der Kolk & Fisler, 1995). In addition, extreme arousal at the time of trauma interferes with the effective memory processing of the experience. The memory traces that are laid down may be unmodified over time or by further experience resulting in the "speechless terror" referred to previously, (van der Kolk & Fisler, 1995).

Thus far this paper has considered the origins of how PTSD came to be recognized as a discrete syndrome; its neurobiology; what happens cognitively in PTSD, namely, how traumatic memories are processed and how the sensory-motor aspects of PTSD manifest. In the light of the importance of cognitive factors in PTSD, cognitive-behavioral treatments will now be considered.

Cognitive behavioural treatment for PTSD

According to Foa & Cahill (2002), there is accumulating evidence for the effectiveness of a number of cognitive behavioural treatments for chronic PTSD - in particular, prolonged exposure and stress inoculation training. Although not as well researched, cognitive restructuring and cognitive processing therapy (CPT), as well as variations of eye movement desensitization and reprocessing (EMDR) promise well.

Foa and Meadows (1998) outlined seven “gold standards” for treatment outcome studies. These standards establish cognitive behavioural therapy as empirical in nature. The standards are as follows:

1. Clearly defined target symptoms (i.e. PTSD or major depression rather than “post-rape distress”)
2. Reliable and valid measures (i.e. standardized rather than idiosyncratic questionnaires)
3. Use of blind evaluators (to avoid expectancy and demand biases)
4. Assessor training (including both initial training and ongoing calibration)
5. Manualised specific treatment programs (to ensure consistency of treatment delivery and to allow replication)
6. Unbiased assignment to treatment (i.e., random assignment or stratified sampling)

7. Treatment adherence (to ensure treatment manuals were followed correctly).

Controlled studies have examined the efficacy of cognitive-behavioural treatments for PTSD. Cognitive-behavioural therapies that appear throughout the literature and which seem most efficacious are: relaxation training, hypnosis, exposure therapy, cognitive restructuring, training in relaxation, hypnosis, Eye Movement Desensitisation Reprocessing, Anxiety Management Training, Cognitive Therapy and combined treatment programs (Yehuda, 1998; Zoellner, Fitzgibbons & Foa, cited in Wilson et al, 2001). Each will be considered in turn.

Relaxation

Relaxation is particularly helpful for the symptoms of hyperarousal and motor tension in PTSD. Progressive Muscle Relaxation (PMR) also assists in dealing with nervousness, hypervigilance, exaggerated startle response, irritability and with symptoms of hyperactivity, such as nausea, muscle tension, tachycardia, and excessive sweating. Insomnia is also alleviated by PMR (Scrignar, 1996).

According to Scrignar (1996), Jacobson's technique of PMR is the "most widely used method of muscle relaxation". An explanation is given to the patient about the relationship between muscle relaxation and stress. The patient is told to take a few deep breaths and then told to breathe normally, then, in sequence to contract and relax the various muscles of the body. After about 30 minutes the patient begins to feel tranquil and relaxed. PMR is required to be practised for several weeks. When they are able to do this, patients can apply the technique anywhere and at any time, with their eyes open, by first noting the presence of any tension in the muscles, then by taking several slow, deep breaths and systematically reducing tension in selected muscle groups during exhalation. Relaxation can, therefore, be done while the patient is "walking, driving, standing

or sitting” as well as during “fearful situations during *in vivo* desensitization” (Scrignar, 1996, p. 126).

Hypnosis

Dissociation occurs during and after a traumatic event. Hypnosis assists in dealing with dissociative symptoms because it facilitates the working through of traumatic memories, allows for painful emotions, increases control of intrusive memories and also helps to strengthen the therapeutic alliance, (Brende & Benedict 1980; Putnam 1992; Foa et al., 1995, cited in Scrignar, 1996).

During hypnosis the patient relaxes, concentration increases and receptivity to suggestion becomes enhanced. According to Schiraldi (2000), peaceful imagery is used for induction into the hypnotic state. The therapist might say, “Imagine walking slowly downstairs”; “Imagine that your limbs are warm and heavy” in order to induce a relaxed state (Schiraldi, 2000, p.252). Once the patient has been inducted, they are open to suggestion. Their traumatic experience can be rewritten, as it were, so that their visualization contradicts the painful state that was part of the trauma and the trauma becomes manageable. At the end of a therapy session, the ‘jettison technique’ can be used whereby the patient is asked to make a fist in which all their fears and problems are clasped and on the count of three they are asked to open their fist and to release their anxieties and are told that they will feel happy, confident and calm at the same time. Hypnosis is useful in dealing with PTSD because it enables a person to process their fears and to rewrite their script in order to assist with healing.

Exposure

Exposure therapy usually involves the confrontation of feared stimuli either through imagination or in person. It usually involves repeated confrontation both with the memories of trauma (imaginal exposure) and with the trauma-related

situations that give rise to unrealistic fears (in vivo exposure). The goal of this intervention is to help the patient “emotionally process the trauma by vividly imagining the event and by describing it aloud, including thoughts and feelings” that occurred during the trauma. In vivo exposure “helps the processing of the trauma by instructing the patient to confront situations, places, or activities that trigger trauma-related fear and anxiety” (Foa & Cahill, 2002, p.50). The patient is asked to remain in the anxiety provoking situation until he or she becomes habituated to their fear. The reason for exposure therapy for PTSD is that emotional processing of the traumatic event and, therefore with recovery, is interfered with by the avoidance of trauma-related memories and reminders.

In order to help patients confront their fears in a systematic manner, learning models are used as a basis. The most influential of these, according to Foa & Meadows (1998) is Mowrer’s two-factor theory (1960) in which it is postulated that “fear is acquired via classical conditioning and is then maintained via operant conditioning”. That is, previously neutral stimuli such as the place of the trauma, become triggers for fear because of their association with the traumatic event. These stimuli are then avoided or escaped to reduce anxiety. Exposure, within this theoretical framework, “is used to break the conditioned associations and to interrupt the pattern of avoidance that maintains the fear” (Foa & Meadows, 1998).

According to Foa and Kozak (1986, cited in Foa & Meadows, 1998), another conceptualization based on information processing, postulates that exposure therapy promotes emotional processing because it modifies pathological fear structures. The pathological fear structure is activated, but then is paired with incompatible, corrective information. In this way PTSD is ameliorated via exposure therapy. The corrective information entails providing the patient with the information that “trauma –related stimuli are not dangerous”, that memories of the trauma are different to the trauma itself, that anxiety reduces without

avoidance/escape, and “that PTSD symptoms are not signs of a loss of control” (Foa & Meadows, 1998).

Systematic desensitization (SD) was one of the earliest exposure techniques used by Wolpe in 1958 to treat posttrauma reactions (Wolpe, 1958 cited in Foa & Meadows (1998). Using this technique initially involves inducing muscle relaxation, which inhibits anxiety, during which the patient is asked to imagine a weak, anxiety-arousing stimulus for a few seconds. As the exposure is repeated, the stimulus progressively loses its ability to evoke anxiety. Successively stronger stimuli are then treated. Uncontrolled studies and case reports have indicated that SD was partially effective in reducing trauma-related symptoms. Subsequently, prolonged exposure with or without relaxation gradually replaced SD. This shift in focus came about because the findings established that “long exposures were better than short ones” (Stern & Marks, 1973, cited in Foa & Meadows, 1998) and “that exposure to real stimuli (was) more effective than exposure to imagined stimuli” (Barlow et al., 1969, cited in Foa & Meadows, 1998). However, prolonged exposure therapies for PTSD usually include both imaginal exposure, when the traumatic memories are relived and in vivo exposure, during which trauma-related situations are confronted.

According to Foa & Meadows (1998), Marks et al. (in press at the time), compared how effective exposure, both imaginal and in vivo, cognitive restructuring, their combination, and a relaxation control treatment were. The three former treatments were found to be “more effective than relaxation in reducing PTSD symptoms” (p.189).

Imaginal exposure has been examined with regard to the question of whether it causes more harm than therapeutic benefit to the patient. A study was conducted by Foa, Zoellner, Feeny, Hembree and Alvarez-Conrad (2002) in which the use of imaginal exposure was examined for the possible exacerbation of symptoms. According to Foa et al., (2002), trauma experts have been unwilling to use

imaginal exposure with victims of traumatic events, for fear of “retraumatising” them and increasing their suffering. Kilpatrick and Best (1984), cited in Foa et al. (2002), asserted that after treatment, sexual assault victims may show higher levels of distress than before treatment.

The findings of Foa et al. (2002) established that a minority of participants receiving treatment for chronic PTSD showed reliable, although small increases in symptoms after introducing imaginal exposure: 10.5% reported an increase in PTSD symptoms, 21.1% increases in anxiety and 9.2%, an increase in depression. One week after the first imaginal exposure in the prolonged exposure (PE) condition, more individuals in this condition reported worsening of their anxiety symptoms, suggesting that introducing imaginal exposure “may be associated with a temporary increase in general anxiety symptoms”. That symptom exacerbation may hinder response to treatment, however, did not receive support, according to Kilpatrick & Best, cited in Foa et al., (2002). Individuals who reported initial symptom exacerbation of PTSD, anxiety or depression, “benefited from treatment as much as those who did not report such an exacerbation”. Therefore, the overall concern expressed by Kilpatrick & Best that prolonged exposure leads to general symptom exacerbation, was not supported in the study undertaken by Foa et al. (2002).

Prolonged exposure, according to Foa et al. (2002, p.1026), “has gained more empirical support for its efficacy than any other treatment for PTSD”.

In a study undertaken by Zoellner, Feeny, Cochran & Pruitt (2003), factors influencing the incentive for seeking help for assault were investigated. Data was collected from 273 women with a trauma history and subsequent PTSD symptoms. The subjects were given the same trauma scenario of sexual assault and three treatment options to choose from, namely setraline (SER), prolonged exposure (PE), or no treatment. It was found that women were more likely to choose PE than SER to treat chronic PTSD. Two hundred and seventy three

women were recruited from the University of Washington (Seattle, WA) and Case Western Reserve University (Cleveland, OH). When they were asked to make a forced choice for treatment from the three treatments offered, 87.4% chose PE, 6.9% chose sertraline, and 5.7% chose no treatment. Therefore, women in the study did not consider medication a viable treatment option for chronic PTSD; the majority of the subjects chose PE, a cognitive behavioural therapy. Sertraline is an effective treatment (as discussed below), although it was not chosen.

Prolonged exposure consists of 9-12 individual therapy sessions, during which a patient meets with their therapist for 60-90 minutes. Procedures in this treatment include: education about reactions to trauma, relaxation, repeated prolonged exposure to traumatic memories, repeated in vivo exposure to situations that the patient may be avoiding because of trauma-related fear. Homework is also given to encourage practicing what has been learnt in therapy.

Medication given takes the form of the sertraline, Zoloft, which is a selective serotonin reuptake inhibitor (SSRI) antidepressant, shown to be effective in the treatment of PTSD, according to Zoellner, Feeny, Cochran & Pruitt (2003). The amount prescribed is 200mg of Zoloft daily for 10 weeks.

The authors, Zoellner, Feeny, Cochran & Pruitt, conclude that what may be important in offering treatment options is how these treatment options are talked about in order to understand the factors that influence treatment choice.

Eye Movement Desensitisation Reprocessing

A form of exposure therapy, eye movement desensitization and reprocessing (EMDR) was developed (Shapiro, 1989, 1995, cited in Foa & Cahill, 2002) to deal with PTSD. EMDR is a therapeutic approach that has provoked a great deal of interest among trauma therapists and researchers. In EMDR, "the therapist asks the patient to generate images, thoughts, and feelings about the trauma, to

evaluate their aversive qualities, and to make alternative cognitive appraisals of the trauma or their behaviour during it" (Foa & Cahill, 2002, p.52). As the patient focuses on the disturbing images and thoughts and on alternative cognitions, the therapist elicits rapid lateral eye movements by instructing the patient to track the therapist's finger visually as it is moved back and forth rapidly in front of the patient's face. This technique, therefore, pairs imaginal exposure with the induction of saccadic eye movements. The early findings of the efficacy of this form of therapy were inconclusive since they suffer from serious methodological flaws, rendering their findings uninterpretable (Foa & Meadows, 1998). The saccadic eye movements are considered to be an important element of treatment.

Originally, Shapiro (1991) considered the lateral eye movements as essential to the processing of the traumatic memory, saying that they reverse the neural blockage caused by the traumatic event. The assertion of the pivotal role of the rapid eye movements has not been supported by studies undertaken by Boudewyn and Hyer, (1996); Pitman et al., (1996); or Renfrey and Spates (1994), according to Foa & Cahill (2002).

In a well-controlled study by Pitman and colleagues (1996), according to Zoellner et al., male veterans with PTSD received up to six sessions of eye movement desensitization reprocessing (EMDR), "either with saccadic eye movements or with eyes in a fixed position". Neither treatment was found to produce clinically significant results. "On average EMDR produced 11% reduction and the fixed-position condition produced 16% reduction". As a result of these findings, Pitman et al. concluded that eye movement per se was not the important ingredient in EMDR. The advantage of EMDR, compared to other exposure techniques, therefore, was questionable.

More recent studies have, yet again, questioned the efficacy of EMDR and their findings differ from the earlier studies. In a study undertaken by Lytle, Hazlett-

Stevens and Borkovec (2002), their findings of a non-clinical college student sample in which eye movement desensitization (EMD) was tested, revealed that there was no support for the superiority of the EMD condition, compared to the condition without eye movements. However, the authors assert that certain limitations of their investigation should be considered because the study was “limited to a sub-clinical student population” and they propose that “had the clinical presentation of the participants been more severe, or had they been seeking treatment, differential responses to the treatment may have occurred”. They also posit that “these data may not reflect the current practice of EMDR” since newer aspects not included in their research may be included in other studies.

EMDR, according to Shapiro (2002), had been compared to treatments and controls in research by the time of publication. These included “ (a) wait list controls, (b) Veterans Administration standard care, (c) biofeedback-assisted relaxation, (d) muscle relaxation, (e) active listening, (f) individual psychotherapy, (g) exposure therapies and (h) combinations of exposure and cognitive therapies” (Shapiro, 2002, p.5). In all except one of these civilian studies EMDR was found to be superior to its control conditions.

In the most stringent EMDR civilian studies (Lee et al., 2002; Marcus et al., 1997; Rothbaum, 1997; Scheck et al 1997; Wilson et al., 1995,1997, cited in Shapiro, 2002), considerable clinical effects were reported , generally indicating that 77-99% of clients no longer suffered from PTSD after 3 to 10 hours of treatment. Large effect sizes were reported on multiple measures “with no relapse at 3-15-month follow-up” (Maxfield & Hyer, 2002; Van Etten & Taylor, 1998, cited in Shapiro, 2002). Randomised comparisons of EMDR and CBT treatments of PTSD generally reported a superiority of EMDR on a few measures and equivalent effects on other measures. Shapiro speculated that individual studies might be hampered by “non-expert fidelity checks”, or a “lack of blind independent assessors” (Chemtob et al., 2000; Maxfield & Hyer, 2002, cited in

Shapiro, 2002). In a meta-analysis of all treatments for PTSD, it was “indicated that behaviour therapy, SSRIs and EMDR” “were the most effective forms of treatment”, according to Van Etten & Taylor, cited in Shapiro (2002). More controlled comparisons were also considered as needed. EMDR appeared to be a more “efficient form” of therapy since it required only one-third of the amount of time to achieve its effects compared to results reported in behavior therapy research (Shapiro, 2002).

Many of the studies of EMDR are beset by a number of methodological problems (Chemtob et al., 2002; Feske, 1998; Shapiro, 1995, 1996, 2001; Smyth, 1999, cited in Shapiro 2002). Component treatment outcome studies that have failed to obtain differences between conditions, are hampered by the use of sub-clinical populations, multiply traumatized populations, omitted standard EMDR procedures and have been methodologically flawed in other ways (Chemo et al, Fescue, Shapiro and Smyth, cited in Shapiro 2002).

EMDR component analyses that were carried out by the time of publication of Shapiro’s article, therefore, generally provided inconclusive results. Clinically and scientifically valid research is needed before the relative importance of EMDR’s various components can be determined.

Anxiety Management Training

Anxiety Management Training (AMT) is based on the idea that pathological anxiety stems from a deficit of skills and treatment and, therefore aims at providing patients with strategies to deal with anxiety (Suinn, 1974, cited in Foa & Meadows, 1998). These strategies have included relaxation training (Jacobson 1938), self-instruction (Meichenbaum 1974), breathing retraining (Clark et al 1985), biofeedback (Blanchard and Abel 1976), social skills training (Becker et al., 1987) and distraction techniques (Wolpe 1973), all as reported by Foa & Meadows (1998).

Anxiety Management Training also deals with Stress Inoculation Training. The theoretical model posited by Veronen and Kilpatrick (1983) in Foa & Cahill (2002) in their Stress Inoculation Training (SIT) program for rape victims is based on social-learning theory. The theory holds that a traumatic event evokes emotional, cognitive, and behavioural fear responses and that cognitive appraisals and attributions mediate these responses. Neutral stimuli are associated with the traumatic event and acquire the power to provoke fear and anxiety. These neutral situations are avoided and the anxiety they provoke is also avoided. The reduction in anxiety in turn reinforces these avoidance or escape responses. Treatment aims at teaching patients skills to manage and decrease fear and anxiety associated with the event. Veronen and Kilpatrick's adaptation of SIT for rape survivors included education about trauma and PTSD, deep muscle relaxation, breathing exercises, role playing, covert modeling, thought stopping, and guided self-dialogue.

One of the most studied AMT programs for PTSD is Stress Inoculation Training (SIT). It incorporates a number of components such as education, relaxation, breathing control, cognitive restructuring, covert modeling, and thought stopping. Several uncontrolled studies have found SIT to be one of the most effective AMT therapies for PTSD (Foa & Cahill, 2002).

Cognitive therapy

Cognitive therapy refers specifically to clinical procedures that alter erroneous and maladaptive thoughts beliefs, and traumatic memories related to PTSD. It is also known as cognitive restructuring. According to Beck et al (1976), cited in Foa & Cahill (2002), cognitive theory underlying cognitive therapy posits that individuals have particular ways of thinking about the world, other people, and themselves.

The areas of PTSD with which cognitive therapy assists are with the re-experiencing aspect. It assists with desensitization of avoidance behaviour and, together with controlled breathing and other relaxation procedures, helps to reduce symptoms of increased arousal (anxiety). Cognitive therapy assists patients deal with negative thoughts associated with depression, one of the symptoms of PTSD, according to Beck et al. cited in Scrignar (1996). The goal of cognitive therapy for PTSD, according to Foa & Cahill (2002), is to teach patients to recognize their trauma-related dysfunctional cognitions that lead to negative emotions. Patients also learn to challenge these cognitions (thoughts or beliefs) in a logical manner, based on evidence. The therapist helps the patient to weigh all of the alternative interpretations of the particular events and to determine whether the belief is helpful and accurately reflects reality and if it does not, to replace it or modify it.

Individuals who have PTSD often engage in what Beck called automatic thinking which includes some of the following characteristics: selective abstraction, discounting, arbitrary inference, and catastrophising. A cue for automatic thinking usually involves some aspect of the trauma. A patient may return to the scene of the trauma and think, "I am in danger"; this stimulates anxiety and, in turn, may result in avoidance behavior. The sequence: thought (I am in danger)- feeling (anxiety)- behaviour (avoidance) can be changed by cognitive therapy.

Intrusive thoughts and ruminations, which occur in PTSD, contain cognitive errors and frequently involve personalization, polarization, and overgeneralisation [Beck 1976; Schuyler 1991, cited in Scrignar, 1996]. Trauma causes the person to become self-centred and cognitive errors of personalization inhibit them from placing posttraumatic behaviour into perspective. Their thinking becomes polarized and everything is perceived as black or white with no grey area. Statements become all or nothing in nature, for example, "I will never get well. I am permanently injured". Another cognitive error, overgeneralisation, occurs, for example the person might think , "The accident has ruined me forever. I am not

worthwhile. No one cares about me.” Cognitive therapy offers the patient the opportunity to dismiss unwanted thoughts from the mind and to substitute these with affirmative, more adaptive thoughts or visual images that are reality-based. Two techniques that can be employed to change erroneous cognitions and which affect subsequent emotions and behaviour, as already discussed, are thought stopping and thought substitution.

Thought stopping, according to Scrignar (1996), is used to control, reduce or eliminate erroneous maladaptive thoughts and to substitute instead cognitions that emphasise a positive reality. The patient is first asked to keep a diary of unwanted, erroneous, distressing thoughts that come into consciousness. They are asked to note internal and external cues resembling the trauma that evoke automatic thoughts. In the therapy session, the patient is asked to close their eyes and to visualize a prearranged scene related to their trauma which includes anxiety-evoking thoughts. After about a minute, the clinician shouts, “Stop the action” or, “Stop! Get out of there!” to momentarily shake the patient out of their thought processes. The procedure is repeated, but then the patient is instructed to silently and emphatically exclaim, “Stop! Get out of there!” each time the therapist taps them on the hand. The patient is then requested to independently use the thought stopping procedure as often as necessary to control and reduce inappropriate and anxiety-evoking thoughts. A rubber band can also be worn around the wrist and snapped simultaneously when the patient silently commands, “Stop! Get out of there” (quoted in Scrignar, 1996, pp. 130-131) . The combination of the pain of the snapping elastic band and the stopping declaration is often effective in halting unwanted mental activity.

Another cognitive restructuring technique, thought substitution, has been used in cognitive therapy. Cognitions are restructured from being negative and catastrophising, such as, “I will never work again” in the instance of a disabling accident, to “ I will work again. I have been hurt, but I am not disabled. My life will change for the better”, (quoted in Scrignar, 1996, p.131). Disaster can be

changed to opportunity as was reported when a 38 year old welder was injured in a serious accident and could not return to his occupation (Scrignar, 1996). His erroneous cognitions were changed by thought substitution.

Möller & Steel (2002) undertook a study in which they investigated clinically significant change after cognitive restructuring for adult survivors of childhood sexual abuse. The participants in this study comprised 42 adult female victims of childhood sexual abuse who did not meet PTSD or mood disorder diagnostic criteria. The measures used were the *Trauma Symptom Checklist-40 (TSC-40)*, the *Beck Depression Inventory (BDI)*, one subscale, State Anxiety from the *State-Trait Anxiety Inventory (STAI)*, the *State-Trait Anger Expression Inventory (STAXI)*, one subscale, the State guilt subscale from the *Guilty-Inventory (GI)*, the *Coopersmith Self-Esteem Inventory* and the *Survey of Personal Beliefs (SPB)*. The findings reflect that cognitive restructuring was very effective “in facilitating for recovery on anxiety, depression and anger, but less effective for guilt and low self-esteem” (Möller & Steel, 2002, p. 49). If the perpetrator was a close family member, there was a poor response to treatment, but if the perpetrator was a friend or stranger, there was recovery. Overall, cognitive restructuring was very effective in reducing State anxiety and in reducing levels of depression and State anger, although to a lesser extent. Recovery was less for Self-esteem (69%) and State guilt (77%).

In Möller & Steel’s study there were significant reductions in depression, State anxiety, State anger, State guilt and low self-esteem from pre- to posttreatment. Rational-emotive behavior therapy was used to bring about change. A further area for investigation was hypothesized, namely that recovery from the emotional sequelae of childhood sexual abuse may be “associated with less severe abuse (no penetration) of longer duration” and that “more severe abuse (even of shorter duration) predicts a poorer response to treatment”. A “poor response to treatment was associated with the perpetrator being a close family member and with more Other-directed Shoulds”, while recovery “was associated with the perpetrator not

being a close family member (friend or stranger), and with more Awfulizing, Self-directed Shoulds and negative Self-worth beliefs” (Möller & Steel, 2002, p.60).

In a cognitive trauma therapy study for battered women (CTT-BW), done by Kubany, Owens, Iannace-Spencer, McCraig, Tremayne et al. (2004), PTSD remitted in 87% of women who completed the treatment and there were large reductions in depression and guilt and substantial increases in self-esteem. Similar treatment outcomes were obtained by male and female therapists with varying levels of education and training. Gains were maintained at 3- and 6-month follow-ups. This study represents the first treatment-outcome research targeting posttraumatic stress in battered women. Sixty-nine percent of participants achieved good end-state functioning, that is, absence of PTSD and depression, after completing CTT-BW which is comparable to results obtained in a treatment-outcome study of cognitive processing therapy and prolonged exposure with samples of rape victims done by Resnick et al., in 2002, cited in Kubany et al., (2004).

Of the women who completed CTT-BW, 85% no longer met the DSM-IV PTSD symptoms of numbing/avoidance. The treatment was efficacious across an educationally and ethnically diverse group of women. Effective results were achieved by therapists with no formal psychotherapy training, and two of the therapists had only baccalaureate degrees. These findings may have important implications for public health because the majority of victim service providers who counsel and support groups for battered women are paraprofessionals who have no formal training in psychological or psychiatric counseling. Such individuals represent a large group of people who could be trained to conduct CTT-BW.

Another finding that may enhance the significance of the study by Kubany et al. (2004) is that efficacious results were obtained by male therapists as well as by female therapists. In most treatment-outcome studies of PTSD in abused women, the therapists have all been women. Therefore, the findings of this study,

suggests that male therapists may not be sufficiently utilized in PTSD programs for women and indicates that research that examines the effects of therapist gender in treatments of abused women should be undertaken.

A limitation on the generalisability of the findings of the study by Kubany et al. (2004) relates to the study inclusion criterion that women had to report at least moderate abuse-related guilt to be eligible to participate. Because guilt is a central construct in the conceptualization of posttraumatic stress and is one of the major treatment components and outcome measures in CTT-BW, it may be important to clarify the meaning of guilt and how guilt relates to other cognitive constructs implicated in posttraumatic stress. Research that examines the convergent and discriminant validity of guilt cognitions with regard to other cognitive constructs may expand the understanding of the role of cognitions in posttraumatic stress.

Blanchard, Hickling, Devineni, Veazey, Galovski, Mundy, Malta and Buckley (2003) did a controlled evaluation of cognitive behavioural therapy (CBT) for posttraumatic stress in motor vehicle accident survivors. They found a significantly greater improvement for those in CBT in comparison to supportive psychotherapy (SUPPORT) and a Wait List control condition. In addition to improvement in PTSD, the CBT condition led to significantly greater reductions in co-morbid conditions, namely in major depression and generalized anxiety disorder (GAD). The results showed that 76.2% of those with PTSD treated with CBT had improved, compared to 47.6% of those treated with supportive psychotherapy, and to 23.8% of those on the Wait List. The fact that co-morbid conditions were not specifically targeted yet led to significant reductions in depression and GAD in the CBT condition, implies that significant benefit in PTSD symptoms generalizes to other conditions.

Seventy six percent of those with PTSD who were treated with CBT in the study by Blanchard et al. (2003) no longer qualified for this diagnosis at post-treatment.

Both men and women suffering from PTSD from the same kind of trauma responded equally well to treatment. This study clearly showed the benefits of CBT as a treatment for PTSD as a result of motor vehicle accidents.

In a study undertaken by Cohen, Deblinger, Mannarino and Steer (2004), trauma-focused cognitive-behavioural therapy (TF-CBT) was compared to child-centred therapy (CCT) for treating posttraumatic stress disorder and related emotional and behavioural problems in children who had suffered from sexual abuse. The findings reflected that those children assigned to TF-CBT, compared to child-centred therapy, improved more with regard to PTSD, depression, behavioural problems, shame and abuse-related attributions. In addition, parents who were given TF-CBT also showed more improvement with regard to depression, distress related to their children's abuse, support of their child and effective parenting.

In a study undertaken by Cohen, Deblinger, Mannarino & Steer (2004) two sites were used, namely a large metropolitan area and a suburban setting. Subjects were randomly assigned to either TF-CBT or child-centred therapy (CCT). The TF-CBT model included elements of "skills in expressing feelings; training in coping skills; recognizing the relationships between thoughts, feelings, and behaviors; gradual exposure (also referred to as creating the child's trauma narrative); cognitive processing of the abuse experience(s); joint child-parent sessions, psychoeducation about child sexual abuse and body safety; and parent management skills". (Cohen, Deblinger, Mannarino & Steer, 2004, p. 398).

Future areas for research arise out of the positive treatment response to TF-CBT in multiply traumatized children. There may be more similarities than differences among children who develop PTSD, therefore similar treatment interventions may be effective for children who have been traumatized by different kinds of traumatic events.

Combination therapies

Studies have examined whether treatment efficacy can be enhanced by combining individually effective treatments. Two studies have explored this possibility: Foa et al (1997), cited in Foa & Meadows(1998) compared PE alone, SIT alone, the combination of PE and SIT, and a wait-list control; Marks et al. (in press at the time), cited in Foa & Meadows (1998) compared PE alone, cognitive restructuring (CR) alone, the combination of PE and CR, and relaxation only (R). The results of both studies did not support the use of combination treatments over individual treatment components, (Zoellner et al., cited in Wilson, Friedman & Lindy, 2001).

According to Foa & Meadows (1998), however, cognitive processing therapy (CPT), which combines components of exposure and cognitive therapy, was found to be efficacious in the treatment of rape-related PTSD. Therefore, it seems that certain combined therapies may be effective for specific trauma-related experiences.

In a study by Bryant, Moulds, Guthrie, Dang and Nixon (2003), the extent to which providing cognitive restructuring (CR) with prolonged imaginal exposure (IE) would lead to greater symptom reduction than providing IE alone for participants with posttraumatic stress disorder (PTSD) was investigated. Their findings were consistent with the proposition that both IE and CR are effective strategies for PTSD and that adding these treatment components would provide more effective gains than IE alone. These results contrasted with the findings of Foa, Dancu, et al. (1999); Marks et al. (1998); Resnick et al.(2002), according to Bryant, Moulds, Guthrie, Dang, and Nixon (2003).

Previous studies to the one under discussion used in vivo exposure in their protocols, while Bryant et al. (2003) limited their protocol to imaginal exposure. They speculated that their combined treatment achieved greater symptom

reduction as their IE had limited treatment efficacy because it omitted in vivo exposure.

According to Marks (2000), cited in Bryant et al. (2003) IE and CR may involve common elements, including processing of emotional memories, integration of corrective information, and development of self-mastery. It was also posited that CR led to greater symptom reduction because it addressed identification and modification of maladaptive cognitions that may contribute to the maintenance of PTSD and associated problems (Ehlers & Clark, 2000, cited in Bryant et al., 2003).

A need for further study concerning the respective roles that IE and CR have to play in reducing PTSD and associated conditions following trauma is urged by Bryant et al. Further research is needed to determine the extent that treatment effectiveness can be enhanced by combining IE and CR together.

Conclusion

In conclusion, this review has explored the conceptualization of PTSD which has been problematic since its inception. It has reflected the official recognition by the APA of the syndrome in 1980. The conflict between seeing victims as suffering from a normal reaction to an abnormal event and being characterized as a psychiatric illness has been elucidated. The fact that what constitutes a traumatic event is as difficult to define as the syndrome of PTSD has been mentioned.

Biological studies have helped to clarify PTSD as a particular type of stress response that is characterised by a progressive sensitization of biological systems. PTSD is characterised by enduring neurophysiological changes. Psychological and physiological aspects are inextricably intertwined, as posited by Shalev (2003).

The cognitive processes of PTSD have been examined with regard to dissociation with its many components of depersonalization, derealisation, amnesia and time distortion. How traumatic memories are processed and stored have been dealt with under the headings of traumatic amnesia, global memory impairment, dissociative processes and sensorimotor organization. That traumatic memory is stored in a fragmented way has been explained.

Cognitive behavioural treatment for PTSD includes prolonged exposure, stress inoculation training, cognitive restructuring, cognitive processing therapy and eye movement desensitization and reprocessing. These have all been shown to be highly effective forms of treatment, with exposure as the most effective and the findings for EMDR as inconclusive.

Hypnosis has been established as assisting with dealing with dissociative symptoms. Exposure involves the confrontation of feared stimuli imaginally or in vivo . Prolonged exposure has found more empirical support for its efficacy than any other treatment for PTSD. Eye movement desensitization reprocessing has been found to be the most controversial of all the PTSD therapies because the findings have been methodologically flawed, however civilian studies have reported a reduction in PTSD symptoms 3-10 hours after treatment was assumed. EMDR component analyses that were carried out generally provide inconclusive results, suggesting that clinically and scientifically valid research is indicated before the relative importance of components of EMDR can be established.

Anxiety Management Training aims at providing patients with strategies to deal with anxiety. One of the most studied AMT programs for PTSD is Stress Inoculation Training (SIT) which incorporates a number of components. Several uncontrolled studies have found SIT to be one of the most effective AMT therapies for PTSD.

Cognitive therapy which alters erroneous and maladaptive thoughts, beliefs and traumatic memories related to PTSD, asserts that individuals have particular ways of thinking about the world, other people and themselves. It has been found to assist with avoidance and helps to reduce symptoms of anxiety. Cognitive therapy helps the patient dismiss unwanted thoughts and to substitute these with more adaptive thoughts or images that are based on reality.

Finally combination therapies were not found to be more effective than individual treatment components, except in the instance of rape-related PTSD where combining exposure and cognitive therapy was found to be efficacious.

Posttraumatic Stress disorder has come to be recognized as a discrete disorder, characterised by a number of components, which can occur as a result of a range of stressful events. It has been established as an area of study warranting further clinical and scientific research so that the various methodological flaws that have occurred to date, owing to its complicated nature, can be addressed.

REFERENCES

American Psychiatric Association (2000). *Diagnostic and Statistical Manual of Mental Disorders. Fourth Edition. Text Revision (DSM-IV-TR)*. Washington DC: American Psychiatric Association.

Baum, A., Soloman, S., & Ursano, R., with Bickman, L., Blanchard, E., Green, B., Keane, T., Laufer, R., Norris, F., Reid, J., Smith, E., & Steinglass, P. (1993). Emergency/disaster studies practical, conceptual, and methodological issues. In Wilson, J.P. & Raphael, B. (Eds.), *International handbook of traumatic stress syndromes* (pp. 125-133). New York, NY: Plenum.

Blanchard, E., Hickling, E., Devineni, T., Veazey, C., Galovski, T., Mundy, E., Malta, L., & Buckley, T. (2003). A controlled evaluation of cognitive behavioral therapy for posttraumatic stress in motor vehicle accident survivors. *Behaviour Research and Therapy*, *41*, 79-96.

Bryant, R.A., Moulds, M.L., Guthrie, R.M., Dang, T., & Nixon, R.D.V. (2003). Imaginal exposure alone and imaginal exposure with cognitive restructuring in treatment of posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, *71*(4), 706-712.

Charney, D., Deutch, A, Krystal, J, Southwick, S. M., & Davis, M. (1993) Psychobiologic mechanisms of posttraumatic stress disorder. *Archives of General Psychiatry*, *50*, 295-305.

Cohen, J.A., Deblinger, E., Mannarino, A.P., & Steer, R.A. (2004). A multisite, randomized controlled trial for children with sexual abuse-related PTSD symptoms. *Journal of the American Academy of Child and Adolescent Psychiatry*, *43*(4), 393-402.

Foa, E., & Cahill, S. (2002). Specialised treatment for PTSD. Matching survivors to the appropriate modality. In R.Yehuda.(Ed.) *Treating trauma survivors with PTSD*. (pp. 43-62). Washington, DC: American Psychiatric Publishing, Inc.

Foa, E., & Meadows, E. (1998). Psychosocial treatments for posttraumatic stress disorder. In R. Yehuda. (Ed.). *Psychological trauma*. (pp.179-204). Washington, DC: American Psychiatric Press, Inc.

Foa, E., Zoellner, L., Feeny, N., Hembree, E., & Alvarez-Conrad, J. (2002). Does imaginal exposure exacerbate PTSD symptoms? *Journal of Consulting and Clinical Psychology*, 70 (4), 1022-1028.

Joseph, S., Williams, R., & Yule, W. (1997). *Understanding post-traumatic stress*. Chichester: John Wiley & Sons Ltd.

Kubany, E.S., Hill, E.E., Owens, J.A., Iannce-Spencer, C., McCraig, M.A., Tremayne, K.J. et al. (2004). Cognitive trauma therapy for battered women with PTSD (CTT-BW). *Journal of Consulting and Clinical Psychology*, 72(1), 3-18.

Lytle, R.A., Hazlett-Stevens, H., & Borkovec, T.D. (2002). Efficacy of eye movement desensitization in the treatment of cognitive intrusions related to a past stressful event. *Journal of Anxiety Disorders*, 16, 273-288.

McFarlane, A. (2003). Early reactions to traumatic events. The diversity of diagnostic formulations. In R. Ørner, & U. Schnyder (Eds.), *Reconstructing early intervention after trauma* (pp.50 -52). New York: Oxford University Press.

Möller, A.T., & Steel, H. (2002). Clinically significant change after cognitive restructuring for adult survivors of childhood sexual abuse. *Journal of Rational-Emotive & Cognitive Behaviour Therapy*, 20(1), 49-64.

National Institute of Mental Health (NIMH) (2001). *Reliving Trauma: Post-traumatic stress disorder*. Retrieved January 27, 2004 from World Wide Web: <http://www.nimh.nih.gov/publicat/reliving.cfm>.

Ørner, R., & Schnyder, U., (Eds.) (2003). *Reconstructing early intervention after trauma*. New York: Oxford University Press.

Schiraldi, G. (2000). *The post-traumatic stress disorder sourcebook*. Los Angeles: Lowell House.

Scrignar, C.B. (1996). *Post-traumatic stress disorder*. New Orleans: Bruno Press.

Shalev, A. Y. (2003). Psychobiological perspectives on early reactions to traumatic events. In R. Ørner, & U. Schnyder (Eds.), *Restructuring early intervention after trauma. Innovations in the care of survivors* (pp. 56-63). New York: Oxford University Press.

Shapiro, F. (2002). EMDR 12 years after its introduction: past and future research. *Journal of Clinical Psychology*, 58(1), 1-22.

Van der Kolk, B. & Fisler, R. (1995). Dissociation and the fragmentary nature of traumatic memories: overview and exploratory study, pp. 1-14. Retrieved June 17, 2002 from the World Wide Web: http://www.trauma_pages.com/vanderk2.htm.

Wilson, J., Friedman, M., & Lindy, J. (Eds.) (2001). *Treating psychological trauma & PTSD*. New York, London: The Guilford Press.

Yehuda, R. (Ed.) (1998). *Psychological trauma*. Washington DC: American Psychiatric Press, Inc.

Yehuda, R. (Ed.) (2002). *Treating trauma survivors with PTSD*. Washington DC, London, England. American Psychiatric Publishing, Inc.

Yehuda, R., & McFarlane, A. (1995). Conflict between current knowledge about posttraumatic stress disorder and its original conceptual basis. *American Journal of Psychiatry*, 152 (12), 1705-1713.

Young, A. (1995). *The harmony of illusions: inventing post-traumatic stress disorder*. Princeton, New Jersey: Princeton University Press.

Yule, W. (Ed.) (1999). *Post-traumatic stress disorders*. Chichester: John Wiley & Sons.

Zoellner, L., Feeny, N., Cochran, B., & Pruitt, L. (2003). Treatment choice for PTSD. *Behaviour Research and Therapy* (41), 879-886.

Zoellner, L., Fitzgibbons, L., & Foa, E. (2001). Cognitive-behavioural approaches to PTSD. In J. Wilson, M. Friedman & J. Lindy (Eds.), *Treating psychological trauma & PTSD* (pp. 159-182). New York, London: The Guilford Press.