

POINT: Eye Movement Desensitization and Reprocessing: Is Psychiatry Missing the Point?

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Psychiatric Times • July 2000 • Vol. XVII • Issue 7

(Please see the [counterpoint discussion](#) by Shawn Cahill, Ph.D.-Ed.)

Posttraumatic stress disorder (PTSD) is a common and disabling condition. Recent estimates of the lifetime prevalence range between 6% and 15%, making this condition possibly more common than major depressive disorder (Breslau et al., 1998; Kessler et al., 1995). In addition, many patients who have been the victims of directed violence, such as rape or assault, continue to meet PTSD criteria 10 years after the incident (Breslau et al., 1998).

The standard of care for PTSD relies on pharmacological and psychological treatments that have limited effectiveness. A variety of outcome studies document only a 30% to 50% reduction of symptoms compared to placebo, with benefits noted only after several weeks of treatment (Brady et al., 2000; Davidson et al., 1997; Solomon, 1997). There is also an unacceptably high dropout rate (up to 40% for pharmacotherapy and sometimes higher with behavior therapy involving re-exposure to the traumatic incident). Finally, a number of treatments that include imagined or real re-exposure to traumatic material can lead to re-traumatization (Pitman et al., 1991; Solomon et al., 1992), often making such treatments risky or impractical. In fact, clinicians often avoid exposure therapy, even when it is clearly indicated (Boudewyns and Shipley, 1983).

In the last 11 years, a controversial treatment of trauma has been introduced (Shapiro, 1995, 1989). Eye movement desensitization and reprocessing (EMDR) has been shown in several controlled studies to reduce PTSD symptoms to the level of nonpatient controls. Results were seen in three sessions or less for more than 80% of patients with a dropout rate of less than 10% and no noticeable side effects (Wilson et al., 1995, 1997).

Such results would normally draw significant attention from the scientific and academic communities. Yet, EMDR has been largely ignored by the high-circulation psychiatric journals in the United States. This, in part, is

probably because EMDR relies on a rather unorthodox treatment component: requesting patients to move their eyes back and forth as in the movement of REM sleep.

The existing literature strongly suggests that there is more to EMDR than a powerful placebo effect. As of March, the National Center for PTSD's PILOTS database (an index to worldwide literature on PTSD) included more controlled, randomized trials of EMDR than any other specific modality, including medications (National Center for PTSD, 1999). Most controlled trials of EMDR for civilian PTSD report 77% to 90% remission in three sessions or less (Lazrove et al., 1998; Rothbaum, 1997; Scheck et al., 1998).

Only the combination of imaginal exposure and in vivo exposure has approached this degree of effectiveness (Marks et al., 1998). This combination treatment, however, requires considerably more time than EMDR. This is true even if a recent survey of psychotherapy experts rated EMDR below imaginal exposure cognitive therapy or in vivo exposure (Foa et al., 1999).

A meta-analysis published in 1998 (Van Etten and Taylor) also identified more controlled studies of EMDR in PTSD than any other psychotherapeutic treatment modality. Van Etten and Taylor also found EMDR to be the most rapidly effective and best tolerated of all the treatments reviewed, including pharmacotherapy and behavior therapy.

Despite a lack of face-validity, the weight of such evidence would seem to justify a closer look at this unconventional approach. To that end, I will briefly describe EMDR and existing treatment outcome studies. I will then propose psychological and neurophysiological mechanisms that may explain EMDR's treatment effects.

EMDR Procedure

EMDR relies on many effective ingredients from well-established psychotherapies, especially cognitive-behavioral therapy. It also has patients move their eyes rhythmically from side to side while thinking of aspects of the traumatic memories associated with their symptoms.

Specifically, the patient is asked to bring to mind a distressing aspect of the traumatic event. The therapist helps the patient focus on the visual representation of the trauma that is most strongly associated with the affect. Then the patient gives a negative self-statement (cognition) as a consequence of that event (e.g., "I'm helpless," "I'm weak," "I can't take care of myself," "I'm not good enough"). The patient also identifies the particular affect evoked by the image (e.g., fear, anger, sadness) and rates

its intensity from 0 to 10 on the Subjective Units of Distress (SUD). At the same time, the therapist helps the patient become aware of, and describe in detail, physical sensations arising when these images, thoughts and affects are held in consciousness.

Together, the therapist and the patient also establish a direction to the therapy by developing a positive cognition. This answers the question, "When you see yourself in that situation, what would you rather believe about yourself than the negative self-statement you just mentioned?" The patient is asked to rate the degree to which they believe this statement at a "gut" level. Together with the distress rating, this assessment helps the therapist gauge the resolution of the trauma and the progress toward a more adaptive interpretation of the past event.

So far, there is nothing in this procedure that differs in an essential way from good psychotherapy of trauma using a mix of ingredients from established approaches such as the cognitive and exposure elements emphasized by Foa (Foa, 1997; Foa and Kozak, 1986; Foa and Meadows, 1997).

Following this initial phase, however, patients then hold the image, cognition, affect and physical sensations in mind and move their eyes back and forth by following the therapist's hand (or by paying attention to another type of alternating stimulation). These sets of alternating stimulation last from 20 seconds to several minutes, depending on the patient's emotional reaction. They are associated with physiological correlates of relaxation such as reduction of blood pressure, heart rate and galvanic skin response (Wilson et al., 1996).

After each pause, the patient reports on "what came to mind." This may be a new image, thought, emotion or, at times, a change in physical sensations. The patient then focuses on this new information and another set of stimulation follows. The process continues until the associations no longer change or until only positive associations and sensations are reported. Depending on the remaining level of distress and strength of the positive cognition, the therapist may then decide to initiate further processing of the initial image or to start processing other aspects of the trauma.

Within a single session, patients often experience intense reliving of some aspect of their trauma. This is quickly followed by a sense of calm and a new understanding of the event that is no longer associated with painful emotions or demeaning negative self-statements. Patients often express new beliefs about the traumatic event such as, "It wasn't my fault," with a puzzled expression on their face followed by a smile of relief. Or they find themselves almost incredulously accepting a painful past mistake that has

harrowed them for decades with statements such as, "I really didn't have any other choice at the time. I did the best I could under the circumstances."

In between eye movement sets, patients engage in normal conversation with the therapist, typically reporting on what occurred during the stimulation. They do not appear to be in a trance state. They typically describe their flow of consciousness during eye movements as resembling focused daydreaming. It starts from a particular event or affect but, as the eye movements proceed, associations arise to other events, self-statements or even fantasies. Emotional states change quickly as if tracking the changes in cognitive associations.

Despite the enthusiasm of EMDR clinicians and a large number of positive case series, there have been some negative controlled studies of EMDR treatment of PTSD. For example, in Jensen's study (1994), EMDR showed no significant improvement above a control treatment. In Boudewyns et al. (1993), patients improved only on some measures of self-rated distress, but not on standardized instruments. In the Jensen study, however, the therapists had only limited acquaintance with EMDR protocols and could not demonstrate reliable use of the procedure. In the Boudewyns study, the patient population consisted of veterans with chronic PTSD whose disability benefits depended on their condition.

Deville and Spence (1999) found that EMDR compared unfavorably to a variant of cognitive behavior therapy called *trauma treatment protocol* for PTSD in civilians, the only study of civilian PTSD to report such a result. This study also reported a drop out rate in the EMDR condition that was three times higher than that of other studies of civilian PTSD.

As a whole, the available EMDR literature shows a more positive picture. Reports include a larger array of patient populations and types of traumas than with any other treatment interventions. There have been reports of positive results with schoolchildren as well as war veterans (Carlson et al., 1998; Chemtob et al., 1999), and traumas have ranged from rape to natural disasters (Grainger et al., 1997; Rothbaum, 1997). In an HMO setting, EMDR was found to be superior to standard care that consisted of a combination of individual psychotherapy, group therapy and medication determined by the treating psychiatrist (Marcus et al., 1997).

Does It Work?

The largest and most methodologically sound study available addressed several of the outstanding issues facing any new psychological treatment method: does the treatment work better than a waiting list; does it show improvement on a variety of clinical measures; how quickly are benefits

noticed; and how long do they last? In a study of 80 patients with trauma-related symptoms (46% meeting *DSM-IV* PTSD criteria and 54% with partial PTSD), Wilson et al. (1995, 1997) observed that average scores on the Impact of Event Scale dropped to a level comparable with the mean of the normal population after a single 90-minute session. Scores continued to decrease with two additional sessions. The same benefits were noted at three- and 15-month follow-up and were confirmed by other measures of treatment effects such as the Symptom Checklist-90, the State-Trait Anxiety Inventory and *DSM-IV* criteria for PTSD.

Van Etten and Taylor (1998) included pharmacotherapy as well as different psychotherapeutic approaches. They found EMDR among the most effective PTSD treatments. EMDR was also the fastest and the best tolerated by patients. Many therapists and patients preferred EMDR over intensive exposure therapy and other psychosocial treatments (Lipke, as cited in Shapiro, 1995; Solomon, 1997).

Studies of civilian PTSD have reported remission rates ranging between 77% and 90% in three sessions or less (Lazrove et al., 1998; Rothbaum, 1997; Scheck et al., 1998).

EMDR has not been as effective for veterans with combat-related PTSD (Boudewyns et al., 1993; Devilly et al., 1998; Jensen, 1994). No treatment, however, has shown more effectiveness in this population. Recent studies of sertraline (Zoloft), for example, have found it to be no better than placebo for veterans, in spite of its documented benefits in women suffering from civilian trauma (Mechcatie, 1999).

A major limitation of EMDR literature is the absence of head-to-head treatment comparison studies. Only these studies provide a scientifically valid comparison of the effectiveness and rapidity of different treatments.

One large trial funded by the National Institute of Mental Health is currently being conducted, but no preliminary information is available at this time. Two smaller studies, however, have been completed and their results described at conferences (Freund and Ironson, 1998; Lee and Graviel, 1998). Both found EMDR superior to either behavior or cognitive therapy protocols.

The Issue of Mechanism

An important factor limiting EMDR's acceptance may be the issue of mechanism. How could moving one's eyes back and forth possibly contribute to the resolution of trauma?

The first issue is whether or not eye movements are indeed a critical component of EMDR's effectiveness. Several studies have compared EMDR with and without eye movements. Some have found eye movements critical to therapeutic effects (Montgomery and Ayllon, 1994; Wilson et al., 1996), whereas others have not (Boudewyns and Hyer, 1996; Pitman et al., 1996; Renfrey and Spates, 1994). In the Renfrey and Spates study (1994), EMDR with eye movements resulted in an 85% remission rate of PTSD in three sessions, whereas the remission rate was only 50% without eye movements. Given the small number of patients in each group, it is unclear whether this study truly argued against a differential effect of eye movements. In addition, in Pitman et al.'s study (1996) the condition in which therapeutic effects were noted without eye movements included other forms of stimulation such as hand- or finger-taps. At this time, EMDR clinicians generally agree that any form of alternating hemispheric stimulation may be sufficient to induce therapeutic effects (Shapiro, 1995); eye movements per se do not seem to be critical.

Some have suggested that eye movements simply provide a distraction that reduces the intensity of trauma recollection (Pitman et al., 1996; Solomon, 1997). In this case, EMDR's efficiency would come from repeated exposure to tolerable images of the trauma, together with progressive desensitization as occurs in other behavioral treatment procedures. This hypothesis fails, however, to explain the rapid desensitization in EMDR, compared to the slower response with other exposure-based modalities. Furthermore, behavioral theorists argue that, to be effective, exposure should be continuous and that intermittent exposure-such as the EMDR protocol entails-tends to increase the conditioned response rather than decrease it (Marks et al., 1998). This difference alone would seem to justify a greater research investment in the neural basis of EMDR treatment effects.

Some research suggests mechanisms through which alternating stimulation or possibly other forms of central nervous system-orienting responses could contribute to a treatment effect in PTSD (e.g., Armstrong and Vaughan, 1996). For example, cognitive neuroscience studies on hemispheric laterality and on the neurophysiological basis of consciousness offer plausible hypotheses that could be tested empirically. In the last decade, PTSD research has confirmed that trauma induces dissociative symptoms (van der Kolk et al., 1996a). It is also now known that dissociative experiences at the time of trauma are associated with more severe posttraumatic symptoms (Bremner et al., 1992; Koopman et al., 1994; Shalev et al., 1996).

Dissociative phenomena illustrate the breakdown of the normal integration in consciousness of the multifaceted aspects of experience. Thoughts, emotions and physical sensations that initially pertained to a single event

are split into separate representations which are no longer recalled together. Yet, some fragments may be recalled separately, often with the vividness of the original experience. Such dissociative memories are often associated with marked emotional lability, unexplained somatic symptoms or negative self-evaluations and self-defeating behaviors (van der Kolk et al., 1996b).

At the same time, recent cognitive neuroscience research has started to clarify the mechanisms underlying the integrity of conscious experience. Consciousness requires the simultaneous apprehension in one's mind of multiple sensory features pertaining to a single scene or object. For example, the conscious perception of a person talking involves the binding together of a visual percept with auditory stimuli, affective responses evoked by their presence and cognitive representations evoked by their speech. These different aspects of perceptions rely on different neural structures, such as inferior temporal regions for facial recognition, posterior temporal regions for speech processing, and limbic and paralimbic regions such as the amygdala for emotional coloring.

How does the brain know that the voice being heard and the emotion being felt are related to the same particular person in the visual field rather than to some other person, or even another object altogether? How do these different neural representations in vastly different anatomical regions coalesce to produce an integrated experience?

A dominant theory of consciousness proposes that this binding of different neural representations occurs through periods of synchronization of oscillating neuronal discharges in the frequency range of 30 Hz to 80 Hz (γ oscillations) between the different anatomical regions (Crick, 1984; Damasio, 1989a; Kinsbourne, 1988). Human studies have demonstrated a link between a cognitive act of perception and diffuse synchronization in the γ band (Joliot et al., 1994; Rodriguez et al., 1999).

The same synchronization patterns of neural activity across vastly different brain regions are thought to be essential to memory recall. Damasio (1989b) referred to it as "time-locked multi-regional retroactivation" to emphasize that the process of recall involves a stimulus—such as a simple smell-evoking activation or related stimuli throughout a large number of brain areas, including visual and somatosensory cortex as well as limbic regions. The "glue" between these different areas is provided by the synchronization of their neural activity in a particular frequency band, which probably relies on the corpus callosum (Liederman, 1995). In humans, the breakdown of this mechanism is best illustrated by the fragmented consciousness of split-brain patients.

Even when the corpus callosum is intact, striking dissociations of consciousness can be demonstrated (Risse and Gazzaniga, 1978). Subjects being prepared for neurosurgery were submitted to anesthesia of a single brain hemisphere at a time to test for the lateralization of critical functions. If they were presented with an object in their left hand (right hemisphere) while their left hemisphere was anesthetized, they did not consciously recall having been presented with anything after recovery from the transient anesthesia. This was true even though both hemispheres were then active-and presumably communicating-at the time patients were asked about the object. The representation of the object was still active and accessible in the right hemisphere because subjects could point correctly with their left hand to a drawing of the object presented among foils.

This experiment illustrated dramatically that information encoded in a dissociated state can be 1) unavailable to conscious awareness; yet 2) continue to influence behavior; and 3) this can occur with a perfectly functional corpus callosum linking the two hemispheres. Hence, any interference with the encoding of information, as occurs during trauma when dissociative symptoms are present, can result in long-term dissociation of information within the CNS and, therefore, in fragmented memories where cognitive, somatosensory and emotional aspects of the original scene are not integrated into the larger context of the patient's verbal representation of the world (Schiffer, 1996).

It is possible that rhythmic eye movements or other types of orienting stimulation occurring while a patient is re-experiencing one aspect of a dissociated memory may simply induce a background of synchronous neural activity across cerebral hemispheres and perhaps within each hemisphere. If this were the case, it could contribute to the reintegration of dissociated aspects of memories by re-establishing a synchrony between functionally disconnected areas. This would explain why alternating stimulation often initially produces powerful abreactions in traumatized patients. Continuing stimulation may then promote integration of the traumatic material with verbal representations of the larger context of the patient's life and knowledge about the world. As this occurs, the emotional impact of the trauma is diluted by associations with verbal material that put it in proper perspective. Though this is the goal of any therapy for traumatized individuals, rhythmic orienting stimulation may accelerate the process by facilitating the neural mechanisms through which dissociated memories are re-integrated into normal consciousness.

Another plausible explanation for the rapid treatment effects of EMDR may be that, with or without eye movements, the EMDR protocol provides a powerful blend of the best ingredients of different therapies. These include a patient-controlled pace of exposure, an emphasis on changes in

the quality of memories and experiences as the treatment progresses, and an integration of cognitive re-structuring with affective and somatic experiences related to the trauma (Smyth, 1995).

Eye movements would be only one element, allowing self-paced style exposure that is much more tolerable to patients than directed-exposure flooding or implosion protocols. Eye movements per se would not be necessary to the protocol, which could be accomplished with eyes closed or fixed (Boudewyns and Hyer, 1996; Pitman et al., 1996). Eye movements would be a way to increase patients' comfort level and their ability to tolerate self-paced exposure, reduce self-consciousness and facilitate relevant associations to the traumatic material (Boudewyns and Hyer, 1996). If this is the case, Shapiro's main contribution (1995, 1989) to the treatment of PTSD would be how she has articulated many available elements of good therapy into a simple and effective protocol that is more acceptable to patients and easier for therapists to learn and use than other forms of treatment.

We are currently testing this hypothesis at our center in a double-blind randomized trial of the effect of different types of stimulation on process measures during EMDR sessions.

It may also be that a satisfying explanation for the role of orienting stimulation will elude research for some time. This would put EMDR in the same category as lithium and electroconvulsive therapy: effective treatments that had equally implausible mechanisms of action when first introduced. It was only through the sheer weight of their effectiveness that they proved themselves, even if their mechanisms of action remain as mysterious today as they were 40 years ago.

Conclusion

If EMDR passes the test of ever-greater scrutiny, the availability of a rapid, effective and well-tolerated treatment for the consequences of traumatic stress will have major implications for psychiatry.

People who have been exposed to traumatic events suffer from many syndromes other than PTSD. As Yehuda et al. (1998) recently reviewed, more than half of patients who experience a traumatic incident develop other *DSM-IV* conditions and, in fact, do not meet PTSD criteria. These include not only major depressive disorder and anxiety disorders, but also substance abuse disorders and eating disorders. Having at our disposal a treatment intervention that can rapidly resolve the consequences of trauma is likely to have a profound effect on our very concept of mental disorders, above and beyond our choice of therapeutic options.

In addition, the availability of an effective treatment will dramatically affect our recognition and recording of the conditions to which it can be applied. It is likely that the growing popularity of EMDR will herald a new interest in PTSD, dissociative disorders and somatoform disorders that are significantly underrecognized today.

For example, a study by Saxe et al. (1993) suggests that at least 15% of state hospital inpatients met *DSM-III* criteria for a dissociative disorder. These patients had high rates of major depression, PTSD, substance abuse and borderline personality disorders, and they frequently had a history of childhood trauma. Yet, dissociative symptoms were typically not explored nor recorded. This is not surprising in a context where treatment options for these conditions are limited. Understandably, clinicians focus on symptoms and diagnoses that affect their treatment decisions.

How long will it take before EMDR becomes a widely accepted treatment in psychiatry? Shapiro's first controlled study of EMDR was published in 1989. Eleven years later, the procedure is rapidly gaining credibility at the periphery of our academic centers. Although empirical evidence weighs in its favor, the lack of a credible explanation for its mechanism of action will slow its acceptance.

In medicine, and in our field especially, caution and skepticism are not only appropriate, but necessary. The claims made by EMDR practitioners and by the majority of available controlled trials are rather extraordinary. It is normal and healthy that they challenge our credulity. Let us hope, however, that the available evidence will also stimulate our curiosity and not let us reject out-of-hand what may turn out to be an enormous treatment advance in psychiatry.

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References

Armstrong MS, Vaughan K (1996), An orienting response model of eye movement desensitization. *J Behav Ther Psychiatry* 27(1):21-32.

Boudewyns PA, Hyer LA (1996), Eye movement desensitization and reprocessing (EDMR) as treatment for post-traumatic stress disorder (PTSD). *Clinical Psychology and Psychotherapy* 3(3):185-195.

Boudewyns PA, Shipley RH (1983), *Flooding and Implosive Therapy: Direct Therapeutic Exposure in Clinical Practice*. New York: Plenum Press.

Boudewyns PA, Stwertka SA, Hyer LA et al. (1993), Eye movement desensitization for PTSD of combat: a treatment outcome pilot study. *The Behavior Therapist* 16(2):29-33.

Brady K, Pearlstein T, Asnis GM et al. (2000), Efficacy and safety of sertraline treatment of posttraumatic stress disorder. A randomized controlled trial. *JAMA* 283(14):1837-1844.

Bremner JD, Southwick S, Brett E et al. (1992), Dissociation and posttraumatic stress disorder in Vietnam combat veterans. *Am J Psychiatry* 149(3):328-332.

Breslau N, Kessler RC, Chilcoat HD et al. (1998), Trauma and posttraumatic stress disorder in the community: the 1996 Detroit Area Survey of Trauma. *Arch Gen Psychiatry* 55(7):626-632.

Carlson JG, Chemtob CM, Rusnak K et al. (1998), Eye movement desensitization and reprocessing (EMDR) treatment for combat-related posttraumatic stress disorder. *J Trauma Stress* 11(1):3-24.

Chemtob CM, Nakashima J, Hamada R (1999), EMDR treatment for children with disaster-related PTSD. Abstract No. 68B. Presented at the 152nd Annual Meeting of American Psychiatric Association. Washington, D.C.; May 15-20.

Crick F (1984), Function of the thalamic reticular complex: the searchlight hypothesis. *Proc Natl Acad Sci U S A* 81(14):4586-4590.

Damasio A (1989a), The brain binds entities and events by multiregional activation from convergence zones. *Neural Comput* 1:123-132.

Damasio A (1989b), Time-locked multiregional retroactivation: a systems-level proposal for the neural substrates of recall and recognition. *Cognition* 33(1-2):25-62.

Davidson JR, Malik ML, Sutherland SN (1997), Response characteristics to antidepressants and placebo in post-traumatic stress disorder. *Int Clin Psychopharmacol* 12(6):291-296.

Devilley GJ, Spence SH (1999), The relative efficacy and treatment distress of EMDR and cognitive-behavior trauma treatment protocol in the amelioration of posttraumatic stress disorder. *J Anxiety Disord* 13(1-2):131-157.

Devilley GJ, Spence SH, Rapee RM (1998), Statistical and reliable change with eye-movement desensitization and reprocessing: treating trauma within a veteran population. *Behavior Therapy* 29:435-455.

Foa EB (1997), Psychological processes related to recovery from a trauma and an effective treatment for PTSD. *Ann N Y Acad Sci* 821:410-424.

Foa EB, Meadows EA (1997), Psychosocial treatments for posttraumatic stress disorder: a critical review. *Annu Rev Psychol* 48:449-480.

Foa EB, Kozak MJ (1986), Emotional processing of fear: exposure to corrective information. *Psychol Bull* 99(1):20-35.

Freund B, Ironson G (1998), A comparison of two treatments for PTSD: a pilot study. Presented at the 32nd Annual Convention of the Association for the Advancement of Behavior Therapy. Washington D.C.; Nov. 5-8.

Grainger RD, Levin C, Allen-Byrd L et al. (1997), An empirical evaluation of eye movement desensitization and reprocessing (EMDR) with survivors of a natural disaster. *J Trauma Stress* 10(4):665-671.

Jensen JA (1994), An investigation of eye movement desensitization and reprocessing (EMD/R) as a treatment for posttraumatic stress disorder (PTSD) symptoms of Vietnam combat veterans. *Behavior Therapy* 25(2):311-325.

Joliot M, Ribary U, Llinas R (1994), Human oscillatory brain activity near 40 Hz coexists with cognitive temporal binding. *Proc Natl Acad Sci U S A* 91(24):11748-11751.

Kessler RC, Sonnega A, Bromet E et al. (1995), Posttraumatic stress disorder in the National Comorbidity Survey. *Arch Gen Psychiatry* 52(12):1048-1060.

Kinsbourne M (1988), Integrated field theory of consciousness. In: *Consciousness in Contemporary Science*, Marcel AJ, Bisiach E, eds. Oxford, England: Clarendon Press, p230-256.

Koopman C, Classen C, Spiegel D (1994), Predictors of posttraumatic stress symptoms among survivors of the Oakland/Berkeley, Calif., firestorm. *Am J Psychiatry* 151(6):888-894.

Lazrove S, Triffleman E, Kite L et al. (1998), An open trial of EMDR as treatment for chronic PTSD. *Am J Orthopsychiatry* 68(4):601-608.

Lee C, Graviel H (1998), Treatment of post-traumatic stress disorder: a comparison of stress inoculation training with prolonged exposure and eye-movement desensitization and reprocessing. Presented at the World Congress of Behavioral and Cognitive Therapies. Acapulco, Mexico; July 21-26.

Liederman J (1995), A reinterpretation of the split-brain syndrome: Implications for the function of corticocortical fiber. In: Brain Asymmetry, Davidson RJ, Hugdahl K, eds. Cambridge, Mass.: MIT Press, pp451-490.

Marcus SV, Marquis P, Sakai C (1997), Controlled study of treatment of PTSD using EMDR in an HMO setting. *Psychotherapy* 34(3):307-315.

Marks I, Lovell K, Noshivani H et al. (1998), Treatment of posttraumatic stress disorder by exposure and/or cognitive restructuring: a controlled study. *Arch Gen Psychiatry* 55(4):317-325.

Mechcatie E (1999), Zoloft may become the first drug targeted at PTSD: beneficial effects seen only in women with posttraumatic stress disorder. *Clinical Psychiatry News* 27(11):1,5.

Montgomery RW, Ayllon T (1994), Eye movement desensitization across subjects: subjective and physiological measures of treatment efficacy. *J Behav Ther Exp Psychiatry* 25(3):217-230.

National Center for PTSD (1999), PILOTS Database for PTSD. Available at: www.dartmouth.edu/~dms/ptsd/PILOTS.html. Accessed March 27.

Pitman RK, Orr SP, Altman B et al. (1996), Emotional processing during eye movement desensitization and reprocessing therapy of Vietnam veterans with chronic posttraumatic stress disorder. *Compr Psychiatry* 37(6):419-429.

Pitman RK, Altman B, Greenwald E et al. (1991), Psychiatric complications during flooding therapy for posttraumatic stress disorder. *J Clin Psychiatry* 52(1):17-20.

Renfrey G, Spates CR (1994), Eye movement desensitization: a partial dismantling study. *J Behav Ther Exp Psychiatry* 25(3):231-239.

Risse GL, Gazzaniga MS (1978), Well-kept secrets of the right hemisphere: a carotid amygdala study of restricted memory transfer. *Neurology* 28(9 part 1):950-953.

Rodriguez E, George N, Lachaux JP et al. (1999), Perception's shadow: long-distance synchronization of human brain activity. *Nature* 397(6718):430-433 [see comments].

Rothbaum BO (1997), A controlled study of eye movement desensitization and reprocessing in the treatment of posttraumatic stress disorder sexual assault victims. *Bull Menninger Clin* 61(3):371-334.

Saxe GN, van der Kolk BA, Berkowitz R et al. (1993), Dissociative disorders in psychiatric inpatients. *Am J Psychiatry* 150(7):1037-1042 [see comments].

Scheck MM, Schaeffer JA, Gillette C (1998), Brief psychological intervention with traumatized young women: the efficacy of eye movement desensitization and reprocessing. *J Trauma Stress* 11(1):25-43.

Schiffer F (1996), Cognitive activity of the right hemisphere: possible contributions to psychological function. *Harv Rev Psychiatry* 4(3):126-138.

Shalev AY, Peri T, Canetti L, Schreiber S (1996), Predictors of PTSD in injured trauma survivors: a prospective study. *Am J Psychiatry* 153(2):219-225.

Shapiro F (1995), *Eye Movement Desensitization and Reprocessing: Basic Principles, Protocols, and Procedures*. New York: Guilford Press.

Shapiro F (1989), Efficacy of the eye movement desensitization procedure in the treatment of traumatic memories. *J Trauma Stress* 2(2):199-223.

Smyth L (1995), *Clinician's Manual for the Cognitive-Behavioral Treatment of Post Traumatic Stress Disorder*. Harve de Grace, Md.: Red Toad Road Publishing Company.

Solomon SD (1997), Psychosocial treatment of posttraumatic stress disorder. *Psychotherapy in Practice* 3(4):27-41.

Solomon SD, Gerrity ET, Muff AM (1992), Efficacy of treatments for posttraumatic stress disorder. *JAMA* 268(5):633-638.

van der Kolk BA, van der Hart O, Marmar CR (1996a), Dissociation and information processing in posttraumatic stress disorder. In: *Traumatic Stress: The Effects of Overwhelming Stress on Mind, Body and Society*, van der Kolk BA, McFarlane AC, Weisaeth L, eds. New York: Guilford Press, pp303-330.

van der Kolk BA, Pelcovitz D, Roth S et al. (1996b), Dissociation, somatization, and affect dysregulation: the complexity of adaptation of trauma. *Am J Psychiatry* 153(7 suppl):83-93.

Van Etten ML, Taylor S (1998), Comparative efficacy of treatments for post-traumatic stress disorder: a meta-analysis. *Clinical Psychology and Psychotherapy* 5:126-144.

Wilson DL, Silver SM, Covi WG, Foster S (1996), Eye movement desensitization and reprocessing: Effectiveness and autonomic correlates. *J Behav Ther Exp Psychiatry* 27(3):219-229.

Wilson SA, Becker LA, Tinker RH (1997), Fifteen-month follow-up of eye movement desensitization and reprocessing (EMDR) treatment for posttraumatic stress disorder and psychological trauma. *J Consult Clin Psychol* 65(6):1047-1056.

Wilson SA, Becker LA, Tinker RH (1995), Eye movement desensitization and reprocessing (EMDR) treatment for psychologically traumatized individuals. *J Consult Clin Psychol* 63(6):928-937.

Yehuda R, McFarlane AC, Shalev AY (1998), Predicting the development of posttraumatic stress disorder from the acute response to a traumatic event. *Biol Psychiatry* 44(12):1305-1313.

Retrieved from www: 8/31/05 <http://www.psychiatrictimes.com/p000736.html>