The Efficacy of Eye Movement Desensitization in the Treatment of Trauma Related Imagery and Cognitions: A Partial Dismantling Procedure

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THE EFFICACY OF EYE MOVEMENT DESENSITIZATION IN THE TREATMENT OF TRAUMA RELATED IMAGERY AND COGNITIONS: A PARTIAL DISMANTLING PROCEDURE

by

George S. Renfrey

A Dissertation
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THE EFFICACY OF EYE MOVEMENT DESENSITIZATION IN THE TREATMENT OF TRAUMA RELATED IMAGERY AND COGNITIONS: A PARTIAL DISMANTLING PROCEDURE

George S. Renfrey, Ph.D.

Western Michigan University, 1993

This study investigated the effects of eye movement desensitization (EMD) on post-traumatic sequelae, and attempted a partial dismantling of the procedure to determine the necessity of EMD's characteristic eye movements. Twenty-three persons participated in three groups: (1) those receiving standard EMD, (2) those receiving a variant of EMD in which eye movements were engendered through a light tracking task, and (3) those receiving a variant of EMD in which fixed visual attention replaced eye movements. All participants had experienced traumata as defined by the DSM-III-R and were having intrusive symptoms of PTSD at pre-treatment. All but two met full DSM-II-R criteria for PTSD. Each received two to six treatment sessions.

Dependent variables included heart rate changes, subjective units of distress ratings, validity of both initial and targeted trauma-related cognitions during trauma-related imagery, overall frequency and intensity scores on the Clinician Administered PTSD Scale, anxiety and depression T-scores on the Symptom Checklist (SCL-90-R), and scores on the Impact of Events Scale. Assessments were conducted at pre- and post-treatment and at a one- to three-month follow-up.

All three interventions produced significant, positive changes in all dependant...
measures between pre- and post-treatments. Further, these changes were maintained at follow-up. No significant differences between groups were observed. These changes were of comparable magnitude to those reported elsewhere, but were brought about through a greater number of treatment sessions.

It was concluded that EMD does bring about fairly rapid therapeutic changes in those post-traumatic sequelae measured, though not as efficiently as most previous reports have suggested. Further, it was concluded that the eye movements peculiar to EMD are not a necessary component of the procedure. The similarities and differences between the present findings and previous reports are discussed, as are the limitations and implications of the present study. Recommendations for future work are made.
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The efficacy of eye movement desensitization in the treatment of trauma related imagery and cognitions: A partial dismantling procedure

Renfrey, George S., Ph.D.

Western Michigan University, 1993
ACKNOWLEDGEMENTS
(English translation found in Appendix E)

Nda miigwechwi'aa n'wiidagemaagan Lyn Raible, gii gnabenmid miinwaa gii aasgaabwitwid epiichi gchi-skonwiyaanagh memdigeego gii mi-miskwiigdewendaagog maanda wii giizhtoyaanh gaa zhibiimaa. Giishpin maaba n'wiidagemaagan bwaanaad-mopa gaawin ndaa gii giizhtosiin maanda gaazhi-skonwiyaanagh.


Suzanne Cross geye ngichi-shpenmaa gii naadmowid wii mino-gnwaabmiwaad miinwaa wii mino-gnwaabmagwaa E'wiijinokiimgog, miinwaa Anishnaabe powaagnan egnwenmaajig gii naadmowaad wii bmoseyaanh maampii msko-miknaang, Miinwa Helen Roy, gii kinomowid wii Anishnaabemyaanh, maanda nango wii shkitoyaanh wii zhibiimaa Anishnaabeg dinwe'wnaaawaa.

George S. Renfrey
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CHAPTER I

INTRODUCTION

Post-Traumatic Stress and Treatment

It has been recognized for centuries that extremely stressful events may result in enduring psychological sequelae. Such post-traumatic stress reactions (PTSR) may include symptoms of reexperiencing aspects of the trauma, and symptoms indicative of both hyperarousal/hyperreactivity and psychosocial avoidance or numbing. Additionally, PTSD may be accompanied by a number of comorbid conditions and can be psychologically and socially debilitating. Presently, the most severe type of formally acknowledged PTSD is referred to as post-traumatic stress disorder (PTSD).

According to the review presented in Chapter II, PTSD may follow exposure to a broad class of events. Between 1.0% and 2.6% of the general population in this culture are at risk of developing it in their lifetime (Davidson & Fairbank, 1993), and a considerably greater number of people can be expected to experience a less severe PTSD. Though a number of pharmacological agents appear to have a palliative effect on some symptoms of PTSD, it has been asserted that pharmacotherapy is insufficient by itself, that some form of psychotherapy is required in the treatment of PTSD (e.g., Friedman, 1988; Peterson et al., 1990). An array of psychotherapies for PTSD exist, and they often differ in technique, focus, and the presumed nature of post-traumatic
It is generally thought, however, that the treatment of PTSD can be a long and difficult process.

Eye Movement Desensitization

A new treatment for post-traumatic sequelae, called eye movement desensitization (EMD), has been reported in the literature by Shapiro (1989a, 1989b) (see Chapters II and III for a complete description). These initial reports (Shapiro, 1989a, 1989b) suggest that EMD is more effective and efficient in treating the intrusive symptoms of PTSD than more established treatments. As such, it may represent a therapeutic breakthrough which could save considerable costs in both therapeutic services and human distress. Though a few subsequent reports seem to support this opinion, Herbert and Mueser (1992) point out serious methodological flaws in most of these, hence research to date does not permit an informed assessment of its efficacy. Replication by well controlled outcome studies are needed to firmly establish EMD as an effective treatment for PTSD, but for now it would appear that EMD holds promise as a treatment for at least some symptoms of PTSD.

A number of important questions about EMD present themselves at this time. As mentioned, Shapiro's results must be reliably replicated by independent parties to establish its treatment efficacy. Another question that presents itself regards those components of the EMD procedure that are necessary and sufficient to produce the reported reductions in post-traumatic sequelae. The EMD procedure outlined in the literature is complex enough that it may be deemed a compact treatment package. If
EMD does prove to be as effective an intervention as it appears, it would be desirable to determine the mechanisms responsible. To investigate such mechanisms it may be fruitful to employ dismantling procedures on the EMD procedure.

Another issue is a technical one. The EMD procedure involves the rapid movement of a therapist's hand across the visual field of the client. Because such movements are unavoidably varied and imprecise, well controlled and standardized investigations of EMD, especially those which might probe into the psychophysiological correlates of successful treatment, may be impaired. If a means of establishing more precise control over the generation of eye movements with no reduction in therapeutic efficacy can be found, well controlled investigations may be made possible. A number of other questions about the apparent effects of EMD will be discussed in more detail in chapter three.

Statement of Purpose

The purpose of the present project was to answer a number of questions about the efficacy and mechanisms of EMD that present from the literature, including those mentioned above. Since an understanding of such may benefit from an approach within the context of existing knowledge in the field, we begin with a review of the literature on PTSR and its treatment. The interpretations or models of PTSR from diverse schools of thought are surveyed as are varied pharmacological and psychological treatments. A fuller explanation of the rationale for the present study is presented in Chapter II, and the methodology is presented in Chapter III. The results of the present study are presented
in Chapter IV, and are then discussed in the context of current understandings of PTSR and its treatment in Chapter V.
CHAPTER II

REVIEW OF RELATED LITERATURE

Introduction to Post-Traumatic Stress Reactions

This chapter reviews the literature on post-traumatic stress disorder in adults. The recognition of post-traumatic stress reactions is reviewed in brief, as is the evolution of etiological concepts. Next, pre-traumatic, traumatic, and post-traumatic risk factors are surveyed, and the symptomatology of post-traumatic stress reactions is discussed. Physiological and psychological explanatory schemes are then reviewed, the latter roughly following major theoretical schools. Finally, both pharmacological and psychological treatments and evidence of their efficacy are examined.

History of Post-Traumatic Stress Reactions

Post-traumatic stress reactions (PTSR) belong to a symptom complex that can vary from brief, mild reactions to stressful events, to chronic debilitating disorders. The character of these reactions might best be understood by an initial exploration of the more severe end of the traumatic stress response spectrum, by an examination of post-traumatic stress disorder (PTSD).

Post-traumatic stress disorder (PTSD) is a DSM-III-R (APA, 1987) diagnostic-category that is characterized by an exposure to a severely distressing event, outside the
range of usual human experience, followed by the exhibition of: (a) recurrent dreams, recollections, or flashbacks of that event and/or discomfort when exposed to stimuli associated with the event; (b) psychosocial numbing and/or avoidance of trauma relevant stimuli; and (c) symptoms indicative of chronically elevated sympathetic arousal. Although this disorder has only been included in the diagnostic system of the American Psychiatric Association since 1980, evidence suggests that similar patterns of post-traumatic sequelae have been recognized for centuries.

Trimble (1985) cites anecdotal evidence of post-traumatic sequelae in diaries dating to the writings of Shakespeare and Dickens, and to the great fire of London in 1662. In Shakespeare's King Henry IV, for example, Hotspur is reported to have nightmares of war. After surviving a railway accident in which many people were injured and killed, Charles Dickens wrote in his diary about feeling weak some time after the accident, as if from a protracted illness, and of developing a phobia of railway travel. Trimble (1985) cites Daly's (1983) recounting of Samuel Pepys' diary following the great London fire. Pepys wrote of not being able to sleep for many months without nightmares of conflagrations, and made reference to the attempted suicides of other survivors.

Despite their longstanding literary and civil recognition, post-traumatic stress reactions did not attract significant medical / psychological interest until the late 19th century, when they made their effects known en masse following the American Civil War and other military conflicts. A debilitating condition referred to as "neurasthenia" was observed in many who survived the extreme deprivations of industrial-era warfare.
Survivors were often referred to euphemistically as "broken men" in reference to their emotional and nervous fragility. DaCosta wrote about a peculiar cardiac malady frequently found in U.S. Civil War survivors which he called "Irritable Heart" (DaCosta, 1871). It was characterized by a variety of cardio-pulmonary and gastro-intestinal symptoms that seemed to have no identifiable physical cause, yet are similar to symptoms of chronic anxiety with acute exacerbations (Wood, 1941). This professional attention received a further boost when the explosive increase in railway use for passenger travel in Europe during the latter half of the 19th century resulted in numerous railway accidents. This gave rise to civilian claims for compensation following such accidents, an increase in medical and legal interest in post-traumatic sequelae, and the subsequent use of the term "compensation neurosis." Though some thought the associated "nervous prostration" was due to mechanical insults to the spine (see Erichsen, 1882), others ascribed it to "nervous shock" (e.g., Page, 1885), speculating that the nightmares, increased nervousness, and railway fears of survivors did not result from mechanical insult to the nervous system at all.

A similar debate about the etiology of post-traumatic stress reactions occurred during and following the First World War. At that time, soldiers exposed to severe combat related stress were observed to develop a symptom complex characterized by: repetitive battle-related nightmares, muteness, paralysis, blindness, confusion, reduced responsiveness to human contact, emotional numbness, memory loss, exaggerated reactions to sudden noises or movements, uncontrollable weeping, restlessness, insomnia, phobias, and/or high levels of anxiety, (Ettdgui & Bridges, 1985; Herman,
1992). Such reactions were initially referred to as "shell shock" (see Mott, 1919), reflecting the wide belief that the condition resulted from neurological damage by explosive shock waves and/or carbon monoxide poisoning. However, the observation that many soldiers not exposed to shelling or gassings developed shell shock could not be reconciled with this position and it was eventually dropped. What replaced it was the vague attribution of shell shock to severe emotional stress. It was still assumed by many, however, that individuals who developed the disorder were constitutionally predisposed to it. In the years following the war, professional interest in post traumatic sequelae diminished, but it became apparent that reactions to severe stress could prove chronic.

The Second World War saw even greater attention to the effects of trauma. Given the toll that shell shock took on allied soldiers during the First World War, accounting for as much as 40% of British battle casualties (Herman, 1992), great effort was spent during the Second World War to devise predictors and treatments for "battle fatigue" as it was then called. Synonymous terms from this era include "war neurosis," "combat stress," "combat fatigue," and "combat exhaustion." As casualties mounted and no useful predictors of vulnerability were determined, it became evident that anyone could succumb to battle fatigue given sufficient stress, and that definitive intervening variables were elusive and complex. The prevalence of PTSR was found to be roughly proportional to the degree of stress endured (Appel & Beebe, 1946), and inversely proportional to the social cohesiveness and morale of the immediate fighting unit (Grinker & Spiegel, 1945). The recognition of PTSR's psychological origins resulted in a concerted effort by the U.S. Government to expand psychology and psychiatry training programs and
treatment facilities during and after the war to service veterans.

It was during the Korean War that the diagnosis of "Gross Stress Reaction" made its way into the first edition of the American Psychiatric Association's Diagnostic and Statistical Manual, the DSM-I (APA, 1952). According to the criteria of the DSM-I, Gross Stress Reaction was considered a transient state that could either abate on its own, respond rapidly to treatment, or progress into a major neurotic or psychotic condition. Since that time, prior to the current terminology, patterns of post-traumatic stress reactions to a variety of traumatic events have been identified and variously labeled as "survivor's syndrome," "traumatic neurosis," "stress response neurosis," "posttraumatic psychoneurosis," and "rape-trauma syndrome" (Ettedgui & Bridges, 1985; Holmstrom & Burgess, 1975; Ramsay, 1990; Trimble, 1985). Despite the variants in terminology, the etiology and symptoms associated with each suggest a common syndrome that can develop following extreme and unusual distress.

The current diagnostic category, PTSD emerged following the Vietnam War. In response to the large number of affected combat veterans returning from the war over a ten year period, and to demands from veteran's support groups and families, the Veterans Administration launched intensive studies of PTSR during the early seventies (Herman, 1992). What eventually emerged from this renewed clinical and research interest is the inclusion in the 3rd edition of the American Psychiatric Association's Diagnostic and Statistical Manual, the diagnosis "post-traumatic stress disorder" (APA, 1980). These diagnostic criteria, and their revised form have since guided clinical and research endeavors. Ongoing investigations have lead to recent distinctions between
immediate, transient stress reactions versus delayed, more persistent stress reactions, and between simple versus complex forms of PTSD (Davidson & Foa, 1993). These distinctions may well become part of the diagnostic criteria of the APA's DSM-IV, now in preparation, and will be discussed later in the text.

Summary

Post-traumatic stress reactions have been recognized to some extent for centuries. It has only been during the last 75 years, and particularly within the last 20 years, that formal efforts have been made to understand the etiology of such reactions. With these efforts, the nosology for PTSR has evolved to reflect increased understanding and continues to do so. Currently, the most severe form of PTSR and the only specific post-trauma syndrome formally recognized at this time is referred to as PTSD. The nosology for PTSD is likely to change with the DSM-IV (in preparation), and such changes may recognize a difference between simple and complex forms of the disorder.

Populations at Risk & Risk Factors

According to the first diagnostic criterion for PTSD in the DSM-III-R, PTSD may result from exposure to any event, outside the range of usual human experience, that would produce significant distress in almost anyone. Such events might be expected to include combat, natural and man-made disasters, victimizations by certain classes of crime, and other events wherein the safety of an individual is acutely compromised. An impressive body of literature exists to support such expectations (though see Quarantelli,
1985, for an analysis of existing data that suggests that victims of natural disasters are not as at risk of developing PTSD as once assumed. The severe and prolonged deprivations and terrors of combat may well represent the epitome of trauma, and so it is no surprise that post-traumatic sequelae have been best documented in war veterans, particularly those serving during the Vietnam era (e.g., Blanchard, Kolb, Pallmeyer, & Gerardi, 1982; Goldberg, True, Eisen, & Hendersen, 1990; Pearce, Schauer, Garfield, Ohlde, & Patterson, 1985; Solomon, 1989a, 1989b; Watson, Brown, Kucala, Juba, Davenport, & Anderson, 1993; also see Fairbank, Langley, Jarvie, & Keane, 1981, for a selected bibliography of earlier literature in the area). PTSD has also been well documented among former prisoners of war, (e.g., Goldstein, van Kammen, Shelly, Miller, & van Kammen, 1987; Kluznik, Speed, Van Valkenburg, & Magraw, 1986; Zeiss & Dickman, 1989) and wartime refugees (e.g., Kroll et al., 1989), many of whom endured extreme deprivations for protracted periods.

Events of briefer durations and/or putatively less traumatic are also known to produce significant psychological sequelae. Veterans of the Gulf War, though the conflict was brief and relatively few were exposed to the severe stressors usually associated with combat, have evidenced post-traumatic stress reactions including PTSD (Department of Veterans Affairs, 1991). Police officers involved in shooting incidents, which may be intense, emotional experiences but are of short duration, have been identified as a high-risk group (Gersons, 1989). Similarly, survivors of marine disasters (Leopold, 1963), nightclub fires (Lindemann, 1944), destructive brushfires (McFarlane, 1986a, 1986b), flash floods (Green, Lindy et al., 1990; Titchener & Kapp, 1976;
Titchener, Kapp, & Winget, 1976), volcanic eruptions (Shore, Vollmer, & Tatum, 1989), and other natural disasters (see Quarantelli, 1985, for a bibliography and extended discussion of victim responses to natural disasters) have been known to exhibit significant post-traumatic sequelae.

Post traumatic sequelae have also been identified in victims of sexual assault (e.g., Calhoun, Atkeson, & Resick, 1982; Kilpatrick et al., 1985; Kilpatrick, Saunders, Veronen, Best, & Von, 1987; Kilpatrick et al., 1989; Kilpatrick, Resick, & Veronen, 1981; Veronen & Kilpatrick, 1980; Wirtz & Harrell, 1987), childhood sexual abuse (e.g., Patten, Gatz, Jones, & Thomas, 1989; Rychtarik, Silverman, Van Landingham, & Prue, 1984; Wolfe, Gentile, & Wolfe, 1989), assault (e.g., Kilpatrick et al., 1985, 1987, 1989; Wirtz & Harrell, 1987), burglary (e.g., Himelein, 1989; Kilpatrick et al., 1987, 1989; Wirtz & Harrel, 1987), kidnapping (Terr, 1981, 1983), robbery (e.g., Kilpatrick et al., 1985, 1987, 1989; Wirtz & Harrell, 1987), sniper attacks (Nader, Pynoos, Fairbanks, & Frederick, 1990; Pynoos et al., 1987), and terrorist activity (Hadden, Rutherford, & Merrett, 1978; Shalev, 1992); and in survivors of complicated surgery (Peebles, 1989b), heart attacks (Hartmann & Burgess, 1985), traffic accidents (Brom, Kleber, & Hofman, 1993; McCaffrey & Fairbank, 1985; Quirk, 1985), industrial accidents (Savitsky & Hartsough, 1985), and the homicide of family members (Burgess, 1975).

Several epidemiological studies have estimated the lifetime prevalence rate of PTSD in the general population to be between 1.0% and 2.6% with much higher prevalence rates for special at risk groups (Davidson & Fairbank, 1993). Quite possibly reflecting the overall prevalence of PTSD in some U.S. urban centers, Breslau, Davis,
Andreski, and Peterson (1991) through a random sample of 1007 users of a health maintenance organization, estimated the lifetime prevalence of exposure to traumatic events in a large eastern metropolitan catchment area at 39.1%, and the lifetime prevalence of PTSD at 9.2%. Additionally, Solomon and Canino (1990) have suggested that common yet highly stressful events may also produce some symptoms of PTSD in some individuals. One might reasonably expect a larger portion of the population to be at risk for developing PTSR less severe than PTSD, and indeed Davidson and Fairbank (1993) have estimated that the overall PTSR (including PTSD) prevalence rate at between 8% and 16%. Clearly, a wide range of traumatic events can produce significant sequelae, and a large number of individuals in our culture can be expected to be affected by such during their lifetimes.

Though the search continues for pre-trauma and trauma-related risk factors for the development of PTSD, it is still widely held that the prevalence and severity of PTSR is dependant upon the nature and severity of the traumata and the immediate reactions thereto. Blank (1993) in a review of the literature posits that the severity and duration of PTSR tend to vary with the duration and complexity of traumata, though the correlation is not a perfect one. Similarly, a number of investigations have suggested that a dose-response curve exists between stress severity and stress reactions (e.g., Goldberg, True, Eisen, & Hendersen, 1990; Green, Grace et al., 1990; Green, Lindy et al., 1990; March, 1993; Shore, Tatum, & Vollmer, 1986; Watson et al., 1993; Weisaeth, 1984). Zeis and Dickman (1989), in studying ex-POWs, proposed that PTSR symptom severity does not vary with trauma intensity within individuals, but functions as a threshold
variable beyond which there is no dose response relationship. At the population level, however, one might expect that the greater the trauma intensity, the greater the number of persons for whom this threshold is exceeded. Trauma-related risk factors include perceived life-threat (e.g., Fontana, Rosenheck, & Brett, 1992; Kilpatrick et al., 1987, 1989), physical injury (e.g., Helzer, Robins, & McEvoy, 1987; Kilpatrick et al., 1987, 1989; Winfield, George, Swartz, & Blazer, 1990), proximity to the trauma (e.g., Shore et al., 1986; Pynoos et al., 1987), participation in atrocities (Breslau & Davis (1987a; Green, Grace et al., 1990; Laufer, Gallops, & Frey-Wouters, 1984), bereavement (e.g., Breslau & Davis, 1987a, Shore et al., 1986), and a low level of personal initiation of the traumata (Fontana et al., 1992).

Though available evidence is not consistent, it has been suggested that, controlling for the effects of other psychiatric problems, most pre-trauma risk factors proposed over the years simply function to increase the probability of being exposed to trauma, and hence to developing PTSD (Davidson, Hughes, Blazer, & George, 1991; Helzer, Robins, & McEvoy, 1987; Green, Grace et al., 1990). Some investigators (e.g., Kilpatrick et al., 1989) have found no evidence to support the notion of predisposing factors, yet others have asserted that the incidence of PTSD cannot be correlated with the intensity or duration of stress (Hendin, Pollinger, Singer, & Ulman, 1981). Still, some evidence does suggest that some premorbid factors may increase vulnerability to developing a PTSD in some people in response to some stressors. These include parental poverty (Cordray, Polk, & Britton, 1992; Davidson, Hughes, Blazer, & George, 1991), family history of psychiatric illness (only for low-level trauma) (Davidson, Swartz,
Storck, Krishnan, & Hammett, 1985; Foy, Resnick, Sipprelle, & Carroll, 1987; McFarlane, 1988), recent or concurrent life stress or change (Davidson & Foa, 1993; Solomon, Mikulincer, & Flum, 1988), prior alcohol abuse (Green, Grace et al., 1990, Ruch & Leon, 1983), minority status (Department of Veterans Affairs, 1991; Egendorf, Kadushin, Laufer, Rothbart, & Sloan, 1981, as cited by Foy et al. 1987), an external locus of control (Davidson & Foa, 1993; Harris & Parsons, 1985; Kushner, Riggs, Foa, & Miller, 1992), prior traumatization (e.g., Blank, 1993; Solomon, Mikulincer, & Jakob, 1987; Solomon, Benbenishty, & Mikulincer, 1988), genetic factors (Davidson & Foa, 1993), poor post-trauma social support (e.g., Green, Grace et al., 1990; Horowitz, 1986a, 1986b; Solomon, Mikulincer, Batiafreid, & Wosner, 1987), pre-traumatic psychological adjustment problems (e.g., Atkeson, Calhoun, Resick, & Ellis, 1982; McFarlane, 1990; Weisaeth, 1984), pre-trauma depression with high levels of subsequent traumatic stress (Resnick, Kilpatrick, Best, & Kramer, 1992), irrational attitudes regarding one's needs for security and one's control over events (Horowitz, 1986a, 1986b), low self-and personality factors (Davidson & Foa, 1993; Helzer et al., 1987; Horowitz, 1974, 1986a, 1986b; Levav, Greenfeld, & Baruch, 1979; Reich, 1990).

An examination of the incidence of PTSD among troops returning from the Gulf War has indicated that a number of pre- and post-trauma factors increased the risk of developing PTSD. These included being female, an ethno-cultural minority, a Reserve of National Guard troop called up with little warning, being the parent of a young child, and suffering economic hardship or family turmoil upon returning (Department of Veterans Affairs, 1991). Though this is an interesting finding and calls for further
investigation, it seems reasonable that most of these factors would decrease the psychological preparedness, increase the general anxiety felt by such individuals throughout the conflict, or compromise post-conflict adjustment. Some factors appear to offer protection against developing post-trauma sequelae. These include a strong internal locus of control, good post-trauma social support, and a high degree of sociability and good pre-morbid social relationships (Gibbs, 1989; Herman, 1992), and, in some cases, pre-existing neurotic states that cause the individual to habituate to the effects of stress (Horowitz, 1986a, 1986b). Similarly, pre-traumatic stress may enable individuals to develop coping strategies that offer psychological protection from traumata (Solomon, Mikulincer, & Flum, 1988; Watson et al., 1993).

Summary

According to this review, PTSR may result from exposure to a wide range of events, and a sizable number of people in this culture are at risk of developing a PTSR in their lifetime. The relationship between exposure to extreme stress and both pre- and post-traumatic influences in the etiology of PTSR is not yet clear. What seems to be the most reasonable position, however, was iterated by Davidson and Foa (1993). According to their summary of the available literature, there is a dose response relationship between trauma intensity and response severity, but premorbid factors can modify an individual's response, particularly at lower levels of trauma. Premorbid factors appear to act idiosyncratically, and can increase or decrease an individuals susceptibility to developing a PTSR subsequent to trauma. Similarly, some post-traumatic factors appear
to increase or decrease the likelihood of developing clinical sequelae and to promote or retard recovery.

**Symptomatology of Post Traumatic Stress Reactions**

**Post Traumatic Stress Disorder**

As indicated earlier, the DSM-III-R (APA, 1987) diagnosis of PTSD can be considered a severe form of PTSR. As such, the symptomatology of PTSD will be considered here. The 17 cardinal symptoms of PTSD are clustered into 3 groups: (1) recurrent dreams, recollections, or flashbacks of that event and/or discomfort when exposed to stimuli associated with the event; (2) psychosocial numbing and/or avoidance of trauma relevant stimuli; and (3) symptoms indicative of chronically elevated sympathetic arousal. To meet current criteria for PTSD an individual must, subsequent to experiencing a severe trauma, exhibit clinically significant levels of one of four Article B symptoms (#1 above), three of seven Article C symptoms (#2 above), and two of six Article D symptom (#3 above) for a period of at least one month. The severe trauma must involve an event that is "outside the range of usual human experience and that would be markedly distressing to almost anyone" (APA, 1987, p. 250). When symptoms develop within six month post-trauma, the disorder is considered acute. When they develop six months or more post-trauma, the disorder is considered delayed. When symptoms persist for six months or more, PTSD is considered chronic.
Other PTSR

Though PTSD may be the most severe of the uncomplicated PTSR, it has become apparent that PTSR may occur along a continuum of severity. In a review of the literature, Blank (1993) argues that PTSR which do not meet diagnostic criteria for PTSD probably occur frequently, both early post-trauma and many years post-trauma. Such reactions can be thought of as sub-threshold PTSD (ST-PTSD). Support for the existence of ST-PTSD has been found in studies of survivors of a number of traumata including natural disaster (e.g., Green, Lindy et al., 1990; McFarlane, 1986a, 1986b) and combat (e.g., Solomon, 1989a, 1989b; Solomon, Garb, Bleich, & Grupper, 1987; Solomon, Kotler, Shalev, & Lin, 1989; Solomon & Mikulincer, 1987, 1988; Solomon et al., 1987, 1988; Solomon, Weisenberg, Schwarzwald, & Mikulincer, 1987).

Immediate, debilitating stress reactions are also known to occur. Solomon and colleagues (Solomon, 1989a, 1989b; Solomon & Mikulincer, 1987, 1988; Solomon et al., 1987, 1988; Solomon, Weisenberg, et al., 1987), have observed that immediate combat stress reactions (CSR) are variable and often diffuse in character. Symptoms may include restlessness, psychomotor agitation or retardation, withdrawal, confusion, nausea, vomiting, startle reactions, paranoia, panic, and dissociative phenomena (e.g., Solomon, 1988, 1989a, 1989b). The occurrence of a CSR is not necessarily predictive of subsequent PTSD, as many who experience a CSR recover sufficiently after cessation of battle stress to avoid such a diagnosis. However, individuals who experienced a CSR continue to experience higher levels of distress than non-CSR controls for years, and are at elevate risk of developing PTSD (Solomon, 1988, 1989a, 1989b). It therefore seems
reasonable to assume that many persons who seem to recover from CSRs and the like continue to experience a ST-PTSD.

More support for the notion of distinguishing between immediate, brief PTSR and protracted PTSR was provided by Shalev (1992). Shalev studied 12 survivors of a terrorist attack immediately after the trauma and 10 months later. He found that symptoms of intrusion and denial immediately after the event, did not predict the occurrence of PTSD or other psychiatric symptoms at 10 months.

Both ST-PTSD and acute stress reactions may become part of the psychiatric nosology in the upcoming DSM-IV. Blank (1993) suggests that ST-PTSD be included in the DSM-IV as a "condition not attributable to a mental disorder that is [in need of] a form of attention or treatment" (Blank, 1993, p. 14). Brett (1993, p. 201) proposes the category "disorders of extreme stress not elsewhere classified" and includes (a) acute stress disorder, (b) posttraumatic stress disorder, (c) disorders of extreme stress not otherwise specified. The third classification would include a complex form of PSTD to be discussed later. Some authors have noted that PTSR can be reactivated by trauma subsequent to recovery or apparent recovery (e.g., Burgess & Holmstrom, 1974; Lindemann, 1944; Solomon, Garb et al., 1987; Solomon, Kotler et al., 1989).

Reactivated PTSD may present complexities often not appreciated. The role of multiple traumata in the development of a complex form of PTSD will be discussed below, but what is of interest here is the diverse presentations of reactivated PTSD. Solomon and colleagues (Solomon, Garb et al., 1987), in studying reactivated PTSD in Israeli combat veterans recalled to duty, identified four levels of reactivation: (a) uncomplicated
reactivation wherein men who had recovered completely from a prior CSR developed PTSD following reexposure to combat; (b) specific sensitivity, wherein men who retained reactivity to some combat-related stimuli developed PTSD quite readily; (c) moderate generalized sensitivity, wherein residual reactivity had generalized somewhat to stimuli not related to combat and PTSD often developed prior to actual combat reexposure; and (d) severe generalized sensitivity, wherein pervasive lingering symptoms of a CSR are acted upon quite readily by diverse stimuli. What these findings suggest is that post-traumatic conditions (e.g., ST-PTSD) can exist which are not readily identified as a PTSR but which can make the individual vulnerable to subsequent stress.

The Case for Complicated PTSD

Brown and Fromm (1986) proposed a distinction between simple PTSD (S-PTSD) and complicated PTSD (C-PTSD) based upon the nature of the traumatic event and stress reaction. They proposed that the more abominable, prolonged, and repeated the trauma, and the younger the affected individual, the greater the probability of developing a complicated versus a simple PTSR (Brown & Fromm, 1986). They proposed that C-PTSD always represents a dysfunctional or pathological adaptation to stress and further posit that such may be relatively permanent (Brown & Fromm, 1986).

Similarly, Herman (1993) has asserted that current diagnostic systems are based upon clinical studies of survivors of single or otherwise circumscribed traumatic events. She argues that the available categories do not address the pervasive and complex sequelae of repeated, prolonged trauma have on people. In a review of the literature,
Herman (1992, 1993) found authors echoing these sentiments from studies of survivors of both prolonged and extreme social upheaval (e.g., Kroll et al., 1989; Krystal, 1968) and childhood abuse (e.g., Gelinas, 1983; Goodwin, 1988). She goes on to assert that survivors of prolonged, repeated trauma may differ from individuals with simple PTSD in three areas: (1) their symptoms are typically more vague, complex, and resistant to treatment; (2) they often develop personality disturbances, particularly in regard to identity and relatedness to others; and (3) they appear to be vulnerable to repetitious harm, either at their own hands (self-abusive behavior) or at the hands of others (self-defeating behavior) (Green, 1978; Herman, 1993, Lynch & Roberts, cited by van der Kolk, Boyd, & Krystal, 1984).

It has been observed that individuals who were abused as children often present with a particularly large number of clinical symptoms as indicated by responses to symptom checklists (Browne & Finkelhor, 1986; Rowan & Foy, 1993). Similar broadband response tendencies have been observed in holocaust survivors (Niederland, 1968; The symptoms which seem to differentiate C-PTSD from S-PTSD are somatic, dissociative, affective, relational, and behavioral (Herman, 1993). Individuals with C-PTSD often present with significant somatic complaints including: abdominal, pelvic, and back pain; headaches, gastrointestinal complaints, insomnia, and nausea (DeLoos, 1990; Hoppe, 1968, Kinzie et al., 1990; Krystal, 1968; van der Ploerd, 1989). This seems to be especially true of survivors of childhood abuse (Mai & Merskey, 1980; Morrison, 1989). Individuals with C-PTSD are prone to dissociative states (Herman, 1993; Kinzie, Fredrickson, Ben, Fleck, & Karls, 1984; Tennant, Goulston, & Dent, 1986). This may
be manifest in subtle ways through time distortions and concentration lapses (Herman, 1993), or in profound ways as seen in multiple personality disorder (MPD) (Herman, 1993; Putnam, Guroff, Silberman, Barban, & Post, 1986; Ross et al., 1990) and borderline personality disorder (BPD) (Herman, 1992; Herman, 1993; Herman, Perry, & van der Kolk, 1989). The affective features of C-PTSD are profound depression (Hilberman, 1980; Kinzie et al., 1984; Krystal, 1968; Niederland, 1968; Tennant et al., 1986) and periodic rage (Herman, 1993; Hilberman, 1980).

As mentioned, individuals who develop C-PTSD have typically experienced prolonged, repetitive abuse at the hands of other people. Herman asserts that most such abuse follows a predictable pattern across settings and populations, and involves systematic use of psychological terror (Herman, 1993). Two common outcomes of such abuse are de-individualization, wherein victims gradually lose sense of themselves as autonomous, independent human beings, and over identification with the abuser, wherein victims become dependant upon and cling to their abusers (Herman, 1993). De-individualization, especially if it occurs at a young age, can create lasting difficulties with establishing and maintaining a functional and stable self concept (Herman, 1993; Kernberg, 1967; Rieker & Carmen, 1986). The attachments which victims can develop to their abusers are bound to be volatile and ambivalent, shifting between over attachment and rejection. After victimization, survivors seem prone to continue experiencing relationships with others in such extreme forms (Herman, 1993), the epitome of which are BPD and MPD.

Finally, individuals with C-PTSD are prone to experience repetitions of abuse
either at their own hands or through abusive relationships with others (Briere, 1988; Burgess, A. W., Hartman, C. E., McCausland, M. P., & Powers, P., 1984; Herman, 1993; van der Kolk, 1989). Herman (1993) asserts that the evidence for a complex form of PTSD is unsystematic at present, but is compelling nevertheless. The fourth edition of the Diagnostic and Statistic Manual of the American Psychiatric Association (In preparation) may well recognize a complex posttraumatic syndrome under the title Disorder of Extreme Stress Not Otherwise Specified (DESNOS) (Brett, 1993).

Other Symptoms Associated With PTSD

A description of the 17 core symptoms of PTSD does not convey the full impact that traumatic events may have on individuals. Traumatized persons are at elevated risk for depression (e.g., Atkeson et al., 1982; Calhoun, Atkeson, & Resick, 1982; Kilpatrick, Resick, & Veronen, 1981; Kilpatrick, Veronen, & Resick, 1979a, 1979b; Paykel, 1978; Pearce et al., 1985; Santiago, McCall-Perez, Gorcey, & Beigel, 1985; Sierles, Chen, McFarland, & Taylor, 1983), elevated levels of general fearfulness and anxiety (e.g., Calhoun, Atkeson, & Resick, 1982; Kilpatrick, Resick, & Veronen, 1981; Kilpatrick, Veronen, & Resick, 1979; Pearce et al., 1985; Veronen & Kilpatrick, 1980; Wirtz & Harrell, 1987), sleep and REM sleep disturbance (Kinney & Kramer, 1985; Pitman, 1993), social withdrawal and other interpersonal problems (e.g., Atkeson et al., 1982; Davidson et al., 1991; Pearce et al., 1985; Resick, Calhoun, Atkeson, & Ellis, 1982; Solomon & Mikulincer, 1987), suicide attempts (e.g., Kilpatrick et al., 1985; Paykel, 1978), substance abuse (APA, 1987; Keane, Caddell, Martin, Zimering, & Fairbanks,
1983; Sierles et al., 1983), personality disorders (Sierles et al., 1983; Westen, Ludolph, Misle, Ruffins, & Block, 1990), and employment problems (e.g., Davidson et al., 1991; Pearce et al., 1985). Blake et al. (1990), in developing a comprehensive structured interview for PTSD, have included excessive guilt, homocidality, disillusionment with authority, feelings of hopelessness, memory impairment, and feelings of being overwhelmed as associated features of PTSD. It would appear that primary post-traumatic sequelae are complex and varied, may be associated with the development of secondary sequelae in some individuals, and may otherwise be accompanied by comorbid conditions.

**Course of Post Traumatic Stress Reactions**

Blank (1993), in a review of the literature, asserts that there is sufficient evidence to support a diagnostic distinction between acute, delayed, chronic, intermittent, and recurrent forms of PTSD. Green and colleagues (Green, Lindy et al., 1990), in studying survivors of the Buffalo Creek disaster, found that some individuals experienced brief stress reactions, others developed PTSD after a considerable delay in time, and others exhibited a chronic or protracted reaction. This has been observed by other workers in the field (e.g., Herman, 1992; McFarlane, 1986a, 1986b; Solomon, 1989a, 1989b; Solomon, Garb, Bleich, & Grupper, 1987; Solomon, Kotler, et al., 1989; Solomon & Mikulincer, 1987, 1988; Solomon et al., 1987, 1988; Solomon, Weisenberg, et al., 1987).

Acute stress reactions have been observed in most studies of posttraumatic sequelae that were conducted as to be sensitive to them. According to Brett (1993),
"Acute Stress Disorder" is a diagnostic category being considered for inclusion in the DSM-IV. The proposed criteria might help illustrate the nature of acute stress reactions as we understand them from the available literature. According to the Brett (1993), an individual must experience a severe stressor as with Criteria A for PTSD. Symptoms must emerge during or within a few days of the trauma, and must last no more than four weeks. The proposed symptoms include anxiety, hyperarousal, psychosocial numbing, amnesia, trance states, derealization and depersonalization. They do not include reexperiencing symptoms. These symptoms must be severe enough to significantly interfere with the individual's functional capacity.

The essential difference between acute reactions and PTSD, according to Brett, is that the former are acute and time limited. The work of Solomon and colleagues, however, suggest that acute stress reactions may produce residual effects that persist for years yet not meet criteria for PTSD (Solomon, 1989a, 1989b; Solomon & Mikulincer, 1987, 1988; Solomon et al., 1987, 1988; Solomon, Weisenberg et al., 1987). Accordingly, acute stress reactions can be thought of as immediate and brief with possible residual effects that may persist over time.

The onset of symptoms meeting criteria for PTSD may be delayed for weeks, months, or years following a traumatic experience. Some authors have argued that delayed PTSD does not exist; that apparent cases of such represent malingering or the effects of substance abuse and the like (e.g., Sparr & Pankratz, 1983), while others have suggested that cases of apparent delayed PTSD are really cases of delayed diagnosis (e.g., Pary, Turns, & Tobias, 1986). Though both positions may be correct to an extent,
present evidence supports the existence of delayed-onset form of PTSD. Solomon and colleagues have found delayed onset PTSD in both individuals who experienced a battlefield CSR and those who did not, though most cases of apparent delayed onset PTSD represented delayed help-seeking behavior (Solomon, Kotler et al., 1989).

Zeis and Dickman's (1989) notion that traumatic stress functions as a critical event which, if it exceeds an individual's threshold of tolerance, can precipitate PTSD, may be relevant in explaining delayed PTSD. According to this view, sub-threshold traumata may accumulate or have residual effects which enable an individual's threshold to be exceeded by subsequent stress or trauma. This is consonant with the assertion that PTSD can exist which are not readily identified as a PTSD but which can make the individual vulnerable to subsequent stress (Solomon, Garb et al., 1987; Solomon, Kotler et al., 1989). Similarly, it has been observed that traumatized individuals, even those not meeting criteria for PTSD, often experience a post-trauma social decline (e.g., Atkeson et al., 1982; Davidson et al., 1991; Pearce et al., 1985; Resick, Calhoun, Atkeson, & Ellis, 1982; Solomon & Mikulincer, 1987). Solomon and Mikulincer (1987) noted that the development of PTSD was associated with a decline in social functioning, but brief CSR was not. Though cause and effect cannot be established, the notion that a post-traumatic social decline with its attending increase in stress can act upon a lowered PTSD threshold and precipitate an episode some time after the original event is compelling. Whatever the true cause or causes of a delay in onset, delayed PTSD is often severe and may become chronic.

The time course of PTSD appears to vary from case to case, but generally it is
a long-term affliction. For example, the current prevalence of PTSD in Vietnam veterans after an average of 19 years post-trauma has been estimated at 15% (Kulka, Schlenger, Fairbank, Hough, Jordan, Marmar, & Weiss, 1988). Two investigations have estimated that, 40 years post-trauma, the current prevalence of PTSD in World War Two POWs at 50% (Goldstein et al., 1987) and at 47% (Kluznik, Speed, Van Valkenburg, & Magraw, 1986). Long-term post-traumatic sequelae have also been observed in victims of crime (e.g., Kilpatrick et al., 1987) and in physically and sexually abused children (see Herman, 1992).

The relationship between these sub-types of PTSD is not entirely clear. It seems reasonable to assert that CSR (e.g., Solomon, 1988) is roughly equivalent to the Acute Stress Disorder (ASD) diagnosis proposed for the DSM-IV. A number of observations seem relevant here. Solomon and colleagues have observed PTSD and delayed PTSD in both men who experienced an ASD and those who did not (Solomon, 1988, 1989a, 1989b). Many individuals who experience an ASD do not develop PTSD (Solomon, 1988, 1989a, 1989b), and early symptoms of intrusion or denial have been found not to predict the subsequent occurrence of PTSD (Shalev, 1992). Enduring distress and vulnerability to subsequent post-traumatic sequelae have been observed in individuals who experienced an ASD and, to a lesser extent, those who did not (e.g., Solomon, Garb et al., 1987; Solomon, Kotler et al., 1989). This distress, whether a ASD was experienced or not, tends to decline with time (Solomon, 1988, 1989a, 1989b). Cyclical or episodic PTSD has also been observed (Davidson et al., 1985; Green, Lindy et al., 1990; Horowtiz, 1986a) with an onset of stress or depression often triggering a reemergence
of PTSD (Davidson et al., 1985; Solomon, Garb et al., 1987).

Summary

Post-traumatic sequelae can vary in time course and the number and severity of symptoms. Generally, PTSR involve symptoms of reexperiencing aspects of the trauma, and symptoms indicative of both hyperreactivity / hyperarousal and psychosocial avoidance or numbing. Additionally, severe PTSR such as PTSD may be accompanied by a number of comorbid conditions. The reviewed observations suggest a possible relationship between the differing forms of PTSR discussed. Traumata may produce an acute stress response, a sub-threshold stress response (ST-PTSD), or an immediate case of PTSD depending upon a combination of individual and trauma characteristics. Any of these three reactions may resolve on their own or with therapy, or, in the case of ST-PTSD and PTSD, persist over time. An acute stress response may partially attenuate to ST-PTSD or escalate to PTSD. It is also feasible that PTSD may partially resolve to a ST-PTSD, which may then resolve completely or escalate to PTSD once again, giving rise to cyclic or episodic PTSD. Repeated traumata, particularly at a young age, may so overwhelm the individual's capacity to cope that pervasive character shifts and developmental issues may become associated features. This model, though untested, does provide a conceptual link between the varied PTSR and may have heuristic value.

Explanations of Post Traumatic Stress Reactions

The term PTSR has been used thus far to designate a broad class of psychological
and physiological syndromes that may develop in reaction to traumata. As mentioned, PTSR can be thought of as occurring along a continuum of severity ranging from an immediate and brief response to a severe, chronic condition. Chronic PTSD represents the severe end of the continuum. The present discussion will focus on PTSD per se.

Physiological Explanations and Correlates of PTSD

A number of researchers have forwarded physiological models of PTSD in recent years. Most of these reports present physiological correlates of PTSD or analogies between PTSD and laboratory preparations with non-human participants. Accordingly, causal relationships between neurophysiological alterations and the emergence of symptoms of PTSD cannot be firmly established. Some authors (e.g., Pitman et al., 1990), citing the paucity of significant findings with other anxiety disorders and the complexity of the neuroendocrine system, assert that finding reliable neurological and endocrinological correlates of PTSD may prove very difficult. The key to the biological basis of PTSD is thought to lie more in the central nervous system (CNS) than in the peripheral nervous system, making controlled investigations extremely difficult (Pitman et al., 1990). Nevertheless, investigations of neurophysiological processes that might underlie PTSD is not a futile venture. Such will likely compliment psychological models and can only further our understanding the nature and effective treatment of the disorder.

One of the earliest reports on the physiological changes with stress relevant to PTSD was made by Meakans and Wilson (1918, cited by Giller et al., 1990) just after World War I. They found evidence of sympathetic nervous system (SNS) hyperactivity
in war veterans as indicated by supersensitivity to war related stimuli and epinephrine injections. Later, Grinker and Spiegel (1945, cited by van der Kolk, Greenberg, Boyd, & Krystal, 1987) found that individuals exposed to extreme stress exhibited an array of symptoms consistent with catecholamine depletion, again suggesting SNS hyperactivity. Subsequent biological models of PTSD have focused largely on the etiology of this SNS hyperactivity.

**Inescapable Shock Model**

The utility of experimental preparations involving inescapable shock with non-human participants in modeling PTSD was first suggested by Krystal, 1978. Later, other authors tested and more fully delineated the model (e.g., van der Kolk et al., 1984; van der Kolk, Greenberg, Boyd, & Krystal, 1985). Non-humans, when exposed to unavoidable and inescapable aversive stimulation, are known to develop a variety of symptoms that appear analogous to symptoms of PTSD. These include (a) reduced capacity to acquire escape responses during subsequent exposure to escapable aversive stimulation, (b) reduced motivation to learn new appetitive contingencies, and (c) evidence of chronic subjective distress (van der Kolk et al., 1984). Other similarities include heightened contextual fear, heightened passive avoidance, and stress induced analgesia (SIA) (Foa, Zinbarg and Olasov-Rothbaum, 1992). These symptoms are sometimes referred to as experimental neurosis (EN) in this context (e.g., Foa et al., 1992).

Several biological response systems have received attention in explicating EN.
Catecholamine systems are known to become hyperactive, producing increased epinephrine and norepinephrine turnover, a depletion of CNS norepinephrine, and decreased CNS levels of dopamine and serotonin (van der Kolk et al., 1984, 1985, 1987). The effects of inescapable shock on the locus coeruleus (LC) is of particular interest. The LC is thought to be the primary source of noradrenergic activity in higher brain centers and to facilitate memory retrieval (van der Kolk et al., 1985). Environmental threats are known to increase LC firing and Van der Kolk and colleagues (van der Kolk, 1985) have proposed that a long-term potentiation of neural pathways of the LC by intense stimulation during trauma may occur. The result of this would be hyperreactivity to stimuli and possibly a priming of traumatic memories such that they may be readily stimulated by diverse events.

Inescapable shock has also been shown to produce SIA. Stress-induced analgesia is a phenomenon wherein, after exposure to extreme stress, subsequent exposure to stressors increases behavioral and subjective tolerance to painful stimulation (Maier, Davies, & Grau, 1980). Pitman and colleagues (1990), in a controlled study, found that combat-relevant stimuli produced analgesia in veterans with PTSD but not in veterans without PTSD. Further, naloxone, an opiate antagonist, was able to block the analgesic effects of such stimuli in veterans with PTSD. The reversibility of this effect in humans and non-human by naloxone, suggests this phenomenon is mediated by endogenous opiate release (e.g., Maier et al., 1980; Pitman, van der Kolk, Orr, and Greenberg, 1990). Additionally, opiates are known to reduce neural activity of the LC (van der Kolk, 1985).
The possible involvement of endogenous opiates in the effects of inescapable shock has lead to speculation that opiate release, contingent on exposure to trauma-related stimuli, may produce effects which reinforce the behaviors that lead to the re-exposure (e.g., Scrignar, 1988; Urback & Renfrey, 1988; van der Kolk et al., 1984, 1985, 1987). Some of these effects might include an anxiolytic action, a reduction in feelings of paranoia and inadequacy, and an antidepressant action (Verebey, Volavka, & Clouet, 1978). Accordingly, the reexperiencing symptoms and re-traumatization proclivities associated with PTSD may represent reinforced operants. Additionally, some symptoms of opiate withdrawal are equivalent to the hyperarousal features of PTSD (e.g., van der Kolk, 1984, 1985, 1987). This has lead to speculation that the SNS hyperactivity of PTSD is due to noradrenergic reactions to a decrease in relative endogenous opiate levels (van der Kolk et al., 1984, 1985, 1987). Accordingly, persons with PTSD might be thought of as being addicted to endogenous opiates. Van der Kolk and colleagues (van der Kolk, 1985) have asserted that the hyperreactivity in PTSD is due to a combination of LC pathway potentiation with an augmentation by opiate withdrawal.

Research using inescapable shock has provided information that may be relevant to the etiology and treatment of PTSD to the extent that EN is a good model for PTSD. Foa, Zinbarg and Olasov-Rothbaum (1992), have asserted that the key elements of the inescapable shock paradigm are the unpredictability and uncontrollability of the aversive stimulation, that without both of these features, participants are significantly less likely to develop an experimentally induced neurosis. Accordingly, the inescapability and
uncontrollability of traumatic events may be of critical etiological importance. Indeed, such has been suggested, as will be discussed in later sections. Also, loss of controllability over aversive stimulation has been shown to have a more detrimental effect than non-controllability per se. (Mineka & Kihlstrom, 1978), as has uncontrollable and unpredictable aversive stimulation in a previously "safe" setting (Masserman, 1943, cited by see Foa et al., 1992). Similarly, prior experience with uncontrollable and unpredictable aversive stimulations has been shown to sensitize study participants to subsequent aversive stimulation (see Foa et al., 1992; Krystal et al., 1989). These observations may have their counterparts in the development of PTSD in humans and there is evidence to suggest they do (see Foa et al., 1992 for discussion).

Regarding treatment implication of the model, to the extent that EN is analogous to PTSD in humans, research into the effects of pharmacological and behavioral manipulations on the development and attenuation of EN may be applicable to PTSD. A number of drugs have been found to decrease the effects of inescapable shock in non-human participants, including clonidine, benzodiazepines, tricyclic antidepressants, and monoamine oxidase (MAO) inhibitors (van der Kolk, 1984). Each has subsequently been found to provide some relief for symptoms of PTSD (e.g., Hogben & Cornfield, 1981; Kinzie & Leung, 1989; Kolb et al., 1984; van der Kolk, 1983). Another implication of the inescapable shock model for treatment is provided by the observation that non-humans with EN, if physically forced to escape escapable shock, will exhibit escape responses upon subsequent shock (van der Kolk, 19984). This suggests that treatments for humans with PTSD which encourage adaptive action in the face of trauma-related
stimuli, may be particularly effective.

Kindling Model

Another phenomenon that may have relevance to the etiology of PTSR is kindling (see Goddard, McIntyre, & Leech 1969). In experimental kindling, repeated sub-threshold chemical or electric stimulation of brain centers eventually come to elicit a generalized neural response from those centers. An example would be the repeated application of sub-seizure electrical stimulation to limbic structures that eventually results in generalized tonic-clonic seizures upon stimulation (Goddard et al., 1969). Post and Kopanda (1976) proposed that kindling may be a biological mechanism by which permanent and semi-permanent changes in neuronal substrata, particularly in the limbic system, cause major psychiatric disturbances.

Several authors (Friedman, 1988; Lipper, Davidson, Grady, Edinger, Hamnett, Mahorney, & Cavenar, 1986; Urback & Renfrey; van der Kolk, 1987; van der Kolk & Greenberg, 1987) have proposed that a kindling-like phenomena may underlie some symptoms of PTSR, specifically, autonomic hyperarousal and susceptibility to intrusive symptoms. According to this position, repeated exposure to "sub-threshold" stimuli or exposure to brief but highly intense stimulation, may bring about neuronal changes that effectively lower the threshold to reactivity, in this case, to autonomic arousal. Carbamazepine, an anti-convulsant, is known to attenuate the seizure inducing effects of kindling. The efficacy of Carbamazepine in reducing intrusive and autonomic hyperarousal symptoms in patients with PTSD (e.g., Lipper, 1990; Lipper et al., 1986;
Wolf, Alavi, & Mosnaim, 1988) lends support for the kindling model of PSTD.

**Emotional Biasing Model**

Similar to the kindling model, emotional biasing has been proposed as a basis for PTSD (Pitman, Orr, & Shalev, 1993). The model is based upon several observations: (a) anxiety proneness in cats is associated with enhanced neural transmissions between the basal amygdaloid nuclei (BAN) and ventromedial hypothalamus (VMH) (Adamec, 1991a, cited by Pitman et al., 1993); (b) electrical stimulation of the BAN-VMH pathway produces lasting tendencies toward defensive responding (Adamec, 1991a, cited by Pitman et al., 1993); (c) results similar to those in #2 above can be produced by the administration of an anxiogenic agent; and (d) the strength of the BAN-VMH transmissions appears to be positively associated with both constitutional and acquired fearfulness (Pitman et al., 1993). According to this model, the elicitation of strong feelings of anger or fear may bring about semi-permanent alterations in the BAN-VMH axis that predisposes the affected individual to increased fearfulness or anger. In extreme cases this could lead to outbursts of either. Some experimental evidence supports this model in non-human preparations (e.g., Britton et al., 1991), but the model is largely untested. Pitman and colleagues posit that their model differs from the kindling model in that emotional experiences cause emotional effects rather than electro-chemical stimulation causing convulsive effects. However, the kindling model previously discussed speaks of a kindling-like phenomenon, and emotional biasing may just be that phenomenon. Accordingly, this model may be synonymous with the kindling model.
Neuropsychological Sensitization Model

Another model for PTSD has been proposed by Kolb (1987, 1988). Based on research findings with both human and non-human participants, Kolb observed that neuronal systems, when exposed to excessive stimulation, can undergo a brief reduction in firing capacity, can be temporarily impaired for longer periods, or may suffer permanent damage (Kolb, 1987). He suggested that the intense neural stimulation produced in response to traumatic events, may cause structural changes in amygdaloid nuclei (structures within the limbic system usually associated with varied species-specific behaviors such as sexual and defensive behavior). Initially, these changes may bring about sensitization, but with continued or repeated excessive stimulation, these changes may retard habituation and discrimination. As a result, the individual would be prone to indiscriminate hypersensitivity to internal and external stimuli with a lowered capacity for habituation.

Noradrenergic Dysregulation Model

Krystal and colleagues (Krystal et al., 1989) proposed a noradrenergic dysregulation model for PTSD that focused on the locus coeruleus (LC) as the center of action. The LC appears to function in part as a key activation center in times of stress; it is a major source of ascending noradrenergic pathways in the brain and activates centers thought to be central to alarm discrimination, fear, and memory formation (Krystal et al., 1989). According to this model, traumatic events may condition an activation of the LC and other related neural pathways such that they become overly
sensitive to external stimuli, causing a frequent or constant alarm activation. It is as though the affected individual was on constant alert for danger and has a tendency to over react to stimuli. Accordingly, the model employs neurophysiological and behavioral mechanisms to explain PTSD.

Supporting this model are several observations. Elevated levels of epinephrine and norepinephrine have been found in the urine and blood streams of PTSD sufferers (Kosten, Mason, Giller, Ostroff, & Harkness, 1987; Mason, Giller, Kosten, Ostroff, & Podd, 1986; Svensson, 1987), suggesting a perpetual alarm response. These elevated levels appear to maintain themselves across time (Krystal et al., 1989). Additionally, a significant downregulation of peripheral adrenergic receptor sites in patients with PTSD has been observed, suggesting an adaptive response to elevated levels of catecholamines (Perry, Giller, & Southwick, 1987). Under severe stress, cortisol is released through activation of the hypothalamic-pituitary-adrenal (HPA) axis (HPA) (Krystal et al., 1989). Krystal and colleagues suggest a link between the dysregulation of LC activation and sympathetic activation of the HPA axis in PTSD, though the exact nature of this link is unknown (Krystal et al., 1989).

Superconditioning Model

It has been proposed that the strength of conditioned responses to trauma-related stimuli is particularly strong in PTSD because of the modulating effects of neurohormones/neuroregulators (Pitman, 1988, 1989, 1993). It has been observed that hormonal activity associated with acute stress can modulate the strength of a memory associated
with that stress (e.g., Delaney, Tussi, & Gold, 1983; Gold, 1988; McGaugh, 1985; Zager & Black, 1985), effectively enhancing consolidation and providing resistance to the extinction of accompanying conditioned responses (e.g., Pitman, 1989). Pitman (1993) refers to this as superconditioning. Accordingly to this model, the primacy of trauma related memories is a result of this memory enhancement, which in turn gives rise to the range of symptoms seen in PTSD. Relevant neuromodulators identified to date include adrenocorticotropic hormone, epinephrine, and norepinephrine (Pitman, 1989; McGaugh, 1985; Zager & Black, 1985). Also proposed is the possibility that, due the an inverted U-shaped dose response curve for the action of these neuromodulators on memory, excessive levels of such may interfere with memory consolidation, possibly resulting in amnesia for trauma-related events (Pitman, 1989). This model also addresses the phenomenon of delayed onset PTSD. Based on a hypothesis originated by Eysenck and Kelly (1987, cited by Pitman, 1989), Pitman suggests that, in cases of subclinical PTSD (i.e., a ST-PTSD as discussed above) recollections of the event may result in a re-release of neuromodulators, which in turn, consolidates the memory further. A positive feedback loop is thereby established which may eventually result in the emergence of PTSD. This is congruent with the kindling model discussed above.

**Pre-Hippocampal Memory**

Jacobs and Nadel (1985) proposed a model for stress induced recovery of phobic anxiety that may prove relevant to the etiology of PTSR. The model is based upon studies of infantile memory. According to their model, highly distressing events that
occur during infancy, prior to hippocampal development (before the age of 18 to 36 months), may result in a context independent conditioned aversive response. After hippocampal development, adult learning characteristics emerge (context specific) and the pre-hippocampal conditioned response becomes "unavailable" to the individual's repertoire. Under conditions of extreme environmental or hormonal stress, hippocampal functioning may be disrupted to an extent that pre-hippocampal conditioned responses re-emerge and may become permanently reinstated. Were this to occur, the affected person would experience a sudden post-stress onset of phobic reactions (related to the original trauma), aversive reactions to a wide array of current stress related stimuli, and/or a relatively enduring increase in autonomic hyperactivity. It has also been proposed that such emergent responses would prove resistant to traditional extinction or counter-conditioning treatments.

Although the model has been offered to explain clinical observations of spontaneous phobia emergence (those occurring in the absence of an identifiable conditioning trial), it might also help explain why some people seem more prone to developing chronic, more serious PTSR than others. The hypothesis being proposed here is that individuals who have been traumatized in infancy, prior to hippocampal development, may experience a reemergence of pervasive pre-hippocampal fears and autonomic hyperactivity that resist treatment. Such individuals would not have a memory of the earlier trauma and so epidemiological studies to date may not have accessed pre-verbal trauma as a pre-disposing factor. The model presents with some interesting features and may warrant closer investigation of its relevance to PTSR,
particularly its prediction that trauma during infancy is a potent pre-disposing factor. Such is merely speculative at this time and its only support, aside from intuitive appeal, is the observation that PTSD has an elevated comorbidity with phobias and other anxiety disorders (Davidson & Fairbank, 1993; Sim & Houghton, 1966; Weekes, 1978).

**Summary of Physiological Explanations**

Seven theories about the physiological basis of PTSD have been presented. Each enjoys indirect empirical support but none has been conclusively demonstrated to adequately model PTSD in humans. Adequacy here might best be thought of as explanatory and predictive utility, rather than as reflecting processes in PTSD on a point to point basis. Though these models differ in their focus, they are not necessarily incompatible with one another. For example, Pitman's (1989) notion of a feedback loop between trauma recollections and neuromodulator re-release is consistent with the kindling model of PTSD. Similarly, the long-term potentiation of LC pathways proposed by van der Kolk (1985) by intense, trauma-induced stimulation is consistent with Kolb's (1987) proposal that intense trauma-induced stimulation of amygdaloid nuclei produce enduring structural changes. In both cases, hyperreactivity to environmental stimuli is a proposed outcome. The fact that the LC is probably responsible for enervating the amygdaloid complex in times of threat suggests that both processes may be related.

It is quite possible that each theory or model reflects a piece of the complex of neurological and endocrinological processes that underlie PTSD. Only further basic and applied research will demonstrate the extent to which each is adequate or useful as such.
The physiological models presented may prove to delineate much of the physiological processes that underlie the development and maintenance of PTSD; however, they should not be considered superior to or otherwise inconsistent with psychological models. The two represent different levels of analysis which may better be thought of as complimentary rather than competing.

**Psychological Explanations of PTSD**

Psychological explanations for PTSD are many and varied. Explanatory schemes tend to conform to the principles and assumptions of psychology's major schools of thought yet within each camp lies a great deal of variation. Because of this, no exhaustive attempt will be made here to cover the field. Rather, the goal is a brief exploration of major psychological explanations.

**Psychodynamic Interpretations**

Classical psychodynamic approaches to understanding PTSD have been criticized for focusing excessively on constitutional factors and too little on the ubiquitous effects of trauma itself (e.g., Peterson, Prout, & Schwarz, 1991). According to these critics, by focusing on pre-traumatic intra-psychic conflicts and other constitutional factors to explain who does and does not develop a PTSD, a measure of "blame" is placed on the affected individual. This not only has important ramifications for survivor rights and compensation, but may negatively impact recovery through negative self-attributions (Friedman, Bischoff, Davis, & Person, 1982; Janoff-Bulman, 1979; Scrignar, 1988).
Herman (1992), in her analysis of the social context of psychological thought regarding PTSD, asserts that until recent times the true potential impact of trauma on the individual was denied at a cultural level. It would appear that classical psychodynamic thinking significantly contributed to this neglect which has resulted in too little attention to the rights and recovery of trauma survivors. Nevertheless, psychodynamic conceptualizations and treatment approaches have contributed significantly to the field of PTSD.

The first psychodynamic formulation of PTSD, the referred to as traumatic hysteria, was put forth by Breuer and Freud (1893, cited by Peebles, 1989a) and was cognitive in nature. According to this model, traumatic sequelae consisted of a memory disturbance, an inability to forget a traumatic experience due to (a) being unable to fully react at a physical level at the time of the trauma thereby preventing a discharge of the emotion, and (b) a disruption of a "normal wearing away process" (Breuer & Freud, 1893, p. 11, as cited by Peebles, 1989a) wherein freely thinking about the event reduces the energy or potency of the memory. This disruption was presumed to be a function of psychological defending against the highly energized state of the traumatic memory (due to the lack of physical discharge at the time of the trauma). Intrusive symptoms were thought to be the result of a lapse in ego defenses and the brief emergence of the memory. Freud speculated that the tendency for traumatized persons to reexperience the trauma represented a natural process toward event mastery. This position has seen a resurgence in recent years, but with a focus on information overload rather than excess memory potential (Horowitz, 1974, 1983, 1986a, 1986b), as will be discussed later.

Psychodynamic thinking, under the influence of developments such as ego
psychology, later focused more on pre-existing neurotic conflicts as a context within which post-traumatic issues were expressed (Peterson, Prout, & Schwarz, 1990). The role of trauma in overwhelming ego defenses and disrupting ego functioning (Peebles, 1989a, 1989b) was an outcome of this, as was the notion that pre-existing neurotic conflicts were the prime precursor for PTSR. A sense of helplessness has been identified as a core feature of traumatic experiences (e.g., Herman, 1992; Horowitz, 1986a, 1986b; Peterson & Seligman, 1983) which, by its very nature, compromises ego mastery. Some authors (e.g., Hendin et al., 1981; Peterson et al., 1990) assert that the effects of trauma depend upon the meaning that the trauma to the individual and the adaptive or maladaptive mechanisms used to cope with it, both of which depend upon pre-traumatic life experiences and unresolved conflicts. It has been suggested that the meaning of a trauma is determined by three factors: (1) perceptions of realistic dangers, (2) subjective distortions of such dangers, and (3) threats to ego defenses against such dangers (Hendin et al., 1981).

The immediate consequence of trauma is the emergence of primary needs as preeminent. This in turn is presumed to cause an enduring weakening of ego structure, excess use of denial as a defence, constant alertness to retraumatization, and shattered basic trust (Grubrich-Simitis, 1981 as cited by Peterson, Prout, & Schwarz, 1990). Some authors have advanced that, in some cases, self and ego structures may not be capable of recovering from traumatic degradation of ego structures and so PTSR may be permanent (e.g., Rappaport, 1968).

In recent years, Mardi Horowitz has proposed a model for the etiology of PTSR
that is similar to Breuer and Freud's original formulation (1893, as cited by Peebles, 1989a) but which emphasizes the import of information overload (Horowitz, 1974, 1983, 1986a, 1986b). Though some (e.g., Peterson et al., 1990) classify Horowitz' model as an information processing / cognitive model, Horowitz writes from a psychoanalytic base and sees his model as a natural extension of that perspective (Horowitz, 1974, 1983, 1986a, 1986b; Horowitz & Kaltreider, 1979). Accordingly, it will be discussed here as a psychodynamic model, though the overlap with cognitive psychology is acknowledged.

The basis of Horowitz model is the concept that, through life experiences, people construct cognitive schemata of themselves, the world, and the relationship between the two. These schemata or internal representations are used to interpret subsequent experiences, to make sense of them. As new experiences accrue and the information is integrated with prior experiences, these schemata may change gradually. Events which cannot be reconciled with existing schemata are held in memory in a potentiated state so that they can be processed and integrated into the inner models. Horowitz called this the completion tendency. Such memories tend to intrude into consciousness as a means of facilitating active processing until they are fully integrated. If there is as large incongruence between the memory contents and inner schemata, the schemata may be altered to accommodate them.

Horowitz has proposed that traumatic experiences, by their very nature, cannot be reconciled with an individual's existing schemata. The memories are therefore held in a potentiated state and this results in many of the symptoms observed in PTSR. He
posits that the intense emotional effects that such intrusions may have can bring about inhibitory controls (ego defenses) and the relative balance of intrusion and inhibition determines the nature of experienced symptoms. For example, if inhibitory controls predominate, a general emotional numbing will occur; if intrusions predominate, prolonged autonomic arousal will occur. An optimal state is one in which intrusions and inhibitions alternate, allowing for processing to occur with periods of respite through numbing. Intrusions bring about the reexperiencing and hyperarousal symptom clusters, while inhibitory processes bring about the psycho-social numbing symptom cluster.

Horowitz' model has probably been the most influential in the area of PTSR, forming basis of the DSM-III-R (APA, 1987) diagnostic criteria (Peterson et al., 1990). Though it is compelling and bridges the psychodynamic and cognitive schools of thought, it is not without its detractors. For example, Russell (cited in Herman, 1992) has proposed a similar model but asserts that emotion is the primary drive behind reexperiencing phenomena. According to this concept, intrusive symptoms produce affective responses that are needed to complete the healing process. Clinicians of a more classically psychodynamic orientation may find Horowitz' model to be too cognitive, as might some behaviorally oriented clinicians, but the model is yet to be matched in its completeness and parsimony.

There are other psychodynamic schools which have produced interpretations of PTSR which have not been discussed. To include all schools of thought is beyond the intended scope of the present paper. What has been presented is a brief outline of some of the principle concepts and assumptions of this broad field regarding the effects of
trauma. Though the school has been criticized for its approach (see Friedman, Bischoff, Davis, & Person, 1982; Janoff-Bulman, 1979; Scrignar, 1988), Psychodynamic formulations, if employed with clear acknowledgement and appreciation for the central role of trauma in the development of PTSR, can aid our understanding of who develops PTSR, how it is expressed, and pre-traumatic issues that warrant attention (Hendin et al., 1981; Horowitz, 1986a, 1986b).

Cognitive & Cognitive-Behavioral Interpretations

Foa, Steketee, and Olasov-Rothbaum (1989) have asserted that cognitive processes have a central role in the etiology and maintenance of post-traumatic sequelae and that information processing theories are necessary to explain reexperiencing phenomena such as flashbacks, nightmares, and intrusive thoughts. Other authors have echoed this position (e.g., Hendin, 1984; Hendin, Hass, & Singer, 1983; Horowitz, 1983, 1986a, 1986b; Kilpatrick et al., 1989; van der Kolk, 1987a). Similarly, Brett and Oстроff (1985) have identified post-traumatic imagery as an overlooked but central feature of PTSD.

Cognitive and cognitive-behavioral theorists generally emphasize the central import of cognitions and cognitive processes to normal and abnormal functioning (see Beck, 1972. For our purposes, cognitions refer to thoughts, beliefs, attitudes, and images, while cognitive processes refer to memory recollection and the assimilation and accommodation of new experiences by existing representations of the self and world (schemata). As with Horowitz' model, cognitive formulations often emphasize the role
of processing or making sense of a traumatic event (e.g., Veronen & Kilpatrick, 1983). Supporting this is the finding by Wong and Weiner (1979) that the more negative and unexpected an event, very characteristic of traumata, the greater the tendency to formulate causal explanations.

Cognitive formulations often make use of expectancy theory, attribution theory, and cognitive appraisal (Veronen and Kilpatrick, 1983). For example, an individual's reaction to trauma may be affected by (a) pre-trauma expectations about the likelihood of being traumatized and the subsequent congruence of the experience with those expectations; (b) the cause attributed to the event, particularly whether self- or circumstantial-blame is leveled; and (c) post-trauma appraisal of the self, others, and the nature of world (Foa, Steketee, & Olasov-Rothbaum, 1989).

Foa and Kozak (1986) proposed a model of PTSR formation that is characteristic of cognitive formulations of PTSR in general. Their model focuses upon both cognition and emotion, positing that both are required for the formation of PTSD, that the association of trauma-related cognitions with fear is necessary to produce clinical manifestations. According to this model, without the affective element, potentially traumatic events will not produce cognitions and cognitive changes with the aversive quality associated with PTSR. They suggest that PTSD is a product of a fear structure, a program to escape danger that was constructed subsequent to trauma. In order to be effective in enabling an individual to avoid or escape future threats, the fear structure must contain both cognitive information about the nature and extent of the threat, and an emotive component that functions to physiologically prepare the individual for
escape.

In a later paper, Foa and colleagues (Foa et al., 1989) go on to proposed that PTSD differs from other anxiety disorders in that (a) the size of the fear structure in PTSD is larger due to a generalized fear response; (b) the intensity of responses is greater due to the extreme threat value of traumatizing events; and (c) the accessibility of the fear structure is greater because its has a lower threshold for activation. These differences are largely accounted for in terms of the threat value of the trauma and its unpredictability and uncontrollability. Regarding that latter, they posit that traumatic events, because of their intensity, unpredictability, and uncontrollability, cause a multitude of stimuli and responses to become associated with threat or danger. These authors also couch this in terms of many previous stimuli losing their safety-signal value (see Seligman & Binik, 1977, cited in Foa et al., 1989).

Foa and colleagues (Foa et al., 1989) also proposed that differences in fear structures differentiate between those who will develop chronic PTSD, and those who will not. Fear structures with greater size, due to greater unpredictability and greater loss of safety-signals, and greater intensity, due to level of perceived threat at the time of the trauma, will likely produce more intense physiological/affective reactions and more frequent escape/avoidance responses in people than lesser fear structures.

Similar to Horowitz (1983, 1986a, 1986b) and Foa and K. zak (1986), Epstein (1990, cited by Peterson et al., 1990) and Janoff-Bulman (1985) have both proffered cognitive models of PTSR but both have focused more on three similar assumptions and beliefs which are altered by traumatic events. Both presume that people construct
personal theories of reality through their life experiences with new information being integrated through both assimilation (fitting the new information within the existing theory) and accommodation (altering the theory to allow new information to fit) (see Epstein, 1973; Parkes, 1971, 1975; Peterson et al., 1990). Both propose that people hold three core assumptions or beliefs about the nature of the world and one's place within it that are essential to healthful functioning: (1) the world is benevolent and one is invulnerable to harm; (2) the world is meaningful, comprehensible, and events are within one's control; and (3) the self is perceived as positive. Traumatic events often cause these core beliefs to shift toward an assumption of malevolence and vulnerability, meaninglessness and uncontrollability, and personal unworthiness. Both authors see PTSD as reflecting a maladaptive response to the challenge of these beliefs, but also assume that traumatic events can alter these beliefs in ways that promote reality based adaptation to life aversive events.

Peterson & Seligman (1983) proposed that learned helplessness model of experimental psychology (Maier & Seligman, 1976) may be applicable to PTSR. According to this model, the uncontrollability of the onset and termination of many traumatic experiences is their core element, and this can lead to a variety of symptoms similar to those seen in non-human subjects exposed to uncontrollable aversive stimulation (see Foa et al., 1989, 1992; Mineka & Kihlstrom, 1978; Peterson & Seligman, 1983). These include passivity, retarded capacity to acquire escape / avoidance behaviors, affective non-responsivity (numbness), outbursts of aggression, general agitation, and withdrawal. Peterson and Seligman (1983) propose that, because
of the uncontrollability of the event, traumatized individuals formulate beliefs that they are unable to affect control over a generalized class of aversive events, and this belief in return prevents adaptive functioning and causes the emergence of the psycho-social numbing symptoms of PTSD. This model is supported by the finding that perceived controllability over aversive events in traumatized persons is inversely related to PTSD symptom severity (Kushner et al., 1992).

Abramson, Seligman, and Teasdale, (1978) proposed that the nature and effect of the acquired dysfunctional belief is dependant upon three dimensions: (1) the attributed cause of the trauma (self vs. other); (2) the perceived likelihood of recurrence across time (stable vs. unstable); and (3) the likelihood of recurrence across situations (global vs. situational). According to this model, the more a traumatic event is attributed to the self and is perceived as stable and global, the greater the sense of helplessness and hence the greater the sequelae.

The learned helplessness model of PTSD is compelling in some ways, particularly its basis in experimental findings, but it does not address the intrusive and hyperarousal symptom classes. Additionally, some forms of self-blame (behavior vs. character) have been associated with a better prognosis (Janoff-Bulman, 1979), though these might be construed as enhancing a sense of control over future traumata by identifying behaviors that can be changed or avoided to prevent a recurrence (Janoff-Bulman, 1979; Foa et al., 1989. Otherwise, the cognitive processes proposed are consistent with other cognitive formulations. Additionally, this application of experimental findings to the etiology of PTSR may be an important step to identifying

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Behavioral Interpretations

To date, the predominant behavioral interpretation of the acquisition of post-traumatic sequelae has been based on Mowrer's (1960) two-factor conditioning model (e.g., Kilpatrick, Veronen, & Best, 1985) wherein both classical (e.g., Pavlov, 1960) and operant (e.g., Skinner, 1953) conditioning principles are drawn upon. According to this model, a victim's natural response to a traumatic event includes high levels of fear and anxiety. Stimuli present during the event, by virtue of being paired with unconditioned and conditioned stimuli associated with the event itself, may become conditioned stimuli capable of eliciting conditioned responses similar to those elicited during the original trauma. Through stimulus generalization and higher-order conditioning, the victim comes to experience fear and anxiety in response to an increasingly wide array of previously innocuous stimulus events. Operant responses that terminate or prevent such fear responses are negatively reinforced and eventually become a pervasive part of the individual's repertoire. Such escape and avoidance behaviors are assumed to prevent the necessary exposure to the conditioned stimuli to effect extinction. Additionally, Keane, Zimering, and Caddell (1985) have proposed that state dependant retention of traumatic memories prevents complete exposure under all but extremely distressing conditions.

Although this Mowrerian model has provided a primary theoretical underpinning of behavioral conceptions and treatments of trauma related fear, anxiety, and avoidance...
responses, it has been criticized by some as being inadequate to account for all post-traumatic sequelae. Accordingly, it may be questionable as a sole guide to treatment rationale. For example, Foa, Steketee, and Olasov-Rothbaum (1989) have asserted that cognitive processes have a central role in the etiology and maintenance of post-traumatic sequelae and that information processing theories are necessary to explain reexperiencing phenomena such as flashbacks, nightmares, and intrusive thoughts. Other authors have echoed this position (e.g., Hendin, 1984; Hendin et al., 1983; Horowitz, 1983, 1986a, 1986b; Kilpatrick et al., 1989; Peterson et al., 1990; Pitman et al., 1993; van der Kolk, 1987a). On the other hand, Urbach and Renfrey (1989), in an analysis of PTSD from a radical behavioral perspective, argued that a strict behavioral analysis can account for reexperiencing phenomena. They went on, however, to suggest that a functional analysis of PTSD based solely upon operant and respondent conditioning may be simplistic. Nevertheless, this simple behavioral model has had a significant impact on exposure based treatment rationales.

Psychosocial Interpretation

Green, Wilson, and Lindy (1985) have advanced a psychosocial model of PTSD which incorporates a number of the conceptualizations discussed thus far. This model speaks to the effects of event characteristics, individual characteristics, and the nature of the recovery environment on post-traumatic cognitive processing and hence on the rate and nature of adaptation. Regarding event characteristics, the model proposes that critical features include severity, duration, sudden onset, lack of preparation, degree of
death threat or bereavement, exposure to atrocities or grotesque deaths, degree of active participation in the event, and the degree of moral conflict. Put simply, the greater the number of these features experienced, the greater the degree of cognitive processing required and the greater the probability of developing dysfunctional sequelae (Green et al., 1985).

Individual characteristics felt to be important include degree of ego strength, efficacy of coping responses and defenses, degree of pre-trauma pathology, prior stressful/traumatic events, and the individual's psychosocial stage of development at the time of the event (Green et al., 1985; Peterson et al., 1990). The greater the ego strength and efficacy of coping mechanisms, the less impact the event will likely have. The greater the degree of pre-trauma pathology and the more numerous and severe any prior traumata, the poorer the prognosis. The individual's psychosocial stage of development is predicted to be a determinant of a number of post-trauma psychosocial conflicts (Erickson, 1968; Wilson, 1977, 1978). The model also posits that a recovery environment which has strong interpersonal / familial / societal supports and minimal social upheaval disruption will promote a more rapid post-trauma adaptation. Additionally, cultural features are acknowledged to have a significant impact on the development of post-traumatic sequelae (Lindy, Grace and Green, 1981). These three factors are thought to combine their effects on the rate of cognitive processing of the event, and whether adaptation will be healthful or pathological (Peterson et al., 1990). Horowitz' model of cognitive processing is used within this model to explicate the nature of the cognitive processing involved (Green et al., 1985).
Other Interpretations

A variety of other theoretical orientations have been used to explain the effects of traumatization. These include a psychosocial-developmental perspective (see Peterson et al, 1990; Wilson, 1977, 1978). According to this model, traumatic events may retard the resolution of which ever developmental challenge the affected individual is working through. Based upon Erickson's (1968) model of psycho-social development, it asserts that a failure to resolve a developmental conflict could result in a pervasive disruption in personal and interpersonal development that manifests in some features observed in PTSD. The nature of the manifestations will depend upon the stage of development affected, but, according to this model, the earlier the stage affected (i.e., the younger the individual) the greater the overall disruption. This model has particular relevance to the etiology of complicated PTSD (Brown & Fromm, 1986; Herman, 1992, 1993).

Several obscure theories and hypotheses have been put forth over the years that may offer useful explanations for some posttraumatic symptoms. Among these is Solomon's opponent-process theory of acquired motivation (Solomon, 1980; Solomon & Corbit, 1973). According to this theory, stimuli that elicit affective responses may, upon repeated presentation, come to elicit an affective response that is hedonically opposite to that originally produced. This is presumably due to the effects of two innate opposing processes. Thus a stimulus that originally produces an aversive affective response may come to produce a pleasant affective response and can thereby reinforce behaviors that produce it. According to Urbach and Renfrey (1989), such a process could explain the occurrence of reexperiencing phenomena. According to their
behaviorally oriented analysis, stimuli embedded within dreams or recollections of a traumatic event may come to reinforce the verbal and perceptual operants involved in such recollections. Accordingly, reexperiencing phenomena associated with PTSR may represent reinforced operants. This is not inconsistent with van der Kolk's "stress-induced analgesia" hypothesis (van der Kolk et al., 1985).

Two hypotheses were forwarded by Urback and Renfrey (1989) to explain the reexperiencing phenomena of PTSD: Such phenomena might reflect (a) the workings of an innate extinction process, or (b) a form of covert behavioral rehearsal. According to the former, the survival of an organism might be compromised by adventitiously acquired conditioned avoidance responses. Reexperiencing phenomena may be a product of an unlearned self-extinction process that involves both operant and respondent behaviors. According to the latter, through surviving traumatic experiences, an organism likely acquires new escape responses or existing repertoires might come under novel stimulus control. These operants are necessarily limited, both in terms of controlling stimuli and response permutations. Episodes of reexperiencing the event, however, might reflect a phylogenically based function that acts to broaden the class of escape responses, thereby enhancing the survivability of the organism. These proposals are not inconsistent with the psychodynamic concept of "repetition compulsion" or Horowitz' (Horowitz, 1983, 1986a, 1986b) "information processing" theory, and probably reflect differences more in the theoretical languages used than in substance.
Summary of Psychological Interpretations

Conceptualizations regarding the nature and etiology of PTSD are apparently wide and varied. The differences, however, may be more of language and focus than of substance. Overlap between the orientations was obvious in the present review. Horowitz' completion tendency seems analogous to Breuer and Freud's (1893, cited by Peebles, 1989a) repetition compulsion and his model in general appears to be a blend of psychoanalytic and information processing (cognitive) fields. The cognitive models discussed are consistent with one another and, though they tend to place greater emphasis on cognitions rather than emotions, are similar to psychodynamic models. All such models discussed assert that pre-traumatic characteristics of the individual affect the impact of trauma. Attention to the effects of social supports and environmental factors is acknowledged by most models, but was brought in formally in the psychosocial model presented. The behavioral model of PTSD is compelling in its austerity. Though some have criticized it for being incomplete (e.g., Hendrin, 1984; Horowitz, 1983, 1986a, 1986b; Kilpatrickik et al., 1989; Peterson et al., 1990; Pitman et al., 1993; van der Kolk, 1987a), Urbach and Renfrey (1989), employing an analysis of verbal behavior, argued that the model can be extended to account for all symptoms of PTSD. When this is done, however, the model becomes quite consistent with and similar to the other models discussed.

Regardless of the similarities and differences in the various conceptualizations of PTSD, it might be argued that of greater import is the effect these have on treatment and prevention.
The Treatment of Post Traumatic Stress Reactions

A variety of treatment approaches, derived from diverse theoretical orientations, have been used with PTSD. The following discussion considers some of these treatments, both pharmacological and psychological.

Pharmacological Treatments

The pharmacological treatment of PTSD is crude at present. No magic bullet exists and at best, pharmacological treatments for PTSD might best be thought of as temporary measures with two primary applications (1) the temporary amelioration of acute post-trauma distress, and (2) the amelioration of debilitating symptoms as an adjunct to psychotherapy. Because of the comorbid associations of other disorders with PTSD, medications are considered helpful in treating these, either by themselves or in conjunction with PTSD symptoms (Friedman, 1990). However, the issue of comorbidity and its treatment complexities is beyond the intended scope of this review. Accordingly, the following discussion with focus on specific pharmacological treatments for PTSD symptoms. Unfortunately, the presence of comorbid symptoms and their responses to medication often confounds findings in drug outcome studies. Well controlled studies in this area remain few.

A number of drugs have been found to decrease the effects of inescapable shock in non-human participants van der Kolk, 1984). These include clonidine, propranolol, benzodiazepines, and tricyclic antidepressants, each of which are also known to decrease central noradrenergic activity (see Krystal et al., 1989) and monoamine oxidase (MAO)
inhibitors (Hogben & Cornfield, 1981). This suggests, to the extent that the inescapable shock model is a good one for PTSD, that such agents might reduce PTSD symptoms and growing evidence supports this. Each of the above drugs do appear to decrease acute and chronic stress responses in PTSD (Kolb et al., 1984; Kolb & Mutalipassi, 1982; Krystal et al., 1989; van der Kolk, 1983). Interestingly, a variety of other substances commonly abused by PTSD sufferers, such as ethanol, barbiturates, and opiates, also suppress CNS noradrenergic activity (Krystal et al., 1989). This suggests that the abuse of such substances in persons with PTSD may reflect a pattern of self-medication of symptoms.

**Tricyclic Antidepressants**

A variety of tricyclic antidepressants have been found to provide relief for symptoms of PTSD (e.g., Blake, 1986; Boehnlein, Kinzie, Ben, & Fleck, 1985; Burstein, 1982, 1984; Embry & Callahan, 1988, Marshall, 1975; Yost, 1980). For example, imipramine, a tricyclic antidepressant, has been found to reduce intrusive recollections, flashbacks, and dream disturbance in patients with PTSD (e.g., Burstein, 1982, 1984; Marshall, 1975). Similarly, amitriptyline and doxepin have both been found to significantly reduce intrusive symptoms of PTSD (Bleich, Siegel, Garb, & Lerer, 1986). According to at least one report, the action of tricyclics appears to be specific to reducing intrusive and hyperarousal symptoms of PTSD, as comorbid depression and anxiety were not affected (see Friedman, 1990). However, other studies have found tricyclics to effectively ameliorate depressive symptoms accompanying PTSD, but to have no impact.
on intrusive and hyperarousal symptoms (see Friedman, 1990). In general, this class of drugs is considered effective in treating the intrusive and hyperarousal symptoms of PTSD along with symptoms of depression if present (Friedman, 1990; Scrignar, 1988). Avoidant symptoms do not appear to be affected.

**Monoamine Oxidase Inhibitors**

Hogben and Cornfield (1981) reported the successful use of phenelzine, a MAO inhibitor, in reducing nightmares, flashbacks, and violent outbursts, which facilitated the psychotherapeutic process. Other authors have found similar results (e.g., Levenson, Lanman, & Rankin, 1982; Shen & Park, 1983; Walker, 1982), yet others have found MAO inhibitors to be a little value (Shestatzky, Greenberg, & Lerer, 1988).

**Benzodiazepines**

Benzodiazepines have been found to be effective in treating excessive anxiety sometimes associated with a acute PTSR or subsequent crises during the course of PTSD (e.g., Friedman, 1988; Peterson et al., 1990; van der Kolk, 1983; Yost, 1980). For example, Bleich, Siegel, Garb, and Lerer (1986) found chlordiazepoxide, a benzodiazepine anxiolytic, to be effective in reducing intrusive symptoms of PTSD. It is generally believed, however, that due to the addictive properties of benzodiazepines, their use should be judicious and short term (Friedman, 1990; Peterson et al., 1990; Scrignar, 1988).
Neuroleptics

Major tranquilizers have been shown to have some positive effects of some symptoms of PTSD, but their use is generally not indicated. For example, Bleich, Siegel, Garb, and Lerner (1986) found thioridazine, a piperidine major tranquilizer, to be effective in reducing intrusive symptoms of PTSD. However, these authors concluded that the effects of thioridazine were due to its potent sedating effect and not to any specific action on these symptoms. Yost (1980) suggests that major tranquilizers should not be used routinely with PTSD for treating psychotic-like symptoms which are sometimes seen in acute exacerbations of PTSD. He asserts that these can normally be effected with tricyclics or benzodiazepines, both of which have fewer side effects and complications. Friedman (1990) also urges caution in prescribing neuroleptics, but asserts that they may be indicated when post-traumatic patients present with marked paranoid, aggressive, or self-destructive behaviors.

Other Pharmacological Agents

Carbamazepine is an anticonvulsant which has shown some promise as an adjunctive treatment for PTSD. Several studies have demonstrated carbamazepine's effectiveness in reducing intrusive symptoms and irritability (Lipper, 1990; Lipper et al, 1986; Wolf et al., 1988). This is of particular interest in light of the kindling model of PTSD previously discussed. Though the results of these studies are not conclusive and are confounded by possible hidden epileptic conditions, they do lend support to the kindling hypothesis (Friedman, 1990). Untested is carbamazepine's prophylactic
Clonidine, an alpha-type-2 adrenergic agonist, has been shown to reduce intrusive recollections, explosive outbursts of anger, and nightmares in cases of chronic PTSD (Kolb et al., 1984). Clonidine is known to reduce adrenergic activity in the locus coeruleus (e.g., Kinzie & Leung, 1989; Kolb et al., 1984), lending support to several models of PTSD which identify the LC as a brain structure central to both intrusive and hyperreactive symptoms. Additionally, clonidine has been used to treat narcotic withdrawal and is thought to exert such action through enhancing the effects of endogenous opiate and/or reducing activity of the noradrenergic system (Davis, 1985). This is consistent with van der Kolk and colleagues hypothesis that PTSD involves an addiction to endogenous opiates (van der Kolk et al., 1985).

Lithium carbonate seems to have similar effects on PTSD as the other drugs discussed thus far. It has been shown to reduce both intrusive and hyperreactive symptoms of PTSD (Kitchener & Greenstein, 1985; van der Kolk, 1983, 1987b). Lithium carbonate is thought to effect the serotonergic system of the CNS though its key function in the treatment of affective disorders is unknown. The serotonergic system has not been a central focus of physiological modelling for PTSD. If its effect on the serotonergic system is responsible for symptom reduction in PTSD, this could enhance our physiological models of the condition.

Propranolol, a beta-blocker often used for its anxiolytic effects, has been shown to reduce intrusive recollections, explosive outbursts of anger, and nightmares in cases of chronic PTSD (e.g., Famularo, Kinscherff, & Fenton, 1988; Kolb et al., 1984).
Friedman, (1990) suggests that propranolol's low-abuse potential makes it a good choice with substance abusing individuals with PTSD when an anxiolytic is called for.

Summary of Pharmacological Treatments

A number of pharmacological agents appear to have a palliative effect on some symptoms of PTSD, particularly the intrusive symptoms and hyperarousal. A number of authors, upon reviewing the literature, have asserted that pharmacotherapy for PTSD is insufficient by itself, that some form of psychotherapy is required as well (e.g., Friedman, 1988; Peterson et al., 1990). It would appear to have its place, however, in relieving excessive agitation or distress during acute stress reactions, during subsequent crises, and during any period that an individual's capacity to cope with the symptoms of PTSD or comorbid conditions is overtaxed (Peterson et al., 1990). Such use may prevent further deterioration and enhance the therapeutic process (Bleich, Siegel, Garb, and Lerer, 1986; Friedman, 1988). An overuse of medications, however, might be expected to retard psychotherapy through reduced motivation and access to the emotional content of traumatic memories, especially during a numbing phase of PTSD.

Psychological Treatments

The treatment of post traumatic sequelae has been the focus of a great deal of clinical and research activity in recent years. In the process, a wide range of interventions have been employed with varying degrees of success, both by themselves and as components of treatment packages. In the following survey, treatment approaches are
categorized according to broad theoretical schools. In should be kept in mind, however, that differences between these schools is often not as significant as they seem, there is overlap between these therapeutic approaches in both content and process. Additionally, it would seem that no treatment approach has emerged as a widely accepted treatment of choice.

**Psychodynamic Approaches**

The goal of psychodynamic therapies in the treatment of PTSD was originally to release the energy contained within traumatic memories through abreaction and/or catharsis (e.g., Peebles, 1989a, 1989b). Hypnosis (e.g., Peebles, 1989b; Scrignar, 1988) and narcosynthesis (e.g., Alexander & French, 1946; Kolb & Mutalipassi, 1982) were and continue to be used sometimes to gain rapid access to suppressed and repressed memories. Later, with greater emphasis on pre-traumatic neurotic conflicts, treatments tended to focus more on helping clients gain insight into the way in which their traumatic issues were primarily reenactments of past conflicts.

A more neo-dynamic approach calls for (a) temporary supportive treatment with judicious use of sedating or anti-depressant medications for immediate stress reactions, (b) brief abreactive, cognitive, and ego mastery work with cases of simple PTSD, possibly adjuncted with modification of prior conflicts, developmental conflicts, and maladaptive ego defenses if appropriate; and (c) long-term work focused upon rebuilding self and ego structures for cases of severe or complex PTSD (Horowitz, 1974, 1986a, 1986b; Peebles, 1989a, 1989b). The core of the therapeutic work, according to
Horowitz (1986a, 1986b) is a reappraisal of the traumatic event and meanings associated with it, and the revision of inner models of the self and world. This is congruent with the early psychoanalytic notion of discharging potentiated memories (Breuer and Freud, 1893, as cited by Peebles, 1989a). Such work can also be conceptualized as involving ego defense rebuilding and restructuring, and together is referred to as ego mastery (Peebles, 1989a, 1989b). In non-analytic terms, ego mastery refers to the client regaining a sense of control over situations or events which became disequilibrated when helplessness was intensely experienced. Horowitz' model suggests differing treatment strategies, depending upon whether the client is in an intrusive versus denial phase (Horowitz, 1986a). For example, in the denial phase, strategies might include narcosynthesis, defense interpretation, facilitating abreaction, encouraging exposure to trauma relevant stimuli (e.g., detailed recall, role playing, imagery, and environmental props). In the intrusive phase, strategies might include sedating medication, providing structure and organization, stress reduction through relaxation and demand reduction, behavior modification, and the avoidance of trauma relevant stimuli (Horowitz, 1973, 1974). The purpose of this flexible approach is to facilitate the processing of the trauma without overwhelming the client's defenses.

The client's processing or character style also has a presumed impact on optimal treatment focus (see Horowitz, 1974, 1986a; Peebles, 1989a, 1989b; Peterson et al., 1990). Horowitz suggests that if a client is obsessive about the event, moving rapidly from memory fragment to memory fragment without drawing connections between them, therapy should focus tightly on specific themes thereby forcing those connections
that will facilitate processing. If the client is hysterical about the event, focusing primarily on the emotional contents of the traumatic memory, therapy should focus on a systematic, cognitive evaluation of the event. If, on the other hand, the client intellectualizes excessively about the event, therapy should focus on the unexpressed emotional components of the memory. Here, we see how clients' pre-trauma personality dynamics presumably affect their response patterns to trauma and hence the optimal treatment focus.

In psychodynamic thought, the therapeutic relationship is assumed primary in the treatment of psychopathology (e.g., Alexander & French, 1946). This has been specifically echoed for the treatment of PTSR (Haley, 1974; Horowitz, 1986a, 1986b; Parson, 1984; Peebles, 1989a, 1989b). Put simply, the relationship is the context within which therapy takes place.

The efficacy of psychodynamic treatments for PTSR is controversial (see Scrignar, 1988). Numerous case reports suggest they are effective, but it has also been observed that psychoanalytic therapists have questionable success in treating severely traumatized persons (e.g., Peterson et al. 1990). Additionally, the absence of well controlled outcome studies of the effectiveness of psychodynamic treatments reduces confidence in them among more research oriented clinicians.

**Cognitive Therapy**

A number of cognitive interventions have been proposed and tested for the treatment of PTSR. Most seem to combine elements of a psychodynamic approach to
treatment (e.g., verbally working through the trauma) with elements of exposure-based behavioral or action-centered interventions (see below). Accordingly, they might be thought of as hybrids of dynamic and behavioral therapies. What seems to differ with cognitive therapies from these and others may lie more in the assumed underlying processes and the language of description than in anything of substance. Several authors have proposed complex cognitive approaches that are quite elaborate and self-contained. Other cognitive strategies for dealing with PTSD might best be considered techniques to be used within the context of an overall treatment program. Examples of both will be presented here.

Complex Approaches. Foa and Kozak (1986) suggested that to eliminate a post-traumatic fear structure, the fear memory must be activated and new cognitive-affective information must be injected into it which is incongruent with existing elements that are dysfunctional. They also assert that the size and complexity of the fear structure pose special problems for the treatment of PTSD. Drawing from the work of Lang (1977), Foa and colleagues propose that PTSD may be resistant to treatment because the complexity and incohesiveness of post-traumatic memories make it difficult to activate the entire fear structure or even the critical amount of it necessary for reprocessing (Foa et al., 1989). Additionally, the strength of the affective responses to the fear structure probably promote strong avoidance responses from clients and this would again retard activation of the fear structure. According to this position, prolonged and repeated exposures to critical trauma-related stimuli (memory fragments), through talking about the event, guided recollection, and/or in vivo exposure is necessary to reactivate the fear structure.
sufficiently for reprocessing to take place.

Foa and colleagues (1989) asserted that prolonged and repeated exposure to trauma-related stimuli brings about change in several ways: (a) decreases in physiological responsivity within sessions due to habituation; and (b) changes in meaning of the fear structure by decreasing the perceived probability of recurrence (i.e., anxiety provoking stimuli no longer reliably predict the occurrence of the trauma) and decreases in the negative valence of the stimuli or physiological/affective responses (i.e., the association between the stimuli and an anxiety state is weakened and/or the perceived threat of an evoked anxiety state is lessened. Accordingly, any number of exposure procedures (see below) may bring about positive therapeutic shifts, and these may be especially useful when combined with specific cognitive work such as rational evaluation of the event and irrational beliefs and reframing of irrational or otherwise dysfunctional associations (e.g., Janoff-Bulman, 1985).

Similar to the above, Janoff-Bulman's (1985) model of PTSR suggests that traumatized individuals need to reestablish elements of the trauma-shattered assumptive world that enables people to cope with life's stresses and uncertainties. Interventions are focused on facilitating the assimilation-accommodation process to reestablish some level of belief that (a) one is invulnerable to harm, (b) the world is meaningful, and (c) the self is decent and worthy. One approach recommended (see Janoff-Bulman, 1985) is redefining the event so as to reduce the incongruence with and hence the impact on the client's assumptive world. Here, rational evaluations and reframing come into play to enhance self-perception, reduce the import of the event, and determining what benefits

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the client might have gleaned from the event.

For an example of the above, characterological self-blame (attributing blame to stable, personal characteristics) has been associated with poor post-trauma coping while behavioral self-blame (attributing blame to behaviors one did or did not engage in at the time of the event) has been associated with good coping (Janoff-Bulman, 1979). By challenging characterological self-blame and replacing it with behavioral self-blame, a client's prognosis is presumably improved. Another broad strategy is to search for meaning in the event. By finding some meaning or purpose in a traumatic event, clients are thought to be more apt to seeing the world as a comprehensible (Frankl, 1963). Often, this involves challenging negative attributions about the event and replacing them with positive, more adaptive forms (Janoff-Bulman, 1985). Behaviors aimed at preventing further trauma and confronting situations irrationally feared because of the trauma, and seeking early social support are recommended (Janoff-Bulman, 1985).

**Stress Inoculation Training.** Stress inoculation training (SIT) was developed by Meichenbaum (1985) as a means of training individuals to better manage stress. Its conceived utility in treating PTSD is based upon the cognitive-behavioral notion that the core problem in PTSD is the individual's responses to memories of the traumatic event (Keane et al., 1985). It follows then, that a primary treatment focus should be to decrease or otherwise alter such reactions.

The core purpose of this treatment approach is to educate individuals regarding the nature of stress reactions and their capacity to cope with them, to facilitate the acquisition of skills which will enable coping, and to apply these skills in daily life to

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facilitate a generalized change in coping style. These correspond to the education, acquisition, and application phases of SIT (Meichenbaum, 1985). During the acquisition phase, any number of cognitive behavioral techniques or skills may be employed, depending upon the problem at hand and the proclivities of the individual. These might include cognitive restructuring; guided self-dialogue; and training in relaxation skills, problem solving skills, assertiveness, social skills, and time management, et cetera (Meichenbaum, 1985). The application phase might include in vivo exposure, homework assignments, imagery rehearsal, modeling, role-playing, and relapse prevention training (Meichenbaum, 1985). A benefit of SIT is that training is relatively short term, usually between one and ten sessions, and is presumed to provide enduring and generalizable benefits (Meichenbaum, 1985). Additionally, SIT lends itself to group administration, and one report suggests it is preferred by therapists and patients alike over systematic desensitization (see below) and peer counseling (Veronen & Kilpatrick, 1983).

The use of SIT in treating PTSD has been limited but some encouraging results have been reported. For example, Resick, Jordan, Girelli, Hutter, and Marhoefer-Dvorak (1988) found that six session of group administered SIT brought about lasting improvement in the symptoms of rape victims, especially with symptoms of fear and anxiety. Others have observed similar results with rape victims (Frank et al., 1988; Veronen and Kilpatrick, 1983). Ayalon (1983, cited by Peterson et al., 1990), in an uncontrolled study, reported significant symptom reduction with child survivors of a terrorist attack. Stress inoculation training does not represent a single intervention but a treatment strategy which employs diverse cognitive-behavioral techniques. According-
ly, its utility in treating PTSD may illustrate the utility of cognitive-behavioral intervention in general. To date, however, insufficient evidence is available to warrant conclusions about its overall effectiveness.

**Positive Visualization.** The use of positive visualization (PV) to reduce distress levels may be practiced within any therapeutic approach. Scrignar (1988) attests that positive encephalic practice (his term for positive visualization) has utility in helping to break the ruminative process common in PTSD. According to Scrignar, teaching clients to practice PV on a regular basis and to substitute PV for intrusive traumatic memories can reduce overall symptom severity. In this, the techniques appears to be analogous to thought stopping and thought substitution. No data is available on the utility of this technique, but it has an intuitive appeal.

**Behavioral Therapies**

As suggested in our survey of cognitive therapies, there is considerable overlap between cognitive and behavioral treatments for anxiety disorders. The term cognitive-behavior therapy, probably employed as much as either element separately, reflects this overlap. That said, the following discussion will focus upon those interventions, based on extinction or counterconditioning, that might readily be characterized as behavioral. Of the non-pharmacological interventions explored, this class has received the most convincing research validation to date, though this may reflect the research-orientation of behavioral psychologists as much as any true difference in treatment efficacy.
Systematic Desensitization. Systematic Desensitization (SD) is a treatment based upon the principles of Pavlovian or respondent conditioning (see Pavlov, 1960). According to this model, a victim's natural response to a traumatic event includes high levels of fear and anxiety. Stimuli present during the event, by virtue of being paired with unconditioned and pre-conditioned stimuli associated with the event itself, may become conditioned stimuli capable of eliciting conditioned responses similar to those elicited during the original trauma. In other words, previously innocuous stimuli come to elicit a fear response, and it is this conditioned response which is seen as dysfunction in PTSD. According to Pavlovian principles, elimination of such a response can be effected through exposure to the conditioned stimuli in the absence of the unconditioned stimuli. This process is called extinction (Pavlov, 1960).

Briefly, in SD the individual is first taught to bring about a state of deep muscle relaxation through any of a variety of relaxation training techniques (see Walker, Hedberg, Clement, & Wright, 1981), and then constructs a fear hierarchy. This is comprised of an identified series of fear producing stimuli of varying strengths (capacity to elicit fear) arranged in ascending order of strength. One variation of the treatment involves the induction of a relaxed state followed by visualization of the weakest fear producing stimulus. When this stimulus can be imagined without a fear response, the individual progresses to the next stronger stimulus and so on. This imaginal treatment is complete when the affected individual can imagine the most fear producing stimulus without experiencing fear (see Walker et al., 1981). Another variation of this involves progressive in vivo exposure to the hierarchy of feared stimuli (Walker et al., 1981). It
has been asserted that such in vivo desensitization is superior to the imaginal form and is the preferred treatment when logistics permit (Marks, 1981; Mavissakalian & Barlow, 1981; Scrignar, 1988).

Systematic desensitization has been used to successfully treat phobic anxiety (e.g., Wolpe, 1982; Marks, 1981); however, it has been proposed as a treatment for the phobic-like anxieties of PSTD as well (e.g., Peterson et al., 1990; Scrignar, 1988). Scrignar (1988) claims that SD (imaginal or in vivo) is the treatments of choice for phobic anxiety within PTSD. Others (e.g., Peterson et al., 1990; Resick et al., 1988) assert that SD has not received enough clinical validation in use with PTSD to warrant such a claim. If clinical practice follows the literature, SD systematic desensitization by itself does not appear to have been widely employed as a treatment for PTSD to date: Though a number of case studies (e.g., Becker & Abel, 1981, cited in Resick et al., 1988; Schindler, 1980; Wolff, 1977), limited scope studies (e.g., Bowen & Lambert, 1986; Kipper, 1977), and studies wherein SD was used as part of a treatment package (e.g., Frank et al., 1988; McCaffrey & Fairbank, 1985) have made their way into the literature, the use of SD in the treatment of PTSD has received less attention than one might expect in a research oriented field. A number of possible explanations suggest themselves: (a) by itself, systematic desensitization may not be a particularly effective treatment for post-traumatic sequela; and (b) for many trauma victims, fear inducing stimuli may not be easily ordered in the required hierarchy, particularly if there are only a few isolated trauma-related stimuli. The latter may be particularly true for in vivo desensitization.

As with all treatments reviewed, further studies are required to adequately
validate SD as a treatment for PTSD. Most of the reports available have been positive regarding the utility of SD with treating PTSD, but Becker and Able (1981, cited in Resick et al., 1988) found it to be ineffective. Among the treatments discussed, a benefit of SD, especially the imaginal variety, is that it lends itself to group- and self-administration (Walker et al., 1981). Additionally, the in vivo form may result in a reduction in avoidance behavior, though there is little evidence to support this.

**Flooding and Implosion Therapy.** Flooding (Malleson, 1959) and implosion therapy (Stampfl & Levis, 1967), like SD, are exposure-based therapies aimed at eliminating aversive conditioned responses through extinction. Unlike SD, however, both flooding and implosion employ the most anxiety provoking stimuli identifiable from the outset. This can be done imaginally or in vivo. The rationale is to expose the individual to the strongest conditioned stimuli and maintain exposure until the conditioned aversive response abates (see Walker et al., 1981). Flooding and implosion are very similar in procedure, differing primarily in theoretic foundations. For the most part, the rest of the discussion will focus upon flooding, since it is based solely on behavioral foundations. Although flooding can be carried out without prior training in relaxation (e.g., Walker et al., 1981), Keane and colleagues' approach (Lyons & Keane, 1989; Keane et al., 1985) employs training in relaxation and practice at inducing imagery. It also calls for the hierarchical presentation of traumatic scenes and stimuli. Accordingly, this variation is a cross between classical flooding and SD.

A number of reports support flooding's effectiveness in treating PTSD. These represent both case studies (e.g., Black & Keane, 1982; Fairbank & Keane, 1982;
Haynes & Mooney, 1975; Keane & Kaloupek, 1982; McCaffrey & Fairbank, 1985; Rychtarik, Silverman, Van Landingham, & Prue, 1984; Saigh, 1978, 1987), and group investigations (e.g., Cooper & Clum, 1989; Keane, Fairbank, Caddell, & Zimering, 1989). Results generally indicate that flooding reduces an individual's reactivity to trauma-related imagery and stimuli, the frequency of intrusive symptoms such as flashbacks and nightmares, levels of depression, and levels of anxiety in general. In some of these studies (e.g., Fairbank & Keane, 1982; Keane & Kaloupek, 1982; McCaffrey & Fairbank, 1985) both self-report and physiological indices of reactivity were used. Flooding generally does not appear to affect numbing or avoidance symptoms.

Flooding and implosion therapy, though they are currently considered by some the treatment of choice for PTSD (e.g., Keane et al., 1985), are troublesome because of the necessity of exposing the patient to high levels of anxiety for a protracted period. Several problems have been identified with this: (a) there is an ethical concern over compelling patients to endure the intense anxiety levels that may be involved (e.g., Fairbank and Brown, 1987); (b) the use of implosion therapy may initially increase patient anxiety levels and precipitate a premature termination of treatment, resulting in treatment-exacerbated sequelae (Kilpatrick & Best, 1984); and (c) the treatment may be contraindicated for some patients because concurrent diagnoses, psychological endurance, and/or physical health (Litz, Blake, Gerardi, & Keane, 1990). Although the results of Shipley and Boudewyns' (1980) survey suggest implosion is a relatively safe procedure, the above remain troubling considerations. Additionally, Peterson and
colleagues (Peterson et al., 1990) point out that the available research results are insufficient to establish flooding or implosion as a treatment of choice. Nevertheless, the literature does support the efficacy of flooding and implosion in reducing reactivity to trauma-related stimuli and the intrusive symptoms of PTSD.

Other Behavioral Techniques. A number of other behavioral interventions and techniques have been used to treat some symptoms of PTSD. These include assertiveness training (AT), thought stopping (Wolpe, 1958) and behavioral rehearsal. The role of AT in the treatment of PTSD has a good rational basis: Traumatized persons, especially victims crimes like sexual assault, often withdraw socially or otherwise develop interpersonal problems (Atkeson et al., 1982; Davidson et al., 1991; Pearce et al., 1985; Resick, Calhoun, Atkeson, & Ellis, 1982; Resick et al., 1988; Solomon & Mikulincer, 1987). Additionally, non-assertiveness, whether trauma-induced or premorbid, is generally associated with elevated levels of anger and resentment (Rimm & Masters, 1979; Scrignar, 1988). Assertiveness training would presumably benefit such individuals to the extent that it facilitates successful social re-engagement. Additionally, behaving assertively is thought to be incompatible with anxiety (Wolpe, 1958, 1969). Accordingly, by facilitating assertive behavior in general and especially in anxiety provoking situations, one might expect overall and situation specific reductions in anxiety. The present review uncovered only on investigation of the efficacy of AT with PTSD. Resick and colleagues (Resick et al., 1988) report that AT, conducted in a group format, brought about lasting reductions in fear and anxiety levels of rape victims as compared to wait-list controls. Unfortunately, the AT used incorporated cognitive
restructuring and so it is not possible to attribute these changes to AT alone. The utility of AT in treating PTSD has reasonable face validity and intuitive appeal, but little evidence to support it at present.

Scrignar (1988) asserts that thought stopping can be employed to successfully reduce anxiety producing traumatic thoughts, but offered no research data to support his claim. Fairbank, DeGood, and Jenkins (1981) employed behavioral rehearsal to reduce anxiety levels of an individual who, subsequent to a motor vehicle accident, experienced anxiety when driving. They employed a combination of relaxation training and in vivo rehearsal (exposure). Though the results were positive, with anxiety levels decreasing significantly, the procedures used are difficult to differentiate from a modified flooding procedure. These and other behavioral techniques may be useful components to a broader treatment program, such as SIT, but there is little to support their solitary use.

Group Therapy

Group therapy for individuals with PTSD appears to have begun in the 1970's with the emergence of self-help discussion groups of Vietnam veterans (Brende, 1981; Shatan, 1973). Group therapy in general has been credited with having several advantages over individual therapy including reduced costs, efficient use of therapist time, built in peer support, and social facilitation (see Corey & Corey, 1987; Yalom, 1985). Some have asserted that group therapy may be of particular benefit to individuals with PTSD because of the homogeneity of symptoms across members (e.g., Scrignar, 1988) and the unique ability of survivors to validate each other's experiences in trauma.
homogeneous groups (e.g., Brende, 1981; Makler, Sigal, Gelkopf, Kochba, & Horeb, 1990; Walker & Nash, 1981).

As with individual therapy, the content and process of group therapy is affected by the theoretical orientation of the therapist(s). Groups can differ in the selection criteria for members, the role of the therapist, the focus of the work, the number of sessions, among other dimensions (see Corey & Corey, 1987; Peterson et al., 1990). Peterson and colleagues (Peterson et al., 1990) have identified four primary group types used in the treatment of PTSD: interactive (support) groups, wherein the focus is on open exchange between group members on diverse issues; cognitive-behavioral groups, wherein the focus is a problem solving and skills acquisition aimed to deal with troublesome thoughts and behaviors; psychodynamic groups, wherein the focus is on the recognition and processing of transference and countertransference as it arises within the group process; and Jungian groups, wherein the focus is upon analysis of dreams. A detailed discussion of these variations is beyond the intended scope of this section.

The effectiveness of group therapy is difficult to assess due to a dearth of reports in the literature. What reports do exist (e.g., Brende, 1981; Cryer & Beutler, 1980; Shatan, 1973; Resick et al., 1988; Walker & Nash, 1981) suggest they are effective in bringing about significant symptom relief, but only Resick and colleagues report findings from a formal investigation of efficacy. Also, the issue is complicated by the use of specific therapeutic techniques within groups. For example, Resick and colleagues' study employed SIT and assertiveness training. The question of whether the group format added to or subtracted from the efficacy of these interventions as compared...
to individual administration cannot be answered this time. The group format does have apparent advantages, and the proliferation of support groups for trauma survivors around the country suggests they offer the paying public something, but clearly more research is needed in this area.

Hypnosis

Hypnosis, though commonly associated with psychodynamic therapy, is a procedure that might be used within any therapeutic approach. Patients with PTSD as a group have been observed to be significantly more hypnotizable than patients with other diagnoses and non-patients (Pettinati, 1982; Spiegel, 1981; Spiegel, Hunt, & Dondershine, 1988; Stutman & Bliss, 1985). This is generally attributed to an enhanced ability to dissociate. One implication of this is that individuals with PTSD may be primed to benefit from hypnosis (Peterson et al., 1990).

Abreaction is the classic goal of hypnosis in treating PTSD (Buchenholz Frank, 1948; Fisher, 1945; Leahy & Martin, 1967; Spiegel, 1981). Accordingly, within a dynamic framework, hypnosis can be used to uncover repressed memories (e.g., Peterson et al., 1990; Silver & Kelly, 1985) and enhance revivication (e.g., Peterson et al., 1990; Scrignar, 1988). Such revivication is considered by many to be an important part of reprocessing or working through the memory as discussed in earlier sections (e.g., Brende & Benedict, 1980; Peebles, 1989b; Spiegel, 1981). In cases of acute PTSD, such abreaction has been observed to effect rapid and dramatic reductions in symptoms; however, in chronic cases of PTSD, abreaction is considered inadequate by itself but
may enhance a more comprehensive therapeutic approach (e.g., Brende & Benedict, 1980; Scrignar, 1988).

The dissociative process common in PTSD is thought to cause intrapsychic splits in which some emotions or aspects of the personality (e.g., competence and autonomy) become hidden or otherwise unavailable to the trauma survivor (e.g., Brende, 1985; Brende & Benedict, 1980; Brende & McCann, 1984; Spiegel et al., 1988). Hypnosis is reportedly effective in aiding the reintegration of these splintered aspects of the self (e.g., Brende, 1985; Brende & Benedict, 1980; Phillips, 1993b; Spiegel et al., 1988).

Just as hypnosis might be used in the context of dynamic therapy, Scrignar (1988) asserts that hypnosis, especially self-hypnosis, can be used to aid relaxation and cognitive restructuring. Here, hypnosis might be used simply to enhance positive imagery, reinforce thought stopping and thought substitutions, alter cognitive distortions, or it might be used to uncover traumatic memories and provide a form of cognitive correction (Phillips, 1993b; Scrignar, 1990). It may also be used to help individuals restructure or rescript traumatic events (e.g., Mutter, 1987; Peebles, 1989b; Peterson et al., 1990). By vividly imaging the event in a new way, one which is less aversive, the re-scripted memory is presumed to permit completion of unfinished business or otherwise counteract the effect of the original.

Several authors have supported the use of hypnosis as an ego-strengthening device in the treatment of PTSD (e.g., Peebles, 1989b; Phillips, 1993a, 1993b; Spiegel et al., 1988). The focus of this type of work is to rebuild ego-strength by engaging in imagery and suggestions of situational and personal mastery (e.g., McNeal & Frederick,
1993; Frederick & Phillips, 1992; Phillips, 1993a, 1993b). In a sense, this constitutes hypnotically enhanced positive visualization. The rationale behind this is that enhanced ego-strength permits the individual to better confront and cope with traumatic memories, thereby aiding the working through process, and providing a buffer against future insult.

Reports on the efficacy of hypnosis in treating PTSD are numerous (e.g., Brende, 1985; Brende & Benedict, 1980; Ebert, 1988; Mutter, 1987; Peebles, 1989b; Silver & Kelly, 1985; Somer, 1990; Spiegel, 1981). These reports are, for the most part, case studies or are otherwise anecdotal and controlled studies are lacking. Additionally, hypnosis is a procedure that is most typically used within a broader therapeutic program, making it all the more difficult to draw conclusions about its unique efficacy in treating PTSD. Accordingly, the use of hypnosis in treating PTSD, like all other interventions discussed thus far, is supported by the literature, but more research is needed to firmly establish it as an effective treatment.

**Summary of Treatments for PTSD**

A variety of approaches to treating PTSD were presented. It is clear that, despite apparent differences in theoretical rationales, a great deal of overlap exists between treatment approaches. When psychodynamic theorists speak of desensitizing a client to a traumatic memory by verbal reexposure an overlap with behavioral clinicians is obvious. Likewise, cognitive restructuring appears to be synonymous with ego restructuring, and all therapies discussed advocate in vivo confrontation (i.e., exposure) of traumatic stimuli. Real conceptual and methodological differences do exist and, in
theory, the content of different therapies should differ accordingly; however, these differences probably disappear to some extent in practice. Additionally, the process of the various therapeutic approaches is probably more similar than different outside of the rigor of controlled investigations. In Jerome Frank's words, "...features common to all types of psychotherapy probably contribute as much, if not more, to their effectiveness than the characteristics that differentiate them" (Frank, 1974, p. 23).

It is generally accepted that medication can be a valuable but temporary measure in treating the effects of trauma, and that some form of psychotherapy is desirable to ameliorate enduring sequelae. As with explanatory schemes, no treatment has emerged as a clear treatment of choice. There is more evidence from formal studies to support the efficacy of implosion/flooding than other treatments, but the evidence is by no means conclusive. Additionally, the choice of therapies for PTSD is as likely to be decided by personal inclinations of both therapist and client as by the support of research. In the absence of clear evidence of superiority, it probably behooves all clinicians in the field to be systematic in their approach to therapy, regardless of their orientation, so as to maximize the efficacy and efficiency of their work.

It seems likely that each therapeutic approach discussed has merit and that, at present, the best approach to therapy might be a flexible one, employing content and process features from several schools to fit the preferences and needs of a given client. This does require greater skill and sophistication on the part of the therapist than a monotheistic approach, however. Because more controlled research into the efficacy of all the treatments discussed is required, outcome research which isolates specific
interventions may be of greatest benefit at this time.

Eye Movement Desensitization: A New Treatment for Post-Traumatic Stress Disorder

In recent years, a new treatment for post-traumatic sequelae has emerged. This procedure, called eye movement desensitization (EMD), was developed by Francine Shapiro (1989a, 1989b, 1990, 1991). Shapiro claims EMD was adventitiously discovered and empirically developed (F. Shapiro, personal communication, April 14, 1991). Accordingly, despite the behavioral connotations of its name, EMD is not associated with any particular theoretical orientation.

The Intervention

Briefly stated, during the treatment the affected patient selects a visual image that subjectively best represents the traumatic event(s) at its worst. Such imagery may correspond to events as they were actually seen during the trauma, or they may be constructed to make the best fit with available memory. The content of recurring trauma-related nightmares can also be used. The patient then constructs a statement that matches the selected image, that is, one that reflects troubling or otherwise dysfunctional cognitions about the event or the self. Such statements may consist of cognitive errors (e.g., Beck, 1976), irrational beliefs (e.g., Ellis, 1962), aversive cognitions that were generated at the time of the incident, or statements that generate aversive emotional responses and/or inhibit adaptive behavior.
The patient then briefly visualizes the image and repeats the statement silently. Any emotions felt during the exercise are identified, the level of experienced distress is rated on a 0-10 SUD (Subjective Units of Distress) scale (Horowitz, Wilner, & Alvarez, 1979), and the perceived validity of the employed statement is rated by the patient on an eight-point scale (0 = completely valid, 7 = completely invalid). The patient then identifies how s/he would prefer to feel in response to the imagery, constructs a cognitive belief statement that reflects this alternate feeling, and rates the perceived validity of this new statement on the same 8-point scale (0 = completely valid, 7 = completely invalid). This procedure establishes the positive and negative belief statements and constitutes an initial assessment of reactivity to both.

The patient then visualizes the image, repeatedly rehearses the aversive statement covertly, and visually follows the therapist's finger as it is repetitiously moved across the patient's visual field. These "sweeps" are made by placing a finger or pen about 14 inches from the patient's face and moving it back and forth from one periphery to the other, typically traversing about 16 inches. This is done at a rate of 2 cycles per second, one cycle consists of movement from one side of the visual field to the next and back. This is done for 6 to 10 seconds and then the patient is instructed to take a deep breath and to "blank-out" the visual image. The patient again visualizes the image, rehearses the statement, and rates the level of experienced discomfort on the SUD scale. This constitutes a single EMD "set".

During the imagery sets, patients are asked to attend to their experiences in four modes: images, cognitions, emotions, and physical sensations. If the patient reports
changes in any mode, those new stimuli are the focus of attention during the next set. During subsequent sets, the patient is instructed to stay with whatever changes occur during the immediately preceding set such that s/he is always attending to emergent stimuli. This is continued until such "movement" ceases, and then the patient is instructed to return to the original image and cognitive statement. This process is repeated until the SUD rating for the original image and cognition drops to zero or asymptotes at one. At this point "desensitization" is considered complete and the "installation" procedure begins.

For installation, the patient is asked to visualize the image and rehearse the desired statement while tracking the therapist's finger as described above. Again, patients attend to emerging stimuli during sets but are always instructed to return to the original image and desired statement. Installation sets are repeated until the rated validity of the desired belief statement reaches seven (completely valid) or asymptotes. If the validity of the desired cognition does not reach seven, the patient is queried about possible reasons. Any perceptions or beliefs that appear to be blocking progress are assessed for appropriateness or ecological validity. For these purposes, appropriate beliefs are those which are rationally defensible or otherwise adaptive for the individual. The ecological validity of a belief statement refers to the effect that such a statement may have on the overall welfare and adaptation of the individual to his or her life as a whole. If they are assessed by the clinician to be irrational or otherwise non-adaptive, those blocking cognitions are the focus of another series of EMD sets. EMD is complete at the end of the installation procedures.
Treatment Effects and Clinical Validation

Shapiro (1989a), in her original assessment of the treatment, reports that in 22 participants presenting with serious post traumatic sequelae such as flashbacks (mean = 3/week), intrusive thoughts (mean = 12/week), and sleep disturbances (mean = 4/week), a complete reduction in emotional reactivity to traumatic imagery and an attenuation of trauma-related cognitions was produced in one 50-minute session. Further, at 1- and 3-month follow-ups, the primary presenting problems (e.g., flashbacks) of all participants had either completely attenuated (N=14) or had significantly improved (N=6). That the traumatic memories for these participants had persisted for an average of 23 years, that they had been in therapy for an average of 6 years, and that the treatment produced less participant discomfort than flooding or implosion would have, further attests to the effectiveness and efficiency of the intervention.

To date, nine other reports of EMD's efficacy in treating PTSD have made their way into the literature (Boudewyns, Stertka, Hyer, Albrecht, & Sperr, 1993; Kleinknecht & Morgan, 1992; Lipke & Botkin, 1992; Marquis, 1991; McCann, 1992; Puk, 1991; Sanderson & Carpenter, 1992; Shapiro, 1989b; Wolpe & Abrams, 1991). Of these, seven are case studies (Kleinknecht & Morgan, 1992; Lipke & Botkin, 1992; Marquis, 1991; McCann, 1992; Puk, 1991; Shapiro, 1989b; Wolpe & Abrams, 1991). Shapiro (1989b) reported a replication of her earlier findings but did not use standardized psychological tests to assess treatment effects. Similarly, Lipke and Botkin (1992) reported mixed but generally positive results with five chronic PTSD patients, McCann (1992) reported very
positive results with one patient, and Puk (1991) reported positive results with two patients, each using a single session of EMD. McCann (1992) and Puk (1991) reported follow-up data of 12 and 6/12 months, respectively, indicating a maintenance of treatment effects. Marquis (1991), reported on using EMD with 78 cases of varied anxiety disorders, including 16 cases of PTSD. Using a estimate of global improvement based on clinical judgement and patient report, he reported highly significant improvements in symptoms (cured or nearly cured) on average. Although these effects appear similar to those reported by Shapiro, standardized psychological tests and diagnostic tools were not used in assessment.

In another study, Sanderson & Carpenter (1992) reported on the efficacy of EMD in treating phobic fears, including the traumatic phobias of eight participants. In this two-treatment crossover study, subjective units of distress (SUD) ranging from zero (no distress) to 100 (maximum distress) were used to assess reactivity to phobic imagery. Subjective ratings of distress for the four post-trauma participants initially receiving EMD dropped from an average of 65.4 to 7.5. For the four receiving EMD as a second intervention, the SUD ratings dropped form 43.8 to 6.3. Unfortunately, because of the cross-over design, no determination could be made regarding the unique efficacy of EMD alone in reducing imagery-related distress, especially at follow-up. Additionally, the control procedure, image confrontation (IC), which was identical to EMD except that participants' eyes remained closed and stationary, produced equivalent therapeutic effects. This differs from the results of Shapiro's (1989a) study in which the control condition, very similar to IC, produced no therapeutic effects at all.
In each of the above studies, standardized or otherwise structured assessment tools were not employed, calling into question the validity of the reported results. Some case studies have fared better, however. Wolpe and Abrams, employing a number of structured assessment tools, reported on the effects of eight sessions of a modified version of EMD with a single patient. The patient's level subjective distress upon trauma recall reportedly diminished significantly with treatment, as did scores of the Fear Survey (Wolpe & Lang, 1969) which provided a more global rating of overall fear level. This patient also reported collateral changes indicative of reduced fearfulness and social dysfunction. The utility of this study in assessing the efficacy of EMD is hampered by several factors. Wolpe and Abrams used a very informal follow-up procedure and reported no specific findings. Additionally, the treatment procedure used was not EMD per se: it was a combination of systematic desensitization and EMD, making conclusions about the efficacy of EMD questionable.

Kleinknecht and Morgan (1992) employed standardized psychological tests for anxiety and depression, a comprehensive test of specific and global psychosocial functioning, and an in vivo exposure test to assess the effects of a single session of EMD. The patient was an individual with an eight-year history of PTSD stemming from multiple traumata. The results indicated a significant reduction in general anxiety level and depressive symptoms, and on all other indices of troublesome psychosocial functioning. The client reported that direct exposure to the trauma scenes and other trauma related stimuli did not elicit an aversive emotional response, and that trauma-related nightmares abated immediately upon treatment. These effects were maintained
or increased at four and eight month follow-ups. The results, therefore, are congruent with Shapiro's (1989a, 1989b) and represent the best documented case-study replication of Shapiro's work to date. Case studies, however, are limited in their capacity to validate previous findings.

Boudewyns and colleagues (Boudewyns, Stertka, Hyer, Albrecht, & Sperr, 1993), in a recently published controlled study of EMD, also employed psychological and physiological measures and a standardized diagnostic protocol. They found a significantly greater decrease in subjective anxiety in the EMD group than with exposure control and milieu control groups during treatment procedures, but no significant differences either in subjective distress or physiological measures during a standardized testing condition. Additionally, no differences were found between groups on pre- and post-treatment measures using standardized psychological tests.

As impressive as the reported efficacy of EMD seems, Herbert and Mueser (1992) point out serious methodological flaws in four of the cited studies that prohibits conclusions about EMD's efficacy in treating PTSD. These include a lack of objective assessment of symptoms, a lack of appropriate controls, and possible experimenter bias and demand characteristics (Herbert & Mueser, 1992). These same criticisms could be applied to three of the five other studies cited. It would appear that EMD holds promise as a new treatment for at least some symptoms of PTSD. Research to date, however, does not permit an informed assessment of its efficacy. Shapiro (1989a, 1990, 1991) draws upon the same limited data base in each of her reports, and, as with all treatments previously discussed, well controlled replicating outcome studies are needed to firmly
establish EMD as an effective treatment for PTSD.

**Proposed Explanations of the Efficacy of Eye Movement Desensitization**

Shapiro has proposed that a Pavlov's (1927) speculations about the neurophysiological basis of neuroses and their treatment may help explain the efficacy of EMD (Shapiro, 1989a). According to her interpretation of Pavlov's work, an excitatory-inhibitory balance within the brain is necessary for complete information processing to occur. During a traumatic event, an over-excitation of neuronal structures takes place, creating a semi-permanent excitatory locus. This upsets the normal excitatory-inhibitory balance and inhibits a complete processing and integration of the event. Post-traumatic sequelae such as nightmares, flashbacks, and intrusive thoughts presumably occur when this excitatory locus is stimulated. According to Shapiro (1989a, 1991), engaging in the type of eye movements engendered during EMD while the excitatory locus is stimulated through imagery and cognitive rehearsal may produce a number of therapeutic effects. It may (a) restore the brain's excitatory-inhibitory balance, (b) reverse neuronal pathology, and/or (c) drive or at least permit complete information processing of the traumata to take place. Shapiro (1989a, 1991) also speculates that the mechanism whereby this occurs may be the effect that the rhythmic eye movements have on cortical activity. According to this idea, rhythmic eye movements may be a component of a natural desensitizing process, as may be reflected in the rapid eye movements observed in sleep. By engaging in such eye movements, one might be driving this desensitization process (Shapiro, 1989a).
Shapiro's citation of Pavlov is questionable as her application of his work in interpreting the efficacy of EMD appears to employ a mixture of Pavlovian and cognitive concepts. A review of Pavlov's original work (Pavlov, 1960) indicates that he did attribute neuroses, and in particular traumatic neuroses, to a disruption in the normal balance between excitatory and inhibitory reflexes by extraordinary stimulation (Pavlov, 1960). However, he did not appear to speak to a process analogous to information processing. Shapiro's interpretation may reflect an integration of Pavlovian and cognitive concepts or a misunderstanding of Pavlov's original work, in either case her citation is questionable. This notwithstanding, Pavlov's work might be relevant to the process underlying EMD's apparent effects. Pavlov reported that the use of weak excitatory stimuli with a dog which was hyperreactive (i.e., inhibitory reflexes were halted and excitatory reflexes were exaggerated) paradoxically resulted in a rapid diminution of this excessive excitability (Pavlov, 1960). It is tempting to speculate that the trauma-related visual and verbal stimuli engaged in during EMD is weakened by the finger tracking task. It would follow that such may be weak excitatory stimuli of the sort Pavlov speaks of and may result in a rapid reduction in reactivity to trauma-related stimuli. This is pure speculation, however, and may be premature prior to confirming EMD's therapeutic effects and identifying the procedural features responsible for it.

As to the relationship between eye movements and a natural desensitization / processing mechanism, it is a compelling idea which may deserve further attention. Lipke and Botkin (1992) point out that enhanced information processing is thought to occur during rapid eye movement sleep. Although no correspondence has been
established between the rate and range of rapid eye movements occurring during such sleep and those engendered during EMD, such cannot be ruled out at this time. However, even if such a correspondence is established, it would remain to be demonstrated that engaging in such eye movements can drive enhanced information processing. Marquis (1991) asserts that the neural correlates of rapid eye movements weaken the neural connections between covert visual stimuli and emotional responsivity: "Perhaps the field currents generated by the eye movements (the same as used to detect REM sleep) interfere with the tracts connecting the frontal lobes with the hypothalamus and hippocampus in such a way as to weaken the connection between stimulus and emotional response" (p. 192). This speculation does not appear to be founded on any accepted neurophysiological principles, however.

In a similar vein, Hedstrom (1991) has proposed a link between the eye movements of EMD and increased alpha activity in the cortex. Cortical alpha activity is usually associated with a state of attentive relaxation. According to this proposal, enhanced alpha activity, brought about through rhythmic eye movements, may force an association between trauma-related stimuli and relaxation. Again, such is purely speculative at this time. An association between rapid, rhythmic eye movements as engendered during EMD and increased alpha activity has not been demonstrated. On the other hand, Hedstrom himself notes that visually focusing on an object tends to block alpha. Nevertheless, determining the electro-encephalographic correlates of EMD may be a fruitful endeavor.

Although the role of eye movements in EMD's apparent therapeutic efficacy has
been the focus of speculation (e.g., Hedstrom, 1991; Lipke & Botkin, 1992; Marquis, 1991; Shapiro, 1989a, 1991), it remains to be established that they have any effect at all. As reviewed earlier, Sanderson and Carpenter (1992) found that image confrontation, produced equivalent results as EMD in reducing phobic-imagery induced distress. Although only one participant in this study was diagnosed as having PTSD, the EMD and IC procedures differed from Shapiro’s protocol in omitting the use of cognitive statements, and the crossover design did not allow for between treatment comparisons at follow-up, the results do suggest that eye movements may not have a specific therapeutic effect.

Shapiro (1991a) has also theorized that EMD may produce its effects through a number of other processes. These include (a) enhancing the client’s sense of mastery over troublesome internal stimuli through repeated self engagement and disengagement of traumatic imagery; (b) enhancing the client’s distinction between physiological reactivity to trauma-related stimuli and cognitive/affective interpretations of the same; (c) the desensitizing effects of exposure to trauma-related stimuli; and (d) incidental cognitive restructuring. These again are purely speculative and there is no basis upon which to critique them at this time.

Though the above speculations are interesting and should be given consideration, it seems premature to engage in serious conjecture until we know more about the treatment’s validity and have identified those components that are necessary and sufficient to produce therapeutic effects.
Questions and Issues Suggested by Reports of Eye Movement Desensitization

As impressive as the reported effects of EMD are, a number of important questions present themselves at this time. To begin with, can Shapiro's results be reliably replicated by independent parties? As indicated by the present review, research to date has been insufficient to validate EMD as an effective treatment for PTSD. Also, most reports to date have not employed standardized assessment tools to determine pretreatment status and to measure treatment effects. Such will be important in establishing clinical confidence in the procedure.

Another question that presents itself regards those components of the EMD procedure that are necessary and sufficient to produce the reported reductions in posttraumatic sequelae. The procedure outlined in the literature consists of at least eight components: (1) exposure to trauma-related visual imagery, (2) the rehearsal of negative self-statements or assessments of the trauma (negative cognitions), (3) rapid eye movements and the associated changes in visual field, (4) active visual attention on an object (moving finger or pen), (5) the eye-movement ritual in combination with close therapist proximity throughout the procedure, (6) thought and image stopping, (7) a deep breath, and (8) the rehearsal of a desired cognitive statement. The EMD procedure is complex enough that it might be thought of as a compact treatment package.

Because early reports suggest EMD may produce lasting therapeutic benefits more efficiently and/or with less discomfort than more established interventions, it may be fruitful to employ dismantling procedures on it. Such investigations may elucidate the mechanisms responsible and lead to further therapeutic advances. They may also help
identify the physiological and psychological processes responsible for the development and amelioration of PTSD. Of the components of EMD listed above, the rapid eye movements and active visual attention during the rehearsal of traumatic images and cognitions are unique in the treatment literature. Because of this, any dismantling procedure might best begin with these features.

Another question presenting itself regards the effects of EMD on the self-assessed validity of the client's negative self/event statements. During the procedure outlined by Shapiro, the validity of only the desired cognition is assessed at pretreatment, posttreatment, and follow-up. No posttreatment or follow-up assessments are made of the negative cognitions. Though one might suspect that any decrease in the perceived validity of the negative cognitions would accompany the reported increases in the perceived validity of the desired cognitions, such should be an empirical issue, not a logical assumption.

Additionally, the possibility exists that the reported increases in the validity of the desired cognitions is, to some extent, attributable to a subtle shaping of patients' verbal responses. Once participant SUDS ratings drop to zero or asymptote at one while using the negative cognition, Shapiro repeated the EMD procedure with participants rehearsing the desired cognition. During every assessment trial following these altered treatment trials, participants were asked to rate the validity of the desired cognition, which was not done with the negative cognitions, and this was repeated until the validity of the desired cognition reached seven or an asymptote. It is feasible that demand characteristics and subtle reinforcement during this final treatment phase account for at
least a portion of the treatment's apparent effectiveness. Alterations of the EMD protocol might be made to avoid or reduce the likelihood of this. Alternately, using more objective, standardized tests to assess pre-treatment status and treatment effects would help to establish the verity of these ratings.

A final issue is a technical one. The EMD procedure, as it is currently conducted, involves the rapid movement of a therapist's hand across the visual field of the client, and such movements are unavoidably varied and imprecise. Though this may not be relevant to the efficacy of the treatment, it may retard attempts to conduct well controlled and standardized investigations, particularly into the psychophysiological basis of the procedure. If the procedure can be automated (i.e., employing a mechanical or electronic device) to enable more precise control over eye movements with no loss of efficacy, well controlled investigations may be made possible.

Purpose of the Current Study

The purpose of the present investigation was to address a number of the questions posed above. Specifically, the study attempted to accomplish the following: (a) to replicate Shapiro's work using the treatment procedure as reported in the literature; (b) to determine the efficacy of the EMD procedure when eye movements are engendered through a light tracking task using a device that allows greater precision and more distal proximity of the therapist; (c) to assess the efficacy of active visual attention without eye movement during a treatment protocol that is otherwise identical to the standard EMD protocol; (d) to assess any changes in negative cognitions that might
accompany successful treatment; (e) to use changes in heart rate to measure EMD's effects on physiological reactivity to trauma-related imagery; and (f) to employ standardized psychological tests to further validate treatment effects.
CHAPTER III

DESIGN AND METHODOLOGY

Participant Recruitment

Participants were recruited through advertisements published in a local newspaper and through referrals by a number of service agencies. Referring agencies included Ingham County Community Mental Health in Lansing, Michigan; Kalamazoo Consultation Center of Kalamazoo, Michigan; Michigan State University Counseling Center in East Lansing, Michigan; Mid-America Consultants of Portage, Michigan; and The Samaritan Center of Elkhart, Indiana.

For participation, persons were required to be at least 18 years of age, to have experienced a traumatic event as defined by the DSM-III-R (APA, 1987), and to have current intrusive symptoms as defined by the DSM-III-R. Screening for participation occurred during the initial assessment interview which, along with exclusion criteria, is described below.

Setting

The investigation was carried out in two centers, the clinic of the Western Michigan University Psychology Department in Kalamazoo, Michigan, and the Counseling Center of Michigan State University in East Lansing, Michigan. At each
treatment center, the intervention was conducted in a quiet, private room with only the therapist and participant present.

Apparatus

The apparatus and materials employed in this study included the Clinician-Administered PTSD Scale (CAPS) (Blake et al., 1990), a condensed version of the Revised Symptom Checklist (SCL-90-R) (Derogotis, 1977), the Impact of Event Scale (IES) (Horowitz, Wilner & Alvarez, 1979), and an informed consent form (see Appendix A). An electronic device designed to produce either "therapeutic eye movements" in participants through tracking alternately flashing lights or to maintain active visual attention through requiring a participant to attend to a fixed illuminated light were used, as was a heart rate monitoring device. In addition, the Drug Abuse Screening Test (DAST) (Skinner, 1983), medical clearance forms (see Appendix B), and information release forms (see Appendix C) were employed.

The CAPS is a 30-item scale that employs specific behavioral referents to assess each of the 17 core symptoms of PTSD; 8 items to assess commonly associated symptoms; and 5 global ratings on occupational and social impact, symptom improvement, report validity, and illness severity. The scale also provides for a frequency and intensity rating for each symptom assessed. The developers of the scale assert that it enables a more specific and quantified assessment of PTSD than has been possible with other assessment aids. The CAPS is reported to have good interrater reliability ($r = .92$ to $.99$), internal consistency (Cronbach's Alpha = .73 to .85), and concurrent validity.
with other measures of PTSD ($r = .42$ to $ .84$) (see Blake et al., 1990). It was employed in this study to provide comprehensive pre- and post-intervention assessments of post traumatic sequelae.

The SCL-90-R is a 90-item symptom inventory designed to yield three indices of general psychological functioning and nine indices of specific symptoms. The SCL-90-R has seen extensive clinical and experimental use and has been rated as the best self-report measure of psychological functioning by a National Institute of Mental Health study committee (Waskow & Parloff, 1975). It is reported to have high internal consistency across scales (Coefficient Alpha = .77 to .90), high test-retest reliability across scales ($r = .78$ to .90) and good construct validity (see Derogatis, 1977). It was employed in this study to assess levels of paranoid ideation and psychosis for screening purposes and to provide pre- and post-intervention assessments of depression and anxiety. To this end, an abbreviated version of the instrument was formed by isolating those inventory items that assess those symptoms.

The IES is a 15-item scale designed to assess the cognitive impact of traumatic events along two themes: (1) the intrusiveness of trauma related thoughts, images, and feelings; and (2) the avoidance of trauma related thoughts, feelings and stimuli. It is reported to have high split-half reliability ($r = .86$), internal consistency (Cronbach's Alpha = .78 to .82), test-retest reliability ($r = .87$) and good clinical sensitivity (see Horowitz, Wilner, & Alvarez, 1979). It was used in the present study to assess participant reactions during the week prior to assessment.

The DAST is a 28-item scale designed to yield a quantitative index of problems
identified with drug and alcohol abuse. It reportedly has good diagnostic validity (see Gavin, Ross, & Skinner, 1989) and was used in the present study to provide a measure of the degree of substance abuse in participants reporting drug use.

The device referred to consists of a 20" length of black 1" diameter doweling mounted on a swing-arm assembly that permits the doweling to be fixed at any orientation and at any desired distance from a point of reference. A small red light is fixed at each end of the doweling (19" apart). These lights are wired such that when one is illuminated the other is not. By operating a hand-held switch, the therapist can cause the lights to illuminate alternately at any desired frequency. A third red light is mounted in the center of the doweling. This light can be illuminated and made to flicker through the aforementioned hand-held switch. The device is powered by a 12-volt rechargeable power pack, is fused, and is entirely safe of electrical hazard. This device will herein be referred to as the "light-bar".

The heart rate monitoring device is called a "pulse monitor" and is manufactured by County Technology Inc. of Minnesota. The device employs a small sensor, capable of detecting changes in blood flow, that is clipped to the participant's ear lobe. The sensor is passive and the participant is electrically isolated from the device. Accordingly, there is no potential for shock. The Unit is powered by 4-AA cell batteries.

Procedures

Initial Assessment Session

All persons referred to the project for treatment were given an appointment for
an initial screening / assessment interview. At the beginning of this interview, each person was provided with an informed consent form and asked to read and sign it (see Appendix A). During this interview, participants were asked general questions about their health and psychological well-being, and whether they had a history of cardiovascular disease and/or eye injury or disease. Persons who reported having a history of heart disease and/or high blood pressure (resting pressures of at least 140 mmhg systolic and/or 90 mmhg diastolic) were required to obtain a physician's informed clearance to participate. They were given a description of the nature of the study and the nature and degree of psychological distress that participants might be exposed to, and they were asked to provide that to a physician of their choice (see Appendix B). Information release forms were also provided to participants to give to their physicians (see Appendix C). This requirement was imposed because exposure to trauma related stimuli was expected to cause a significant increase in heart rate which might pose a health risk to persons with cardiovascular disease.

Those who reported a history of eye injury / disease were asked to consult with a physician to obtain medical clearance to participate in the study. Because two of the three treatments require that participants move their eyes very rapidly, individuals with weak retinal attachments or other structural weaknesses could suffer further injury by their participation.

Participants were asked several general questions about their use of drugs and alcohol. If they reported significant alcohol use, the use of any other psychoactive drugs, or having experienced any problems with substance use within the past two years, they
were given the DAST. Those individuals who were assessed as current substance abusers by receiving a score of 7/8 or more on the DAST were excluded from the investigation. The rationale for this is based upon clinical wisdom and practical considerations: it is widely held that active substance abuse interferes with therapeutic efforts. Accordingly, such participants might add unnecessary variability to the data. The score of 7/8 or greater on the DAST was used as this was a compromise between achieving optimum sensitivity (cut-score of 6/7) and optimum specificity (cut-score of 9/10) while maintaining high accuracy (Gavin et al., 1989). Participants were then asked general questions about their traumatic experiences (e.g., when, where, what happened?) and the symptom(s) that were causing them the greatest concern (primary symptoms). Individuals who had not experienced a traumatic event, as defined by criterion "A" of the DSM-III-R criteria for PTSD were screened from the study.

Participants were also asked about any benefits from the trauma, such as compensation payments, that they have realized, that they currently enjoyed, or that they anticipated. Individuals who reported significant current or anticipated benefits that may be terminated upon successful treatment, were excluded from the study.

The CAPS, Impact of Events Scale, and SCL-90-R were then completed. Individuals who did not report sufficient post-traumatic sequelae were screened from the study. This latter criterion for exclusion was as follows: For each of the four symptoms of Criterion B of the CAPS the frequency and intensity scores were multiplied together; if the "frequency by intensity" product of at least one of the four symptoms was not equal or greater than two, the individual was excluded from the study.
Persons who presented with significant levels of psychotic thinking or extreme levels of paranoid thinking were to be excluded from the study. Scores from the SCL-90-R and clinical judgement were both used for this assessment. Persons scoring two standard deviations above the mean for psychiatric outpatients on psychotic thinking were to be excluded from the study unless their presentations seemed incongruent with such elevated scores. In such cases, the person would be carefully screened through targeted questioning of responses to the SCL-90-R which indicated psychotic thinking. Similarly, persons scoring two standard deviations above the mean in paranoid thinking were excluded from the study unless their presentations were incongruent with such scores. Again, targeted questioning was used to determine the nature of the paranoid ideation.

All participants who were not screened from participation were given an appointment for an initial treatment session, no more than one week after the interview.

Group Assignment

Participants were initially assigned to one of the three treatment groups according to gender and the order in which they were interviewed. The first male to be interviewed was assigned to group one (EMD), the second to group 2 (automated eye movements - AEM), and the third to group three (active visual attention - AVA). This sequence of ordered assignment was repeated as necessary. The same method of assignment was used for female participants. This segregation of assignments according to gender functioned to ensure that gender representation in each group was roughly equal.
**Treatment Session One**

Treatment sessions were conducted according to Shapiro's previously described protocol, except for changes imposed by the different visual tasks involved and the assessment of negative belief statements. At the beginning of the first treatment session, participants were attached to the heart rate monitoring equipment and introduced to the 11-point subjective units of distress (SUD) scale previously described and a comparable 11-point validity of cognition (VoC) scale similar to that previously described. This 11-point VoC scale (0 = completely untrue; 10 = completely true) was a departure from Shapiro's use of an eighth point scale, making it similar to the 11-point SUD scale to reduce participant confusion.

Next, the traumatic imagery to be used was identified, and both negative and positive belief statements were constructed. Two initial assessments of participant reactions to the imagery were conducted, one with the negative belief statement, and the other with the positive belief statement. During these, SUD and VoC ratings were obtained. Also, the maximum heart rate exhibited by each participant was recorded and the baseline heart-rate was used to determine the change in heart rate. Accordingly, these assessments produced six datum: (1 & 2) heart rate change scores for both negative and positive assessments, (3 & 4) SUD ratings of traumatic imagery during both negative and positive assessments, and (5 & 6) validity ratings of both negative and positive belief statements. These procedures, and the previously completed CAPS, IES and SCL-90-R constituted the initial assessment.
Participants were then told the following, which was taken from Shapiro (1989a, pp. 204): "What we will be doing is often a physiology check. I need to know from you exactly what is going on with as clear feedback as possible. Sometimes things will change and sometimes they won't. I may ask you if the picture changes -- sometimes it will and sometimes it won't. I'll ask you how you feel from "0" to "10" -- sometimes it will change and sometimes it won't. I may ask you if something else comes up -- sometimes it will and sometimes it won't. There are no "supposed to's" in this process. So just give as accurate feedback as you can as to what is happening, without judging whether it should be happening or not. Just let what happens happen."

Treatment procedures used with the three groups differed only in the form of visual task employed. Participants in the eye movement desensitization (EMD) group were to visually track the therapist's index and middle fingers as they were moved back-and-forth across the line of vision between left and right extremes of the visual field as previously described. Participants in the automated eye movement group (AEM) were to attend to the light-bar and to switch visual attention to whatever light is illuminated as the two red lights were turned on and off alternately. The light-bar was adjusted so that it was horizontal, at participant eye-level, and 12 to 14 inches from the face. The lights were alternately illuminated at a rate of two cycles per second. Participants in the active visual attention group (AVA) were to maintain visual attention on the light mounted in the center of the light-bar. The light-bar was adjusted as above. At this distance, active visual attention on the light was evident through eye convergence caused by focusing on a close object. A loss of visual attention was evident by a loss of
convergence. At the start of the set the center light was illuminated and then flicked off (about one-fifth of a second) periodically to help maintain attention. If a participant's eyes appeared to lose convergence, the light was made to flicker very rapidly to recapture participant attention and, if necessary, the participant was verbally directed to reestablish focus.

Desensitization trials were repeated as necessary, until the reported SUD level dropped to "0", asymptoted at "1", or until it became clear that no progress had been made after 20 treatment sets. In the former case, the installation and final assessment procedures were conducted. In the latter case, a second appointment was made for within a week's time.

If, during the course of treatment, a participant reported no changes in SUDS level over two consecutive treatment trials, s/he was asked if the imagery, belief statement, or any other aspect of the memory had changed from the original. Such changes, when they result in a mismatch between the image, cognition, or affective response, reportedly accompany failures to respond to the treatment with fair reliability (Shapiro, 1991, personal communication). When changes were reported, the new features were employed in the procedure. As soon as the SUDS rating dropped to "0" or asymptoted at "1" with these new features, the original memory features were returned to until the SUDS rating for them dropped to "0" or asymptoted at "1". In some cases it was necessary to temporarily drop the statement rehearsal because the affective response to the imagery or the imagery itself changed and became incongruent with the original statement.
Participants were periodically asked about changes in imagery, belief statements, and affect. If new cognitions were reported, participants were instructed to employ them along with the existing imagery until these new statements did nothing to further improvement in terms of SUDS ratings or in helping to generate a more desired affective response to the imagery.

**Subsequent Treatment Sessions**

When called for, additional treatment sessions were employed to a maximum of six. Except for the initial briefings outlined in the description of the first treatment session, subsequent sessions followed the same protocol outlined above. At the end of the final treatment session, two assessments of reactivity to traumatic imagery were employed as in the first session. Those data represented the post-treatment assessment.

**Follow-Up Session**

After the post-intervention assessment during the final treatment session, an appointment was made for a one to three month follow-up assessment. During the follow-up session, the CAPS, SCL-90-R, and IES were administered and two assessments of reactivity to traumatic imagery were employed as in the first and last sessions. No further treatment was offered during a follow-up session.

**Dependent Variables**

The primary dependent variables in this study were: (1 & 2) heart rate change
scores for both positive and negative assessments, (3 & 4) SUD ratings of traumatic imagery during both positive and negative assessments, (5 & 6) validity ratings of both positive and negative cognition, (7 & 8) SCL-90-R depression and anxiety T-scores, (9 & 10) overall frequency and intensity scores of the CAPS, and (11) IES scores.

**Human Subjects Protection**

**Informed Consent**

All participants were informed about the nature of the treatment, and of the nature of the investigation and their participation in it, prior to participation. Each was provided with an informed consent form and was asked to read it completely before signing it (see Appendix A). Any participant who did not respond to the altered EMD procedures after six sessions, was offered up to six sessions of the original treatment procedure following the follow-up assessment.

**Information Release**

Participants were informed that referring agencies might be contacted for treatment consultation and were asked to sign a release of information form (see Appendix C). Additionally, when referring agencies requested information about participant responses to treatment, participants were asked to sign a release form to permit this.
Treatment Contraindications

As described, a self-report screen was used to assess contraindications for treatment. Individuals who presented with clinically significant symptoms of psychosis or paranoid ideation were excluded from the study. Those participants who reported a history of heart disease or high blood pressure were asked to obtain medical clearance from a physician to participate (see Appendix B). Additionally, participant heart rates were monitored during the brief stressful components of the protocol. Whenever a participant's heart rate exceeded 70% of his/her age determined maximum heart rate (or any limit set by his/her physician), the stressful component was terminated. Those who reported a history of eye muscle or retinal weakness were to be asked to obtain an informed medical clearance to participate.
CHAPTER IV

RESULTS

Preliminary Data Analyses

Participant Characteristics

Participants in the study initially consisted of 8 men and 27 women who were at least 18 years of age. Twenty-three people, consisting of 5 men and 18 women, completed all phases of the study. Unless otherwise indicated, all references to participants will be to these 23 individuals. Of those who did not complete the study, 7 were screened out because they were deemed inappropriate (see below) and 5 began participation but dropped out prior to completing the study.

Traumata included mistreatment by health-care providers following a diagnosis of cancer (N = 1), escaping near-death during Hurricane Andrew (N = 1), motor vehicle accidents (N = 4), combat-related trauma (N = 2), physical assault and injury (N = 1), loosing children to a fire (N = 1), childhood physical and emotional abuse (N = 2), abusive relationships involving repeated rapes (N = 2), sexual assault (N = 4), and incest (N = 5). Eight participants had suffered from traumata other than that which the present treatments were primarily focused on. Six of these were known or suspected incidents of incest and two were a sexual assault. All but two participants, one in the EMD group
and one in the AVA group, met full DSM-III-R (APA, 1987) criteria for PTSD. Seven of these cases could be described as complicated PTSD.

The age of participants ranged from 19 to 56 years. The mean age for men and women was 34.6 and 32.2 years respectively, with a combined mean age of 32.7 years. Education levels ranged from 12 to 22 years. The mean education level was 14.5, 16.0, and 15.7 for male, female, and combined participants respectively. Ten participants were single, 8 were married, and 5 were separated, divorced, or widowed. The median and modal household income level for all participants was between 10 and 20 thousand dollars per year, with 26% earning less than $10,000.00, and 26% earning greater than $40,000.00. The ethnic make-up of the participants included 19 European Americans, two African Americans, one African/Native American, and one African.

Regarding the screening out of participants, two persons scored above criterion on paranoid thinking but it was subsequently assessed that treatment would not be hampered in either case. One person scored above criterion for psychotic ideation, but it was subsequently determined that the score was spuriously elevated. Though no participants were excluded from participation due to psychotic or paranoia ideation, two persons were excluded from participation because they did not appear to be emotionally stable enough to cope with the potential stress of treatment. Four participants were excluded because they either had not experienced a traumatic event, or did not report sufficient post-traumatic sequelae. One participant was screened from the study when his physician recommended against participation due to his history of heart disease. No other participants were excluded from participation in the study.
Group Characteristics

The respective demographics for the EMD, AEM, and AVA groups were as follows: The gender balances were two males and six females, two males and five females, and one male and seven females; mean ages were 30.75, 35.57, and 32.25 years; mean education levels were 16.1, 15.78, and 15.2 years; modal incomes were $40,000.00 or more, $10,000.00 to $20,000.00, and $10,000.00 to $20,000.00; median incomes were $20,000.00 to $30,000.00, $10,000.00 to $20,000.00, and $10,000.00 to $20,000.00; there were three, three, and two married persons; there were three, four, and three single persons; there were two, zero, and three persons who were divorced, separated, or widowed; there were seven, seven, and five European Americans; and there were one, zero, and three persons who were not European Americans. Of those participants who dropped out of the study prematurely, 1 was originally assigned to EMD, 3 to AEM, and 1 to AVA. All participants met DSM-III-R criteria for PTSD except for one participant in the EMD group and one in the AVA group. Seven participants presented with complicated-PTSD, as previously described. Of these, three, one, and three were in the EMD, AEM, and AVA groups respectively.

Measures of Treatment Received

Between two and six sessions were employed in the present study. Participants in group one received an average of 3.9 sessions containing an cumulative average of 43.5 sets of eye movements (EM). Participants in group two received an average of 4.3 sessions containing an average total of 40.3 sets of EM. Participants in group three...
received an average of 5.4 sessions containing an average of total 57.6 sets of visual attention (VA). A one-way analysis of variance revealed that the differences in the mean number of sessions ($F = 3.49$, $p > 0.05$) and sets ($F = 1.78$, $p > 0.05$) were statistically insignificant.

Primary Data Analyses

For heart rate change scores, SUD ratings, and VoC ratings, a two factor group (EMD / AEM / AVA) by assessment phase (pre-treatment / post-treatment / follow-up) ANOVA with repeated measures on phase was conducted, and Scheffe-F multiple comparison tests performed. For SCL-90-R depression and anxiety T-scores, CAPS overall frequency and intensity scores, and IES scores, a two factor group (EMD / AEM / AVA) by phase (pre-treatment / follow-up) ANOVA with repeated measures on phase was conducted and Scheffe-F multiple comparison tests performed as appropriate. Also, the mean and standard error of the mean (SEM) for each dependant variable per phase was calculated. Group means for each dependant variable for each assessment phase were also graphed.

The results of these analyses are presented in Tables 1 to 28 and Figures 1 to 11. Figure 1 depicts mean heart rate change scores associated with negative cognitions. Table 1 presents the results of the repeated measures ANOVA, and Tables 2 and 3 present multiple comparisons test results and the means and SEM for this same data. As indicated, there were no significant differences between groups at any phase of the study on this variable, nor was there a significant interaction between group effects and...
Table 1

Repeated Measures ANOVA for Negative Heart Rate Change Scores

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Group (A)</td>
<td>2</td>
<td>25.353</td>
<td>12.677</td>
<td>1.674</td>
<td>0.2127</td>
</tr>
<tr>
<td>Within Group</td>
<td>20</td>
<td>151.458</td>
<td>7.573</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phase (B)</td>
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<td>1274.812</td>
<td>637.406</td>
<td>88.974</td>
<td>0.0001</td>
</tr>
<tr>
<td>AB</td>
<td>4</td>
<td>16.629</td>
<td>4.157</td>
<td>0.580</td>
<td>0.6786</td>
</tr>
<tr>
<td>B X Within Group</td>
<td>40</td>
<td>286.56</td>
<td>7.164</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 2

Multiple Comparisons Tests for Negative Heart Rate Change Scores

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Mean Difference</th>
<th>Scheffe F-test</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre to Post</td>
<td>8.652</td>
<td>59.186</td>
<td>0.0001</td>
</tr>
<tr>
<td>Pre to F-U</td>
<td>9.522</td>
<td>71.681</td>
<td>0.0001</td>
</tr>
<tr>
<td>Post to F-U</td>
<td>0.870</td>
<td>0.598</td>
<td>-</td>
</tr>
</tbody>
</table>

Table 3

Means and SEM for Negative Heart Rate Change Scores

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre-Tx</th>
<th>Post-Tx</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>EMD</td>
<td>11.125 +/- 1.315</td>
<td>1.25 +/- 0.796</td>
<td>0.375 +/- 0.754</td>
</tr>
<tr>
<td>AEM</td>
<td>8.857 +/- 1.143</td>
<td>0.857 +/- 0.633</td>
<td>-0.714 +/- 0.865</td>
</tr>
<tr>
<td>AVA</td>
<td>8.375 +/- 1.362</td>
<td>0.375 +/- 0.353</td>
<td>0.125 +/- 0.580</td>
</tr>
</tbody>
</table>

assessment phase effects. Averaged for all groups, negative heart rate change scores dropped from 9.48 beats per minute (bpm) at pre-treatment (Pre) to 0.82 bpm at post-
treatment (Post) \( (p < 0.0001) \) but did not change significantly between post-treatment and follow-up (F-U) sessions.

Figure 2 depicts mean SUD ratings associated with negative cognitions. Tables 4 to 6 present corresponding ANOVA results, multiple comparisons test results, and group by phase means respectively. No significant group differences or interactions between group and phase were revealed. Negative SUD scores (scale range = 0 to 10) changed from 7.78 at pre-treatment to 0.78 at post-treatment \( (p < 0.0001) \) but did not change between post-treatment and follow-up assessments.

Table 4

Revised Measures ANOVA for Negative SUD Scores

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Group (A)</td>
<td>2</td>
<td>7.760</td>
<td>3.880</td>
<td>0.494</td>
<td>0.6176</td>
</tr>
<tr>
<td>Within Group</td>
<td>20</td>
<td>157.196</td>
<td>7.860</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phase (B)</td>
<td>2</td>
<td>756.029</td>
<td>378.014</td>
<td>138.087</td>
<td>0.0001</td>
</tr>
<tr>
<td>AB</td>
<td>4</td>
<td>9.804</td>
<td>2.451</td>
<td>0.895</td>
<td>0.4757</td>
</tr>
<tr>
<td>B X Within Group</td>
<td>40</td>
<td>109.500</td>
<td>2.737</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 5

Multiple Comparisons Tests for Negative SUD Scores

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Mean Difference</th>
<th>Scheffe F-test</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre to Post</td>
<td>7.000</td>
<td>65.417</td>
<td>0.0001</td>
</tr>
<tr>
<td>Pre to F-U</td>
<td>7.043</td>
<td>66.232</td>
<td>0.0001</td>
</tr>
<tr>
<td>Post to F-U</td>
<td>0.043</td>
<td>0.003</td>
<td>-</td>
</tr>
</tbody>
</table>
Figure 1. Negative Heart Rate Means.

Figure 2. Negative SUD Score Means.
Means and SEM for Negative SUD Scores

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre-Tx</th>
<th>Post-Tx</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>EMD</td>
<td>8.0 +/-0.463</td>
<td>0.75 +/-0.313</td>
<td>0.875 +/-0.875</td>
</tr>
<tr>
<td>AEM</td>
<td>7.428 +/-0.841</td>
<td>1.714 +/-1.409</td>
<td>1.286 +/-1.128</td>
</tr>
<tr>
<td>AVA</td>
<td>7.875 +/-0.833</td>
<td>0.000 +/-0.000</td>
<td>0.125 +/-0.125</td>
</tr>
</tbody>
</table>

Figure 3 depicts mean validity ratings of negative cognitions. Tables 7 to 9 present corresponding ANOVA results, multiple comparisons test results, and group by phase means. No significant group or interaction effects between group and phase were shown. Negative VoC scores changed from 8.61 at pre-treatment to 0.96 at post-treatment (p < 0.0001) but did not change significantly between post-treatment and follow-up.

Repeated Measures ANOVA for Negative VoC Scores

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Group (A)</td>
<td>2</td>
<td>21.140</td>
<td>10.570</td>
<td>3.320</td>
<td>0.0569</td>
</tr>
<tr>
<td>Within Group</td>
<td>20</td>
<td>63.669</td>
<td>3.183</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phase (B)</td>
<td>2</td>
<td>839.229</td>
<td>419.614</td>
<td>190.433</td>
<td>0.0001</td>
</tr>
<tr>
<td>AB</td>
<td>4</td>
<td>8.149</td>
<td>2.037</td>
<td>0.925</td>
<td>0.4593</td>
</tr>
<tr>
<td>B X Within Group</td>
<td>40</td>
<td>88.139</td>
<td>2.203</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Figure 3. Negative VoC Rating Means.

Figure 4. Positive Heart Rate Means.
Table 8
Multiple Comparisons Tests for Negative VoC Scores

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Mean Difference</th>
<th>Scheffe F-test</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre to Post</td>
<td>7.652</td>
<td>118.421</td>
<td>0.0001</td>
</tr>
<tr>
<td>Pre to F-U</td>
<td>7.261</td>
<td>106.619</td>
<td>0.0001</td>
</tr>
<tr>
<td>Post to F-U</td>
<td>-0.391</td>
<td>0.310</td>
<td>-</td>
</tr>
</tbody>
</table>

Table 9
Means and SEM for Negative VoC Scores

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre-Tx</th>
<th>Post-Tx</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>EMD</td>
<td>8.767 +/- 0.442</td>
<td>1.357 +/- 0.450</td>
<td>2.054 +/- 0.971</td>
</tr>
<tr>
<td>AEM</td>
<td>7.286 +/- 0.837</td>
<td>0.571 +/- 0.297</td>
<td>0.571 +/- 0.369</td>
</tr>
<tr>
<td>AVA</td>
<td>9.5 +/- 0.327</td>
<td>1.0 +/- 0.534</td>
<td>1.375 +/- 0.532</td>
</tr>
</tbody>
</table>

Figure 4 depicts mean heart rate change scores of positive cognitions. Tables 10 to 12, present ANOVA results, multiple comparisons test results and group by phase means. No significant group differences or interaction effects between group and phase were revealed. Positive heart rate change scores changed from 8.65 at pre-treatment to -0.26 at post-treatment (p < 0.0001) but did not change significantly between post-treatment and follow-up assessments.

Figure 5 depicts mean SUD scores associated with positive cognitions. Tables 13 to 15 present corresponding ANOVA results, multiple comparisons test results, and group by phase means. No significant group differences or interactions between group and phase were revealed. Averaged positive SUD scores changed from 6.56 at pre-
treatment to 0.56 at post-treatment ($p < 0.0001$) but did not change significantly between post-treatment and follow-up assessments (SUD scale range = 1 to 10).

Table 10
Repeated Measures ANOVA for Positive Heart Rate Change Scores

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Group (A)</td>
<td>2</td>
<td>11.384</td>
<td>5.692</td>
<td>0.878</td>
<td>0.4309</td>
</tr>
<tr>
<td>Within Group</td>
<td>20</td>
<td>129.601</td>
<td>6.480</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phase (B)</td>
<td>2</td>
<td>1230.116</td>
<td>615.058</td>
<td>78.811</td>
<td>0.0001</td>
</tr>
<tr>
<td>AB</td>
<td>4</td>
<td>49.717</td>
<td>12.429</td>
<td>1.593</td>
<td>0.195</td>
</tr>
<tr>
<td>B X Within Group</td>
<td>40</td>
<td>312.167</td>
<td>7.804</td>
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</tbody>
</table>

Table 11
Multiple Comparisons Tests for Positive Heart Rate Change Scores

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Mean Difference</th>
<th>Scheffe F-test</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre to Post</td>
<td>8.913</td>
<td>59.953</td>
<td>0.0001</td>
</tr>
<tr>
<td>Pre to F-U</td>
<td>9.200</td>
<td>61.128</td>
<td>0.0001</td>
</tr>
<tr>
<td>Post to F-U</td>
<td>0.087</td>
<td>0.006</td>
<td>-</td>
</tr>
</tbody>
</table>

Table 12
Means and SEM for Positive Heart Rate Change Scores

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre-Tx</th>
<th>Post-Tx</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>EMD</td>
<td>8.625 +/- 1.133</td>
<td>0.143 +/- 0.719</td>
<td>0.500 +/- 0.626</td>
</tr>
<tr>
<td>AEM</td>
<td>6.857 +/- 0.508</td>
<td>0.286 +/- 1.149</td>
<td>0.857 +/- 0.633</td>
</tr>
<tr>
<td>AVA</td>
<td>10.250 +/- 1.687</td>
<td>-1.125 +/- 0.718</td>
<td>-0.375 +/- 0.962</td>
</tr>
</tbody>
</table>
Table 13

Repeated Measures ANOVA for Positive SUD Scores

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Group (A)</td>
<td>2</td>
<td>1.526</td>
<td>0.763</td>
<td>0.206</td>
<td>0.8153</td>
</tr>
<tr>
<td>Within Group</td>
<td>20</td>
<td>73.952</td>
<td>3.698</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phase (B)</td>
<td>2</td>
<td>594.899</td>
<td>297.449</td>
<td>124.153</td>
<td>0.0001</td>
</tr>
<tr>
<td>AB</td>
<td>4</td>
<td>14.601</td>
<td>3.650</td>
<td>1.524</td>
<td>0.2137</td>
</tr>
<tr>
<td>B X Within Group</td>
<td>40</td>
<td>95.833</td>
<td>2.396</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 14

Multiple Comparisons Tests for Positive SUD Scores

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Mean Difference</th>
<th>Scheffe F-test</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre to Post</td>
<td>6.000</td>
<td>73.486</td>
<td>0.0001</td>
</tr>
<tr>
<td>Pre to F-U</td>
<td>6.435</td>
<td>84.522</td>
<td>0.0001</td>
</tr>
<tr>
<td>Post to F-U</td>
<td>0.435</td>
<td>0.386</td>
<td>-</td>
</tr>
</tbody>
</table>

Table 15

Means and SEM for Positive SUD Scores

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre-Tx</th>
<th>Post-Tx</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>EMD</td>
<td>6.625 +/- 0.497</td>
<td>0.125 +/- 0.125</td>
<td>0.000 +/- 0.000</td>
</tr>
<tr>
<td>AEM</td>
<td>6.143 +/- 0.769</td>
<td>1.714 +/- 1.409</td>
<td>0.000 +/- 0.000</td>
</tr>
<tr>
<td>AVA</td>
<td>6.875 +/- 0.875</td>
<td>0.000 +/- 0.000</td>
<td>0.375 +/- 0.263</td>
</tr>
</tbody>
</table>

Figure 6 depicts mean validity of positive cognition ratings. Tables 16 to 18 present ANOVA results, multiple comparison test results, and the group by phase means.

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Figure 5. Positive SUD Score Means.

Figure 6. Positive VoC Ratings Means.
Table 16

Repeated Measures ANOVA for Positive VoC Scores

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Group (A)</td>
<td>2</td>
<td>0.112</td>
<td>0.056</td>
<td>0.026</td>
<td>0.9742</td>
</tr>
<tr>
<td>Within Group</td>
<td>20</td>
<td>42.983</td>
<td>2.149</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phase (B)</td>
<td>2</td>
<td>659.721</td>
<td>329.861</td>
<td>187.045</td>
<td>0.0001</td>
</tr>
<tr>
<td>AB</td>
<td>4</td>
<td>4.076</td>
<td>1.019</td>
<td>0.578</td>
<td>0.6804</td>
</tr>
<tr>
<td>B X Within Group</td>
<td>40</td>
<td>70.541</td>
<td>1.764</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 17

Multiple Comparisons Tests for Positive VoC Scores

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Mean Difference</th>
<th>Scheffe F-test</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre to Post</td>
<td>-6.565</td>
<td>138.010</td>
<td>0.0001</td>
</tr>
<tr>
<td>Pre to F-U</td>
<td>-6.609</td>
<td>139.844</td>
<td>0.0001</td>
</tr>
<tr>
<td>Post to F-U</td>
<td>-0.043</td>
<td>0.006</td>
<td>-</td>
</tr>
</tbody>
</table>

Table 18

Means and SEM for Positive VoC Scores

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre-Tx</th>
<th>Post-Tx</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>EMD</td>
<td>2.284 +/- 0.538</td>
<td>8.892 +/- 0.469</td>
<td>9.321 +/- 0.279</td>
</tr>
<tr>
<td>AEM</td>
<td>2.786 +/- 0.575</td>
<td>9.286 +/- 0.286</td>
<td>8.571 +/- 0.719</td>
</tr>
<tr>
<td>AVA</td>
<td>2.500 +/- 0.801</td>
<td>9.000 +/- 0.267</td>
<td>9.250 +/- 0.163</td>
</tr>
</tbody>
</table>

No significant group differences or interaction effects between group and phase were revealed. Positive VoC scores changed from 2.48 at pre-treatment to 9.04 at post-treatment (p < 0.0001) but did not change significantly between post-treatment and
follow-up

Figure 7 depicts mean anxiety T-scores on the SCL-90-R. Tables 19 and 20 present corresponding ANOVA results and group by phase means respectively. As there were no significant group or interaction effects, multiple comparison tests were not required. Averaged anxiety T-scores changed from 53.82 at pre-treatment to 42.26 at follow-up ($p < 0.0001$).

Table 19

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Group (A)</td>
<td>2</td>
<td>70.125</td>
<td>35.062</td>
<td>0.415</td>
<td>0.6658</td>
</tr>
<tr>
<td>Within Group</td>
<td>20</td>
<td>1688.875</td>
<td>84.444</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phase (B)</td>
<td>1</td>
<td>1561.391</td>
<td>1561.391</td>
<td>49.044</td>
<td>0.0001</td>
</tr>
<tr>
<td>AB</td>
<td>2</td>
<td>42.877</td>
<td>21.438</td>
<td>0.673</td>
<td>0.5212</td>
</tr>
<tr>
<td>B X Within Group</td>
<td>20</td>
<td>636.732</td>
<td>31.837</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 20

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre-Tx</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>EMD</td>
<td>54.375 +/- 3.520</td>
<td>43.250 +/- 2.042</td>
</tr>
<tr>
<td>AEM</td>
<td>53.714 +/- 3.871</td>
<td>44.286 +/- 2.495</td>
</tr>
<tr>
<td>AVA</td>
<td>53.375 +/- 1.591</td>
<td>39.250 +/- 2.555</td>
</tr>
</tbody>
</table>

Figure 8 depicts mean depression T-scores on the SCL-90-R. Tables 21 and 22 present corresponding ANOVA results and group by phase means. As there were no
significant group or interaction effects, multiple comparison tests were not required.

Averaged T-scores changed from 50.77 at pre-treatment to 41.87 at follow-up ($p < 0.0001$).

Table 21

Repeated Measures ANOVA for SCL-90-R Depression T-Scores

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Group (A)</td>
<td>2</td>
<td>96.512</td>
<td>48.256</td>
<td>0.459</td>
<td>0.6385</td>
</tr>
<tr>
<td>Within Group</td>
<td>20</td>
<td>2103.357</td>
<td>105.168</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phase (B)</td>
<td>1</td>
<td>913.587</td>
<td>913.587</td>
<td>54.024</td>
<td>0.0001</td>
</tr>
<tr>
<td>AB</td>
<td>2</td>
<td>84.699</td>
<td>42.349</td>
<td>2.504</td>
<td>0.107</td>
</tr>
<tr>
<td>B X Within Group</td>
<td>20</td>
<td>338.214</td>
<td>16.911</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 22

Means and SEM for SCL-90-R Depression T-Scores

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre-Tx</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>EMD</td>
<td>49.000 +/- 3.708</td>
<td>43.250 +/- 2.426</td>
</tr>
<tr>
<td>AEM</td>
<td>52.571 +/- 2.767</td>
<td>43.857 +/- 4.038</td>
</tr>
<tr>
<td>AVA</td>
<td>50.750 +/- 1.810</td>
<td>38.500 +/- 1.647</td>
</tr>
</tbody>
</table>

Figure 9 depicts mean overall CAP frequency scores. Tables 23 and 24 present corresponding ANOVA results and group by phase means. As there were no significant group or interaction effects, multiple comparison tests were not required. Averaged overall CAPS frequency scores changed from 1.83 at pre-treatment to 0.64 at follow-up ($p < 0.0001$).
Figure 7. Anxiety T-Score Means.

Figure 8. Depression T-Score Means.
Table 23

Repeated Measures ANOVA for Total CAPS Frequency Scores

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Group (A)</td>
<td>2</td>
<td>0.466</td>
<td>0.233</td>
<td>0.772</td>
<td>0.4754</td>
</tr>
<tr>
<td>Within Group</td>
<td>20</td>
<td>6.039</td>
<td>0.302</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phase (B)</td>
<td>1</td>
<td>16.178</td>
<td>16.178</td>
<td>119.184</td>
<td>0.0001</td>
</tr>
<tr>
<td>AB</td>
<td>2</td>
<td>0.104</td>
<td>0.052</td>
<td>0.383</td>
<td>0.6868</td>
</tr>
<tr>
<td>B X Within Group</td>
<td>20</td>
<td>2.715</td>
<td>0.132</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 24

Means and SEM for Total CAPS Frequency Scores

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre-Tx</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>EMD</td>
<td>1.682 +/- 0.207</td>
<td>0.536 +/- 0.128</td>
</tr>
<tr>
<td>AEM</td>
<td>2.021 +/- 0.113</td>
<td>0.694 +/- 0.211</td>
</tr>
<tr>
<td>AVA</td>
<td>1.798 +/- 0.172</td>
<td>0.696 +/- 0.153</td>
</tr>
</tbody>
</table>

Figure 10 depicts mean overall CAPS intensity scores. Tables 25 and 26 present corresponding ANOVA results and the group by phase means. As there were no group
Figure 9. CAPS Overall Frequency Means.

Figure 10. CAPS Overall Intensity Means.
Table 26
Means and SEM for Total CAPS Intensity Scores

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre-Tx</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>EMD</td>
<td>1.60 +/- 0.194</td>
<td>0.485 +/- 0.095</td>
</tr>
<tr>
<td>AEM</td>
<td>1.68 +/- 0.216</td>
<td>0.677 +/- 0.190</td>
</tr>
<tr>
<td>AVA</td>
<td>1.68 +/- 0.176</td>
<td>0.585 +/- 0.100</td>
</tr>
</tbody>
</table>

or interaction effects, multiple comparison tests were not required. Averaged CAPS intensity scores changed from 1.65 at pre-treatment to 0.58 at follow-up (p < 0.0001).

Figure 11 depicts mean IES scores. Tables 27 and 28 present the ANOVA results

Table 27
Repeated Measures ANOVA for IES Scores

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Group (A)</td>
<td>2</td>
<td>34.497</td>
<td>17.249</td>
<td>0.269</td>
<td>0.7669</td>
</tr>
<tr>
<td>Within Group</td>
<td>20</td>
<td>1282.938</td>
<td>64.147</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phase (B)</td>
<td>1</td>
<td>3548.174</td>
<td>3548.174</td>
<td>75.806</td>
<td>0.0001</td>
</tr>
<tr>
<td>AB</td>
<td>2</td>
<td>110.710</td>
<td>55.355</td>
<td>1.183</td>
<td>0.327</td>
</tr>
<tr>
<td>B X Within Group</td>
<td>20</td>
<td>936.116</td>
<td>46.806</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 28
Means and SEM for IES Scores

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre-Tx</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>EMD</td>
<td>29.500 +/- 2.352</td>
<td>13.125 +/- 2.648</td>
</tr>
<tr>
<td>AEM</td>
<td>31.571 +/- 1.645</td>
<td>9.428 +/- 3.657</td>
</tr>
<tr>
<td>AVA</td>
<td>26.625 +/- 2.178</td>
<td>11.875 +/- 3.204</td>
</tr>
</tbody>
</table>

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for scores on the IES and the group by phase means. As there were no significant group or interaction effects, multiple comparison tests were not required. The IES scores changed from 29.23 at pre-treatment to 11.48 at follow-up (p < 0.0001).

Although no formal analysis of charges in DSM-III-R diagnoses was made, the numbers of participants meeting full criteria are reported here. All but two participants met criteria at pre-treatment, one in each of groups one and three. At follow-up, only five met criteria, one in each of groups one and two, three in group three.

Figure 11. Impact of Events Score Means.
CHAPTER V

DISCUSSION

Discussion of Present Results

The present study attempted in part to replicate Shapiro's (e.g., 1989a, 1989b) study of the effects of EMD on post-traumatic sequelae. To this end, it has done so. The nature and size of the treatment effects in the present study are comparable with those reported by Shapiro. For example, Shapiro (1989a) reported that SUD (negative) ratings dropped from an average of 7.45 at pre-treatment to an average of 0.13 at post-treatment, and that these ratings only increased insignificantly by one and three month follow-ups. In the present study, negative SUD ratings within the EMD group dropped from an average of 8.0 to 0.75 with only an insignificant increase (0.875) at follow-up. The size of these effects is greater than the 7.4 to 3.4 drop reported by Boudewyns and colleagues (Boudewyns et al., 1993) after two session of EMD. This will be discussed further below.

The changes in preferred or positive VoC ratings reported here are also consistent with those reported by Shapiro (1989a) but of a greater magnitude: Shapiro reported an increase from 3.95 at pre-treatment to 6.75 at post-treatment with maintenance at follow-up. The present study found an increase in VoC of 2.3 to 8.9 with a further increase to 9.3 at follow-up. These findings suggest that desired changes in self-reported cognitions
do accompany treatment with EMD, suggesting that greater cognitive processing of traumatic memories.

Additionally, the present investigation met several deficiencies left by most previous studies of EMD by (a) including an assessment of SUD ratings associated with initial or negative cognitions at pre, post, and follow-up assessments; (b) assessing the effects of treatment on physiological reactivity to trauma-related imagery by formally monitoring HR changes during pre, post, and follow-up assessments; and (c) employing standardized instruments in assessing pre-treatment and follow-up status on key PTSD measures. Each of these indicated significant therapeutic changes accompanying treatment, giving greater credence to the efficacy of the procedure.

Changes in initial SUDS ratings were expected to drop as they did with successful treatment, so the present study provided an empirical confirmation of this. Participants often reported a perceptual split in their validity ratings of the initial cognition. Though it might seem untrue to some extent at an intellectual level, it usually seemed very true at an emotional level. For example, one woman who was a survivor of early childhood incest rated the intellectual validity of her initial cognition, "I'm bad, I deserve this," very low (about 2) but rated her "gut" feeling at a 10. In recording SUD ratings, the emotional or gut-level estimates were used because these seemed to better reflect unresolved post-traumatic issues and the irrational nature their post-traumatic cognitions. It was observed that in persons exhibiting these perceptual splits, successful treatment was accompanied by a congruence between intellectual and emotional ratings.

Changes in heart rate change scores also accompanied successful treatment in the
expected direction. Although Shapiro (1989a) used a different method of calculating changes in HR reactivity to trauma-related imagery, the direction and magnitude of the present changes were congruent with those she observed; Shapiro recorded a 13 bpm drop in mean HR between pre- and post-treatment assessments, while a +11.125 bpm to +1.25 bpm drop in HR change scores was presently observed. Additionally, this lower HR reactivity was maintained at follow-up with an average mean +0.375 bpm. Although HR is not considered a reliable indicator of physiological reactivity, the present findings suggest lowered sympathetic arousal in response to traumatic imagery. This finding differs, however, from that reported by Boudewyns and colleagues (Boudewyns et al., 1993). They found that two sessions of EMD had no effect on HR, electromyographic responses, skin conductance, or peripheral temperature observed during exposure to a taped trauma exposure script. This difference may be due to the use of the tape which provides a consistent traumatic stimuli across assessments. This issue will be discussed in the final section of this chapter. The difference might also be due to Boudewyns and colleagues' use of only two EMD sessions when more might have been required to effect a critical level of treatment.

The present use of established psychological tests has corrected a major weakness in most studies of EMD to date. These instruments confirmed the nature and severity of participant symptoms at pre-treatment and tracked changes in these across assessments. The results confirm the clinical efficacy of EMD in ameliorating the post-traumatic symptoms measured. Levels of depression and phobic-anxiety, as measured by the SCL-90-R, dropped significantly. Though the average T-scores at pre-treatment
for depression and anxiety were 49 and 54 respectively, indicating average levels for psychiatric outpatients, scores for both depression and anxiety were 43, indicating below average levels. Significant improvements were also measured in overall PTSD symptom levels as measured by the CAPS, and in intrusive and avoidant symptoms as measured by the IES. This finding differs from that reported by Boudewyns and colleagues (Boudewyns et al., 1993). They found that two sessions of EMD had no effect on scores on the Mississippi Scale, the CAPS, or the IES. As speculated above and discussed further below, this difference may be due to Boudewyns and colleagues' use of only two sessions, which may have been insufficient to reach a critical level of resolution for their participants.

One major difference with Shapiro's (1989a) results was the greater number of sessions and sets required to produce the observed effects. This suggests that the present use of EMD was not as efficient in reducing sequelae as indicated in Shapiro's report. This is consistent with Boudewyns and colleagues' (Boudewyns et al., 1993) finding that SUD ratings dropped less after two sessions of EMD than reported by Shapiro (1989a) after one. Also, their failure to observe changes in physiological and psychological measures is inconsistent with the present findings. It is plausible that if Boudewyns and colleagues had employed more sessions as in the present study, treatment effects of a comparable magnitude would have been observed.

A number of possible explanations for this difference present themselves. First, it seems reasonable to suspect that the number of sets of EM are more important than the number of sessions over which they are executed. Shapiro does not formally report data
on the number of EM sets provided to her participants. Although she states that complete desensitization requires 3 to 12 sets (Shapiro, 1989a), this figure does not appear to include sets required for installation whereas the number of sets reported here does. Also, a significantly greater number of sets may be required with some patients and the participants in the present study, as unlikely as it may seem, may simply have required more sets than Shapiro's.

Some anecdotal evidence from the present study supports the above possibility. The majority of participants in this study seemed to be taken by surprise at the level of their affective reactivity during the initial session and opted to terminate the day's work after a small number of sets. Several clients seemed to only be able to tolerate a few sets in a given session. Additionally, as previously noted, seven of the present participants could be described as suffering from complicated PTSD, and an additional eight had experienced traumata other than those that were the primary focus of treatment. Six of these eight were known or suspected incidents of incest and two were sexual assaults. Accordingly, 65% of the present participant pool presented with complications that may have demanded more sessions and sets.

Another explanation is that the general therapeutic skill level, and in particular the level of skill in using EMD, of the present investigator may be less than that of Shapiro. Although the present investigator was trained in the use of EMD by Shapiro, a lower skill / experience level may have retarded therapeutic progress through (a) failure to aid participants in identifying optimal positive and negative cognitive statements; (b) reacting to slowed or stalled progress less quickly, resulting in more
unproductive sets; or (c) generally inefficient guidance of participants through the EMD process. Also, the present investigator was very wary of pressing participants to confront their traumatic memories and may have erred too much on the side of caution.

The present study also attempted a partial dismantling procedure on EMD, in effect to determine the importance of eye movements (EM) to the efficacy of the procedure. The results suggest that EM are not an essential component of the intervention: No significant differences were found between the treatment effects of any of the three interventions employed. However, there is evidence to suggest that the two interventions which employed EM were more efficient in bringing about these changes. As reported above, there were differences in the mean number of sessions and sets received by the members of the three groups. Although the differences are insignificant, the observed trend is worthy of consideration, particularly since groups with EM tended to require fewer sessions and sets, than the VA group.

There are several plausible explanations for this trend. First, EM may simply be a better task than VA for fixing an individual's perceptual attention. Tracking a moving object or the alternating illumination of lights likely requires greater attention than focusing on a fixed point. Also, a loss of focus or attention to the visual task is more readily apparent to the therapist and more quickly reacted to with EM than with VA. Therefore, to the extent that perceptual attention is important to the efficacy of EMD, the use of EM with groups one and two may have promoted a more effective nexus with the therapeutic process. Alternatively, EM may play some other role in the efficacy of EMD, though it is apparent from the present findings that it is neither critical nor substantial.
A post-hoc inspection was made of the number of participants who met DSM-III-R (APA, 1987) criteria for PTSD over the course of the study. This revealed that seven met criteria at pre-treatment for each of the three groups, but at follow-up only one met criteria in groups one and two and three met criteria in group three. Though this is suggestive that the two interventions involving eye-movements were more clinically effective, it is felt that the other dependant measures formally employed are more sensitive indicators of therapeutic change.

The Efficacy of EMD

Given that EMD does appear to be an effective treatment for the post-traumatic sequelae assessed and that EM do not appear to play a necessary role, it remains for the efficacy of the procedure to be explained. Providing the basis for a definitive explanation of the mechanisms underlying EMD’s effects is beyond the intent here. However, several possibilities suggest themselves from the literature and from reports by participants in the present study.

Drawing from cognitive processing models might provide an initial basis for speculation. As previously discussed, Lang (1977, cited by Foa et al., 1989), asserted that a critical number of information units might need to be activated to effect reprocessing of traumatic memories. Foa and colleagues (Foa et al., 1989) suggested that PTSD might be particularly resistant to effective treatment, in part, because the complexity and fragmentation typical of traumatic memories makes them difficult to activate sufficiently. One could reasonably surmise that the mindful control over recall
that typifies traditional verbal therapies might retard access to such fragmented memories. It was apparent in working with EMD during the present study that it is a very fluid procedure, enabling the spontaneous emergence of verbal, affective, and kinesthetic memories. Since patients are instructed to attend to whatever memory fragments arise during a given set without attempting to supervise the process, cognitive control over recall may be reduced enough to enable access to fragmented memories. Related to this, is the common experience of patients undergoing EMD that they recall memory fragments in varying sense modalities (e.g., a smell, a sound, or a kinesthetic sensation). The fluidity of EMD may permit easier access to multiple memory modalities than traditional verbal therapies. It is possible, then, that EMD's fluidity might function to rapidly access enough multiple memory units to reach the critical number proposed by Lang. It may also permit effective Pavlovian desensitization through exposure to a multiple trauma-related stimuli.

It was observed that a number of participants with extensive experience with traditional therapies were inclined to verbally process their experiences between sets. In each case, their verbal reflections initially predominated and therapeutic progress seemed slow. When these participants were persuaded to engage in a rapid succession of sets with minimal verbal exchange between, therapeutic progress appeared to accelerate rapidly. It occurred to the present investigator that some of these patients were simply taught to engage in excessive verbal exchanges through their experiences with traditional therapy; however, others appeared to actually avoid aspects of the traumatic memory, especially affective components, through an excessive cognitive focus. In essence, they
appeared to avoid confronting the traumatic memory in its entirety by talking about it. It follows then, that by minimizing verbal processing, EMD may prevent a form of avoidance that traditional therapies may inadvertently foster. This may account for some of the efficacy of EMD with some patients, and possibly for the relative inefficiency of traditional therapies. It is also feasible that focusing on a simple belief statement inhibits more complex covert verbal chains during sets thereby reducing verbal or cognitive control over the emergence of memory fragments.

Related to the above, several participants in the current study claimed that not having to explain or talk about their "recall set" experiences in detail was beneficial. They asserted that this enabled them to reexperience their memories unhindered by verbal interchange. Several also asserted that not having to talk about their recollections in detail, as they had in previous therapy, made the memories easier to confront: It provided them with an assurance of privacy if and when it was desired.

The possible role of the visual task in EMD and the present variants should also be weighed. Shapiro (1991) has proposed that EMD may owe part of its efficacy to the distraction provided by the eye-movement task. One participant, who had an advanced degree in clinical psychology and a penchant to extensively analyze the therapeutic process between sessions, proposed this as well. She submitted that the visual task (group two) distracted her enough from the traumatic memories as they were unfolding that fragments would emerge into her consciousness before she had a chance to suppress them. The effect for this participant was to loose the tight control that she normally maintained over the intrusion of trauma-related memories. It seems plausible that, for
some patients, the visual attention task is a sufficient distractor to reduce memory avoidance or suppression and permits greater exposure and processing.

Another possibility is that the object attended to in the visual task of EMD or other features of the EMD procedure may come to function as safety signals. According to the safety signal hypothesis, "the omission of anticipated punishment is a reinforcing event...(and it) confers conditioned, or secondary, rewarding properties on stimuli (safety signals) which occur in association with it" (Gray, 1971, p. 170). Just as agoraphobics may expose themselves to otherwise feared and avoided situations in the presence of a trusted person or talisman (safety signal), it is proposed here that patients may expose themselves to greater content of traumatic memories in the presence of some features of the EMD milieu. According to this proposal, patients might anticipate overwhelming anxiety upon initially engaging a traumatic memory. The brief exposure period and the thought-stopping technique used in EMD may keep anxiety to within tolerable limits. Relatively speaking, the anticipated level of distress may be thought to be omitted thereby endowing salient features of the EMD process with safety signal properties. Such would likely increase the probability of reengaging trauma-related memories sufficiently to enhance processing.

In the present study, a number of participants related that they found it empowering to intentionally engage their painful memories after laboring since their traumata to avoid them. This is congruent with Shapiro's (1991) proposal that EMD may enhance a patient's sense of mastery over troublesome internal stimuli through repeated self engagement and disengagement of traumatic imagery. This phenomenon may
simply be due to a cognitive reappraisal of the threat value of traumatic memories, but it may also be due to the acquisition of relevant safety signals. The two processes would not be mutually exclusive.

The above speculation is by no means exhaustive. It is argued here, however, that extensive speculation may be premature until we have a better understanding of what features of EMD are critical to its efficacy. Some speculation may be fruitful, however, to the extent that it generates hypotheses which can be experimentally tested. Some possibilities for future endeavors are discussed at the end of this section.

**Study Limitations**

The current study suffers a number of limitations that reduce confidence in its internal and external validities. To begin with, clients were not required to cease ongoing treatment with other therapists during their participation in this study. Of the 23 participants, six were in insight-oriented treatment for post-traumatic issues during their participation, and one was in behavioral treatment for an eating disorder. Of these, two, two, and three participants were in group one, two, and three respectively. Because of this, the degree to which the observed changes in symptoms and reactivity to trauma-related imagery in these participants can be attributed to the present interventions is unclear. An informal inspection of the responses of these participants to the interventions suggests, however, that they did not differ appreciably from the other members of their respective groups. Also, as previously discussed, post-traumatic sequelae tend to be long-lasting and resistant to rapid therapeutic amelioration. It is generally thought that
the treatment of PTSD is a long-term venture, especially using traditional verbal therapies alone. Accordingly, the rapidity and significance of the shifts observed in these participants suggests that they are largely due to the present interventions. Feedback from five of these participants and three of their primary therapists indicates that the present interventions were felt to have an immediate and significant impact on sequelae. Two participants independently described the procedures as "therapy in fast-forward" and two others stated that they experienced greater levels of cognitive insight and emotional catharsis during sessions of the present interventions than they had in many sessions of previous therapy. Each of these clients had extensive histories of therapy. Accordingly, the ongoing therapy of these participants is not considered a serious threat to the overall internal validity of the study.

Another concern about the present study is the heterogeneity of the participants in terms of the nature of the traumata, the age of insult, and concurrent diagnoses. Though the heterogeneity of the participants might argue for generality of the present findings to a broad client population, the low number of participants in each class prohibits a determination of differential efficacy. Similarly, since no formal assessment of concurrent diagnoses was made in the present study, the differential efficacy of the present interventions for individuals with varying concurrent problems cannot be determined. On the other hand, the seven cases of complicated PTSD added complexity to the treatment process and it seems reasonable to assume that the overall treatment effects would have been greater had these complexities not existed. Though the heterogeneity of the participants poses questions that cannot be answered from the
study results, it is not thought to be a serious threat to external validity.

Another problem with the current study is the absence of a no-treatment control group. Accordingly, the observed changes in this study cannot be compared with the simple passage of time. Similarly, because no attention control group was employed, the present changes cannot be contrasted with those associated with simple mental health-care attention. Neither of these is considered a major problem, however, given the severity of the presenting symptoms, the enduring nature of such symptoms, and the size of rapidity of the changes observed. It seems highly unlikely that such changes would occur in either of the above mentioned control groups had they been used.

The use of a single therapist may have resulted in a biasing effect which could reduce the internal validity of the study and the generality of the findings across therapists. It is unclear whether such a biasing could have significantly affected the present findings, especially on the standardized tests employed. It seems unlikely that such an effect would have acted upon the assessment procedures since they were fairly simple and standardized within the study. However, it is feasible that therapist bias might have affected the efficacy of one or more of the interventions. Accordingly, if multiple therapists had been employed, significant differences between intervention efficacies may have been found. This seems unlikely, however. All three interventions were shown to reduce post-traumatic sequelae significantly with no meaningful differences between them. Since it is assumed that therapists attempt to maximize the efficacy of their interventions, the greatest threat to the present findings would be a bias that would reduce the efficacy of the standard EMD treatment, thereby inflating the relative efficacy
of the alternative interventions. Since the size and nature of the treatment effects for the
EMD group, for all groups, were congruent with those reported elsewhere, it seems
unlikely that the efficacy of the EMD procedure used in this study was impaired.

Another possible weakness of the present study lies in the nature of the
assessment for reactivity to trauma-related imagery. Boudewyns and colleagues
(Boudewyns et al., 1993) employed a taped script of the traumatic event in assessing the
reactivity of participants to trauma-related imagery. They speculated that such a script
might better enable an assessment of reactivity to what they refer to as the "original
memory" (Boudewyns et al., 1993, p. 32) of the event as opposed to treatment-affected
memory. They conjectured that if, as Shapiro (1989a, 1989b) speculates, the EMD
procedure affects the cognitive appraisals and attributions of the traumatic memory,
assessments of post-treatment reactivity may only reflect reactivity to an altered
memory, not to the original memory. Accordingly, the post-treatment and follow-up
assessments of the present study may not reflect true reactivity to the original memory.
The meaning of this to the present results, however, is unclear. It seems reasonable to
assume that what is most important, from a therapeutic perspective, is the nature of and
reactivity to an individual's recollection of a traumatic event. It could be argued that
whether such recollections are altered is irrelevant, and may in fact be deemed a primary
goal of treatment. Accordingly, the assessments used in the present study would be most
appropriate. On the other hand, if treated individuals retain aversive reactivity to original
traumatic stimuli, then subsequent events which resemble the original trauma along key
stimulus dimensions may produce aversive reactions which would be of clinical concern.
Further studies are needed, however, to determine how much of a concern this should be.

Finally, the follow-up assessment was conducted one- to three-months from the last treatment session. A follow-up period of six-months to a year would generate greater confidence that the observed therapeutic changes maintain themselves after treatment.

Future Directions

Although the present findings suggest that eye movements may not be a necessary component of EMD, replication of these results by other researchers is important to confirm this. To maximize comparability of treatment groups, it is suggested that they be fairly homogeneous in terms of precipitating traumata, severity and nature of sequelae, and of concurrent diagnoses. Such studies would also permit comparisons of the efficacy of EMD (and alternatives) along these same dimensions. It is also suggested that the number of treatment sessions and sets received by participants be examined as quantitative measures of treatments received. Correlations between such measures and treatment effects might provide a sensitive indicator of relative treatment efficacy.

The use of visual attention on a fixed object in the present study was selected to enable the role of eye movements in EMD to be isolated. As discussed above, the trend toward greater efficiency of the interventions using EM warrants further investigation. Whether EM simply function as an effective means of holding perceptual attention or play another role might be investigated. To this end, future investigations might employ
a treatment condition in which participants close their eyes during sets and move them back and forth as in EMD to the beat of a metronome. Such would separate the effects of eye movements from active visual attention. Also, Sanderson and Carpenter's (1992) study comparing image confrontation with EMD requires replication using participants with PTSD. The use of timed eye movements with eyes closed would make a good comparison treatment with image confrontation.

It was observed during the present study that participants often experienced shifts in emotional reactivity to traumatic imagery. For example, many participants who initially experienced anxiety or fear during imagery, suddenly began to feel anger instead and this shift was usually accompanied by a shift in the cognitive appraisal of the nature or meaning of the event. Usually these feelings of anger were quite intense (SUD > 7) but abated with subsequent sets. A number of participants experienced three or more such shifts, usually accompanied by enhanced insight and / or cognitive shifts. Though a detailed analysis of these shifts is beyond the intended scope of the present study, such may be a fruitful goal for future investigations. An analysis of the relationship between affective and cognitive shifts during EMD or similar interventions might help illuminate the nature of the therapeutic process involved and lead to a better understanding of the nature of traumatic sequelae and their treatment. For example, if it were observed that cognitive shifts typically precede affective shifts, this would suggest a primacy of cognitive processes in the maintenance and treatment of post-traumatic sequelae and vice versa. The relationship between such apparent primacy and the nature of the traumatic event or the unresolved traumatic issues may be a fruitful
study as well.

If active visual attention does prove to be a necessary component of EMD, it would be interesting to assess active attention in other sense modes. It would be of advantage to know whether a variant of EMD using touch or auditory stimuli as an attention focus was efficacious. If so, such a variant could be used with the visually impaired. Additionally, clients in the present study reported having the strongest or most provocative traumatic memories in differing sense modes. For some it was visual, for some it was auditory, and for others it was kinesthetic. Some recollected stimuli, a smell for example, seemed to function as triggers to a rapid flood of trauma-related memories for some clients. The possible use of attention foci in sense modalities which are similar to those for which memories are strongest is an intriguing possibility.

Finally, the use of some variant of EMD with trauma-related stimuli similar to those employed in flooding remains unexplored. The combined provocativeness of the flooding stimuli and the reactive fluidity of the EMD procedure might combine to effect an even more rapid and effective intervention, particularly for individuals with poor imaginative facilities for whom covert stimuli may prove ineffective.
Appendix A

Informed Consent Form
INFORMED CONSENT

When people experience traumatic events, problems may later develop for them. We invite you to participate in an evaluation of a procedure that may have a positive impact on some of those problems. Your participation will involve an initial interview (about 1 hour long), 1 to 3 treatment sessions of 50 to 90 minutes length, and brief interviews 1 and 3 months later.

During the initial interview, we will ask you general questions about traumatic events that you have experienced and about some problems that may have developed for you as a result. We will also ask you a number of specific questions about particular experiences that you may or may not have had during the preceding week. During treatment, we will ask you to identify a visual image and verbal statements of the traumatic event(s) that are of greatest concern to you. You do not have to provide detailed descriptions of these to the investigator if you do not wish. We will ask you several times to call-up that image and statements and perform a simple visual task for about 10 seconds at a time. Your heart rate may increase during these periods and we will monitor it continuously. We will ask you some questions about the imagery, statements. During the two brief follow-up interviews we will ask you the same series of specific questions that will be asked during the initial interview.

All information gathered during your participation is considered confidential and will not be released to others without your written permission. All personal identifying information will be removed for use in the preparation of scientific reports.

Your participation is needed and would be highly valued. If you agree to participate, it is our hope that you will see the process through to completion; however, you may choose not to complete a task requested of you, and you may choose to end your participation any time you wish. You may direct any questions or concerns that you have about the procedures and/or your participation to the principal investigator, George S. Renfrey (349-3897), or to Dr. C. Richard Tsegaye-Spates of the Department of Psychology at Western Michigan University (387-4496).

If you wish to participate in this study, please answer the two questions below and initial them. By signing your name below, you are just acknowledging that you have read and understood this form: It is not a binding agreement to participate in the study. You will receive a copy of this form for your records.

1. Do you have a history of heart disease or high blood pressure?
   YES ____ NO ____ Initials: ______

2. Do you have a history of eye injury, disease, or weakness?
   YES ____ NO ____ Initials: ______

_________________________________________ ________________________ Participant
Investigator Date
Appendix B

Medical Clearance Form
MEDICAL CLEARANCE

To ____________________________:
(physician's name)

_________________________ has expressed a desire to participate in a study currently underway at Western Michigan University. While participating s/he may be asked to visually track a rapidly moving object. Though these movements are of short duration (about 10 seconds at a time), and are within the normal range of motion and speed, they may cause fatigue and otherwise act upon any existing underlying weaknesses in eye musculature and retinal structure. Additionally, s/he will be asked to briefly (about 10 seconds at a time) recall certain past experiences that were traumatic, which may result in sympathetic reactivity and hence heart rate increases. During all procedures, heart rate will be continuously monitored and if it ever exceeds 70% of the subject's age determined maximum, the task will be terminated and a relaxation exercise will be used. All such periods of arousal will be brief.

Each of the procedures of concern in the present investigation have been used on hundreds of persons, and they posed no hazard to anyone within normal limits of cardiovascular and eye/retinal health.

If, after examining ______________________ you believe that s/he is within normal limits of cardiovascular health and has no current condition of the eye that would contraindicate repeated and rapid eye-movements, please indicate this on the form below. If you have any questions about the nature of the task that your patient will be asked to complete, please contact either George S. Renfrey (349-3897) or Dr. Richard Spates (387-4496).

I, ____________________________ have examined ______________________
on this date ____________________, and believe that the procedures involved in the above research would {Please Check One} [POSE_] [NOT POSE_] a significant health risk to him/her. This judgement is made upon the basis of the description of those procedures described herein, and does not imply an intimate knowledge of the same.

______________________________
Physicians Signature

__________________________
Date

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Appendix C

Release of Information Form
Release of Information

The purpose of this form is to establish permission for Party A to provide specified information about the individual granting permission (the patient) to Party B. Because of need and desire to guard patient confidentiality, the completion of this information release is necessary for any communication about the condition and progress of the patient to take place between the two specified parties. Only information relevant to the treatment of the patient will be communicated and it will only be given in the best interests and welfare of the patient.

By signing below, the patient is not waiving any rights to confidentiality, but is merely granting permission for a very limited sharing of information about his/her condition and treatment responses. Party B is obligated by law to respect the confidentiality of the information provided. The patient may withdraw this permission at any time by informing Party A of his/her decision to do so.

Under no circumstances will a patient be coerced into signing this form. If you, the patient, feel pressured to complete this form or are otherwise unsure whether you should, we ask you not to complete it at this time.

I, __________________________, hereby grant Party A __________________________ permission to provide Party B __________________________ with information about my current state of health and welfare. I release only information that pertains to my receiving treatment for post-traumatic stress reactions at Western Michigan University’s Psychology Department Clinic. I understand that all information about me in this context is considered confidential and that I may withdraw this permission at any time.

Patient: __________________________ Date: ___/___/___

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Appendix D

Western Michigan University Human Subjects Institutional Review Board Research Protocol Clearance
Date: May 21, 1991
To: George Renfrey
From: Mary Anne Bunda, Chair
Re: HSIRB Project Number 91-05-08

This letter will serve as confirmation that your research protocol, "The Efficacy of Eye-Movement Desensitization: A Partial Dismantling Procedure," has been approved after full review by the HSIRB. The conditions and duration of this approval are specified in the Policies of Western Michigan University. You may now begin to implement the research as described in the approval application.

You must seek reapproval for any change in this design. You must also seek reapproval if the project extends beyond the termination date.

The Board wishes you success in the pursuit of your research goals.

xc: R. Wayne Fuqua, Psychology

Approval Termination: May 21, 1992
Appendix E

English Translation of Acknowledgements
I would like to thank my wife, Lyn Raible, for her understanding and support throughout my graduate work and especially through the frustrations of conducting my dissertation research. Without Lyn's encouragement, I would not have gone on to do graduate work.

I would also like to thank my advisor, Richard Spates, for his guidance through University bureaucracy, and my other committee members, Michele Burnette, Gordon Hare, and Mal Robertson, for their efforts. I am grateful for the support of my co-workers at Michigan State University who helped me remain relatively sane while trying to conduct my research and complete my internship at the same time.

I owe a special thanks to Suzanne Cross for her personal encouragement and support of both my professional and personal development, to the Aanishinaabe pipe carriers who helped me onto the Red Road and helped to keep me there during my graduate work, and to Helen Roy who helped make it possible to write these words in the language of the people.
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