

Sleep-Dependent Memory Processing and EMDR Action

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The unique efficacy of eye movement desensitization and reprocessing (EMDR) in the treatment of post-traumatic stress disorder is thought to result from changes in the brain/mind state induced by bilateral sensory stimulation, but the nature and specific consequences of these changes remain unknown. The possibility that bilateral stimulation induces a brain/mind state similar to that of rapid eye movement sleep is supported by studies showing that sleep facilitates forms of memory processing arguably necessary for the resolution of trauma. Such studies, along with direct studies of the impact of bilateral stimulation on memory and emotional processing, and dismantling studies identifying the requisite features of such bilateral stimulation for effective trauma processing, will eventually lead to an understanding of the neurobiological basis of EMDR.

Keywords: EMDR; sleep; REM sleep; memory; associative processing

Posttraumatic stress disorder (PTSD) is classified by *DSM-IV* (American Psychiatric Association, 1994) as an anxiety disorder. While this may be a clinically appropriate designation, from a mechanistic perspective it may be more appropriate to treat PTSD primarily as a memory disorder. On a most basic level, PTSD develops when memories of traumatic events, encoded during an actual trauma, fail to be processed normally over time (van der Kolk, 1994). Such normal processing acts over days to months to reduce both the intrusiveness of the memory and the affect associated with such recall and to integrate the memory into the individual's larger network of related memories. In doing so, it provides a meaningful and accurate understanding of both the event and its implications for the individual's future. It is when this processing fails, that PTSD develops.

Memory Processing and Trauma

Memories are not like photographs. They evolve. After a memory is initially formed, it goes through an extended period of consolidation—a complex set of automatic processes, occurring without intent and outside of conscious awareness—that modifies the

memory. In the end, a memory can be substantially different from its original form, with some parts still as vivid as the day they were formed and other parts forgotten. At the same time, the memory becomes integrated into wide-ranging memory networks that create a context for the original memory and, in the process, construct an implicit interpretation of the memory (Walker & Stickgold, 2006).

While the processing of small, everyday, distressful events is normally handled efficiently by these automatic mechanisms, grief and painful traumas can overwhelm them. In such circumstances, processing is often aided by social interactions, through speaking with family and friends or with professionals trained in trauma processing. Indeed, one meta-analysis found a perceived lack of social support to be the strongest predictor of the development of PTSD in a variety of populations (Brewin, Andrews, & Valentine, 2000).

One possible reason for the failure of automatic processing under these circumstances could be an inappropriate encoding of the memory at the time of the trauma, resulting in a memory that lacks key components that are critical for subsequent automatic processing. For example, van der Kolk (1994) has suggested that individual features of the traumatic

memory may fail to be bound together into a coherent episodic memory at the time of the trauma, producing a memory that consists of unintegrated elements that the brain/mind cannot process as a unitary event.

But a second possibility is that the off-line processing system itself fails, leaving the memory frozen in its original form—raw, intrusive, distressing, and unexplained. Various PTSD treatments, as well as the theories that inform them, seek to facilitate this posttraumatic processing. Unfortunately, the nature of this processing system is, at best, poorly understood, and our failure to understand this system impairs our ability to develop both effective models of the disorder and treatments for it. Thus, exposure therapy (Foa & Kozak, 1986; Marks, 1979), based on classical extinction paradigms, attempts to reduce the emotional response to the recall of a trauma, or, in the terminology of classical conditioning theory, to uncouple the conditioned stimulus from the conditioned response. When successful, such treatment reduces the symptomatology to below clinical levels. However, there is no suggestion that such treatment facilitates the function of the normal posttraumatic processing system, namely the integration of a modified traumatic memory into the patient's larger network of related memories and the construction of a more accurate understanding of the traumatic event and its implications for the patient's future.

In contrast, eye movement desensitization and reprocessing (EMDR) takes a more complex approach to trauma processing (Shapiro, 1995), processing not only the emotion associated with the trauma, but the entire trauma memory within its network of associated memories. While the treatment procedure is, in many ways, straightforward from a psychological perspective, the role of repetitive, alternating bilateral sensory stimulation in the process remains unclear and controversial.

In what follows, we take two different approaches to this question of EMDR's mechanisms of action. First, we present additional evidence for our previously published model of EMDR that suggests that EMDR activates normally sleep-dependent memory processing (Stickgold, 2002). We then discuss a series of potential dismantling studies that could be used to further clarify the role of bilateral stimulation in the efficacy of EMDR. It is hoped that juxtaposing one theory with many unanswered questions will help some readers find a way forward toward studies that will clarify the role of eye movements in EMDR.

Sleep and Memory Processing

We have presented a model of trauma processing, and of EMDR, proposing that the unique benefits

of EMDR's bilateral stimulation result from its ability to activate normally sleep-dependent memory processing, which has broken down in the face of overwhelming trauma (Stickgold, 2002). This model was based on relatively recent findings of a role of sleep in normal memory processing (Stickgold, 2002, 2007). Since then, a rich and rapidly growing literature has demonstrated that sleep plays a critical role in the natural, automatic, and unattended processing of memories, across days, months, and even years (Stickgold, 2005). But it is now clear that this processing is more sophisticated than the simple "memory consolidation" originally proposed. Instead, sleep-dependent memory processing also results in the identification, integration, and enhancement of those aspects of memories calculated to be most important. It is these more complex forms of sleep-dependent processing that are presumably in play in normal trauma processing. Three examples of these sleep-dependent processes will help make clear how powerful these processes are.

Sensing the Solution: The Tower of Hanoi

Often, trauma survivors appear to have all the pieces of the puzzle but are unable to put them together, either explicitly, in words, or even implicitly, in a deeper, nonverbal understanding of what happened. Recent evidence indicates that, in some cases, sleep can facilitate the development of exactly this type of nonverbal understanding.

The Tower of Hanoi is an ancient puzzle in which the player must move a stack of disks from one of three pegs on a board to another, following two deceptively simple rules. First, only one disk can be moved at a time, and, second, a larger disk can never be placed on a smaller one. Thus, in the example shown in Figure 1A, play must start with the top disk being moved to one of the empty pegs, perhaps the middle one. Next, the second disk can be moved, but not to the peg with the first disk, because the second disk is larger. Instead, it must be moved to the empty peg at the right. With the top disk on the middle peg and the second disk on the right-hand peg, there is no place to move the third disk, at the top of the left peg, because it is larger than the disks on the other two pegs. The reader is left to solve the puzzle, as were the subjects in a study by Smith and Smith (2003).

In this study, subjects completed the puzzle five times in a first session and then another five times in a second session a week later. Figure 1B shows

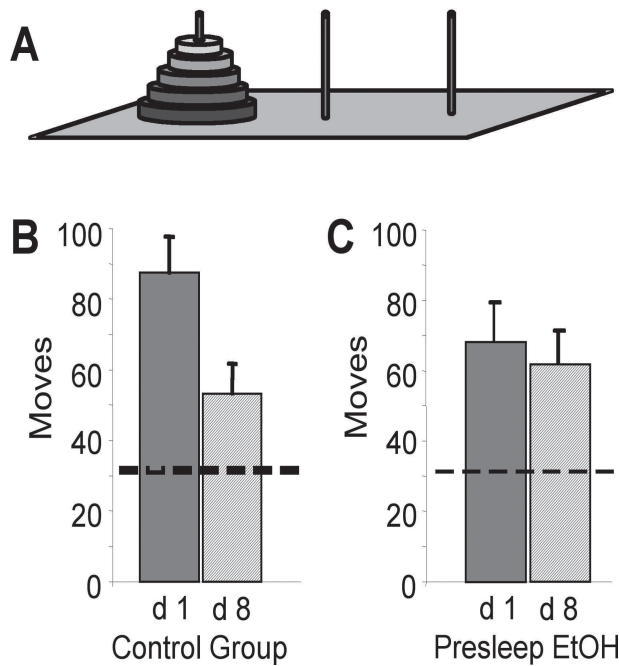


FIGURE 1. Tower Of Hanoi.

Note. (A) Subjects must move all five disks to the right-hand pole, moving only one disk at a time and never placing a disk on top of a smaller disk; (B) Control Group: Moves required to complete task during afternoon training (d1) and 7 days later (d 8); (C) Presleep EtOH: Subjects consumed alcohol shortly before bedtime the night after training and show no improvement on d 8. From Smith & Smith, 2003.

the results for normal control subjects. In the first session, subjects took, on average, 86 moves to complete the task. But a week later, on day 8, they were significantly better, taking only 51 moves, solving the problem with 40% fewer moves. Thus, they had retained the knowledge they gained during the initial training and were able to apply it during the second session.

But what is it that subjects remembered? At the end of training, subjects' performance was still markedly worse than the theoretical minimum of 31 moves (dashed line), and subjects were unable to explicitly state the rules for optimal performance. Instead, they were still working from a learned sense of which moves were more likely to move them toward their goal. In other words, they had only developed a partial, nonverbal understanding of how to perform the task. It was this implicit knowledge that was maintained, and perhaps even enhanced, by sleep.

The evidence of sleep's role in this process comes from the data shown in Figure 1C. This shows the

performance of subjects who consumed several alcoholic drinks shortly before going to bed (but several hours after the training session). Alcohol inhibits REM sleep, and thus these subjects were partially REM deprived the night after training. Unlike the normal control subjects (Figure 1B), subjects who consumed alcohol showed no improvement at retest a week later. Instead, all the potential benefits of the initial training were lost. Additional studies demonstrated that the alcohol effect required that it be consumed near the time of sleep onset, providing strong evidence that it was the impact of the alcohol on sleep, rather than on memory per se, that caused the subsequent deficit. Apparently, REM sleep is critical after this type of learning if the implicit knowledge gained is to be retained over time.

Practicing psychologists watch clients struggle with such knowledge all the time, trying to learn how to expand and use it. The results of the Tower of Hanoi study suggest that sleep-dependent processes may play an important role in maintaining progress from one session to the next.

Naming the Solution: The Number Reduction Task

In resolving traumatic memories, one wants to go beyond a felt sense of what happened and develop an explicit verbal description that provides insight into the significance of the trauma. Again, sleep can facilitate the development of such insight.

An impressive example of such sleep-dependent memory was reported by Wagner and colleagues (Wagner, Gais, Haider, Verleger, & Born, 2004). In their study, subjects were taught a complex set of rules for solving a group of mathematical problems (see Figure 2A). Unknown to the subjects, a simpler

solution also existed that allowed the problem to be solved without any calculations. When subjects were retested 12 hours after their initial training, a number of subjects discovered this simpler method of performing the task. But the number of subjects gaining this insight was more than doubled after a night of sleep (see Figure 2B, right).

Not all sleep is equally effective in facilitating this development of insight. The 60% of subjects who gained insight the next day had significantly less deep non-REM sleep (Stage 3 and 4, referred to collectively as slow-wave sleep, SWS) than the 40% who did not develop insight. Interestingly, neither REM sleep nor

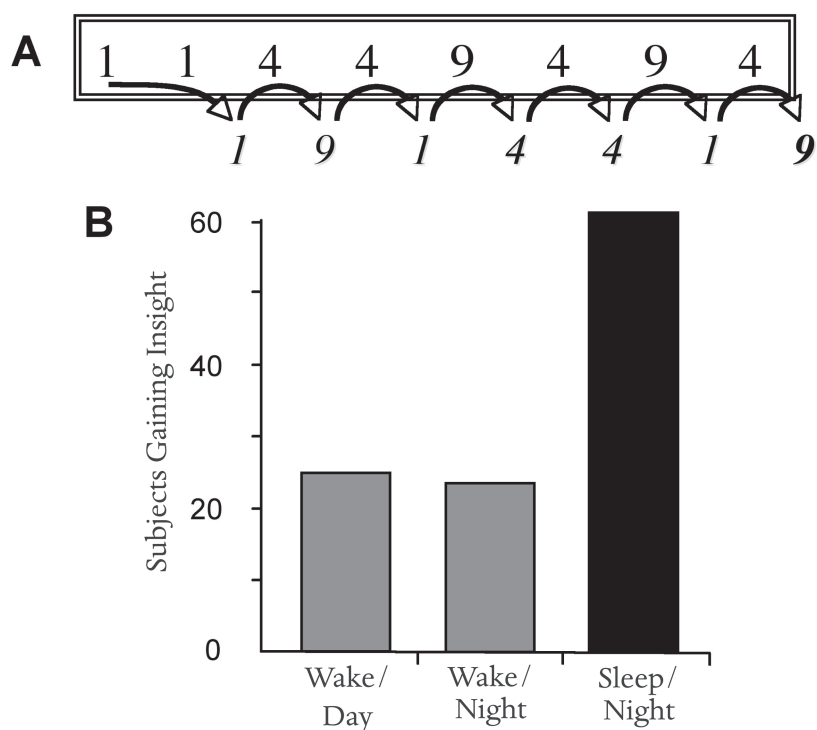


FIGURE 2. Number Reduction Task.

Note. (A) Subjects are taught to reduce an eight-digit string of 1s, 4s and 9s (shown in the box) to a single digit by sequentially processing pairs of digits, producing intermediate answers (numbers in italics below original string) using a set of explicit rules: (1) Use the first digit as the first intermediate answer; (2) if an intermediate answer is the same as the next digit in the sequence (e.g., 1 and 1), the next intermediate answer is the same digit (in this example, 1); (3) if they are not the same (e.g., 1 and 4), the next intermediate answer is the third possible digit (i.e., 9); (4) the final intermediate answer is the solution to the problem. However, a simpler rule exists; the second to last unique digit in the original eight-digit sequence (the 9 near the end of the sequence inside the box) is also the answer. (B) Percent of subjects who successfully identified and implemented the shortcut. Wake/Day = subjects who were trained at 9:00 A.M. and were tested at 9:00 P.M.; Wake/Night = subjects who were trained at 9:00 P.M., were kept awake all night, and were tested at 9:00 A.M. the next morning; Sleep/Night = subjects who were trained at 9:00 P.M., slept normally overnight, and were tested at 9:00 A.M. the next morning. From Wagner et al., 2004.

light (Stage 2) non-REM sleep showed a significant increase. Nonetheless, the decreased SWS suggests that, as with the Tower of Hanoi task, it is REM sleep that is critically important.

This surprising finding demonstrates an even more powerful consequence of sleep—during sleep, the brain is able to analyze and manipulate information gleaned from earlier experiences to facilitate the development of insights during subsequent wake, even when the individual is unaware that there is any insight to discover. For the clinician, these results indicate an additional benefit of sleep to that proposed above. Not only does sleep help maintain nondeclarative, nonconscious learning from one session to the next, but it can even further process the information from the previous session, allowing for even greater progress during the next session, with the development of explicit insight into one’s situation. In many ways, this feels similar to the common concept of “sleeping on a problem,” where one goes to bed not knowing how to make a complicated choice, but wakes the next morning with a clear decision. But most importantly

from the perspective of EMDR treatment, these results suggest that inadequate, impoverished, or defective sleep—perhaps sleep with abnormally low levels of REM sleep or with incompletely established REM sleep—may slow or even prevent normal psychological processing of the events in one’s life. The possibility that EMDR can facilitate REM-like processing during wake (Stickgold, 2002) would then explain how EMDR could facilitate recovery from PTSD.

Sleep and Emotional Memory

These two examples of sleep-dependent memory processing demonstrate the unique ability of the sleeping brain to perform complex cognitive processing of relatively abstract information, putting together information that is poorly understood or whose relevance to the problem is unclear. Sleep also processes explicit emotional memories, and again these sleep-dependent processes show more subtlety and sophistication than one might expect. A recent study comes almost directly from trauma literature (Payne, Stickgold, Swanberg, & Kensinger,

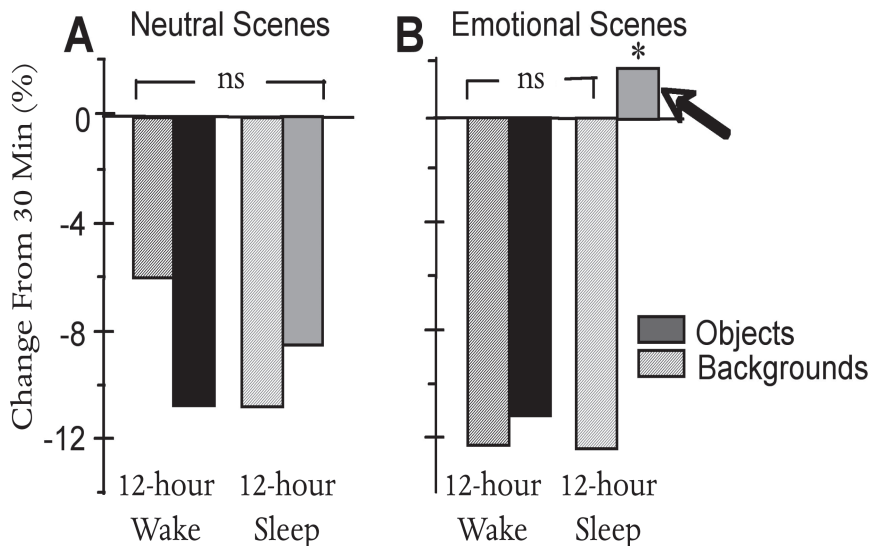


FIGURE 3. Emotional Trade-Off.

Note. Subjects were shown 64 scenes with either neutral or emotional objects on neutral backgrounds and were subsequently tested separately on the objects and backgrounds during a recognition test that also included objects and backgrounds from previously unseen scenes. (A) Recall of neutral objects and their backgrounds: percent decrease in correct recognition compared to recall tested 30 minutes after training; (B) Recall of emotional objects and their neutral backgrounds: percent decrease compared to 30-minute recall; 12-hour wake = subjects who were trained at 9:00 A.M. and were tested at 9:00 P.M. that evening; 12-hour sleep = subjects who were trained at 9:00 P.M. and were tested at 9:00 A.M. the next morning after a night of normal sleep. From Payne et al., 2008.

ns = nonsignificant differences.

* $p < .05$ compared to all other values.

2008), investigating the basis of the “weapon focus effect.” “Weapon focus” refers to the focusing of crime victims’ attention on a weapon, to the detriment of memories for other details of the crime, such as the assailant’s face, and has been investigated experimentally by researchers including Elizabeth Loftus (Loftus, Loftus, & Messo, 1987). Recent research has clarified this “emotional trade-off,” which more generally leads to improved memory for emotionally charged objects at the expense of memory for a neutral background on which the object is displayed. While this trade-off can be seen as soon as 30 minutes after subjects are shown such scenes, it now appears that sleep, again, plays an important role in how these memories change over time.

When subjects were shown a series of pictures of neutral and emotional objects, each displayed on a unique neutral background, and were tested on their ability to recognize the objects and backgrounds 12 hours later, their ability to do so was about 10% lower than it had been just 30 minutes after seeing the pictures (Payne et al., 2008). When the 12 hours were across a day of wakefulness, this 10% decrease was seen for both emotional and neutral scenes and for both the objects and the backgrounds (see Figure 3A). When the 12 hours were across a night of sleep, the decreased recognition was seen for both the objects and backgrounds of the neutral scenes (Figure 3B, left) and for the backgrounds of the emotional scenes (Figure 3B, right). But in stark contrast to all these examples of time-dependent deterioration of memories, recognition of the emotional objects actually increased across a night of sleep (Figure 3B, right, arrow).

As a result, sleep provides both a quantitative effect of maintaining or even enhancing memory for the emotional objects observed and a qualitative effect, enhancing memory for emotional objects while allowing their neutral background scenes to be forgotten. What remains unknown is whether sleep also weakens the emotional intensity of the memory, as one would want for the effective processing of traumatic memories. Studies of this question remain to be done. But while there is no direct evidence of sleep selectively reducing the intensity of emotional memories, there is considerable evidence that sleep, and REM sleep in particular, can play a role in mood regulation. Studies of sleep and depression (Cartwright, Baehr, Kirkby, Pandi-Perumal, & Kabat, 2003; Cartwright, Luten, Young, Mercer, & Bears, 1998) have shown that REM sleep reduces depressed moods.

The mechanisms that underlie the selective retention of emotional memories remain unclear. Although one possibility is that the brain has evolved to specifically strengthen emotional memories during

sleep, it is also possible that this is simply a special case of the sleeping brain calculating which aspects of memories are most advantageous to the individual and preferentially strengthening those components. Indeed, sleep can even selectively enhance false memories when such memories represent a more useful, gistlike summary of larger bodies of information (Payne, Propper, Walker, & Stickgold, 2006). Interestingly, the sleep-dependent facilitation of these gist memories, like the development of insight described above, correlates with a decrease in slow-wave sleep (Payne et al., 2006), again suggesting a role for REM sleep in this process.

Sleep, Memory, and EMDR

How might these findings of sleep-dependent processing be relevant to EMDR? A recent study (Rasch, Buchel, Gais, & Born, 2007) suggests that reactivation of memories during sleep can enhance the sleep-dependent consolidation of those memories. In this study, subjects learned to find pairs of matched cards in an array of cards, similar to the children’s game of Concentration. Whenever a pair was successfully matched, one group of subjects was briefly exposed to the odor of roses. The following night, half the subjects were exposed to the rose scent during sleep. The following morning, those subjects exposed to the rose odor, both when learning the card pairs and during their sleep the next night, showed significantly better recall of the locations of matched card pairs.

Remarkably, this study demonstrated that exposing individuals to sensory cues related to a memory when the individual is in an appropriate brain/mind state leads to the effective processing of the entire memory. The similarity between this finding and the instructions, “Hold that image of the traumatic event in your mind and just watch my fingers moving back and forth,” is inescapable (Stickgold, 2007). If the bilateral stimulation of EMDR can, as previously argued, alter brain states in a manner similar to that seen during REM sleep (Stickgold, 2002), then there is now good evidence that EMDR should be able to take advantage of sleep-dependent processes, which may be blocked or ineffective in PTSD sufferers, to allow effective memory processing and trauma resolution.

Associative Networks in Sleep and EMDR

One of the striking features of REM-sleep-dependent memory processing is that networks of associated memories are activated differently during REM sleep.

Specifically, REM sleep appears to facilitate the activation of more distant associations than seen either in non-REM sleep or in the normal wake state. In one study, we awakened subjects during the night from either REM or light Stage-2 non-REM sleep and quickly (while their brains were still shifting back to the wake state) tested them on a semantic priming task, which measures the strength of verbal associative networks (Stickgold, Scott, Rittenhouse, & Hobson, 1999). When subjects were awakened from non-REM sleep, they displayed the same general activation patterns as they did during the day. But when we awakened them from REM sleep, normally strong associations (e.g., hot-cold) showed no signs of activation, and normally weak associations (e.g., thief-wrong) were highly activated (Stickgold et al., 1999). This apparent shift toward activation of distant associations could explain the uniquely bizarre nature of REM sleep dreams (Williams, Merritt, Rittenhouse, & Hobson, 1992).

In a second study, we tested subjects on their ability to solve simple anagrams (Walker, Liston, Hobson, & Stickgold, 2002) and found that they performed significantly better after awakening from REM than from non-REM sleep. These findings suggest that REM sleep facilitates the discovery of previously unrecognized connections between apparently unrelated memories. Finding such connections is perhaps the key feature of the creative process. But it is also an important goal of any form of psychotherapy. If EMDR activates these normally REM-dependent brain processes, one would expect to see it reflected in the increased intrusion of unexpected associative trains of thought.

A difference in attitudes toward such intrusions is a distinctive theoretical difference between how EMDR and exposure therapy approach the treatment of PTSD. While EMDR encourages exploration of such associative trains (the classic “stay with that” instruction), exposure therapy strives to keep the patient tightly focused on the central trauma memory. But the extent to which patients spontaneously bring up such unexpected associative trains during these two different forms of therapy is unknown. Interestingly, while the model of EMDR action proposed here would predict that outcome would correlate *positively* with the frequency and magnitude of such intrusions of weakly related memories, exposure therapy arguably would predict a *negative* correlation.

The Mechanism of Action of EMDR

The valuing of weakly related memory intrusions is just one distinguishing difference between EMDR and exposure therapy. But the most obvious difference is

the use of eye movements or other forms of bilateral stimulation, a feature of EMDR that has been ridiculed by some, who argue that EMDR adds little if anything to the benefits seen from simple exposure therapy. One author has gone so far as to warn readers, with all apparent seriousness, that, “One day, clinicians may find themselves in front of reasonable fellow citizens, having to explain why they waved fingers in front of a patient’s face” (Rosen, 1999). Although it can be frustrating to have to deal with such attitudes, the claim that EMDR is simply exposure therapy plus useless “waved fingers” is a testable hypothesis and can provide a framework within which to investigate EMDR’s mechanism of action. Recognizing that others have also addressed the question of how EMDR differs from exposure therapy (e.g., Rogers & Silver, 2002), I offer some hopefully novel and useful approaches to investigating these differences, while simultaneously investigating the mechanism of EMDR action.

How Does EMDR Differ From Exposure Therapy?

How Much “Exposure” Is There in EMDR Treatment?

Our “REM sleep mimicry” model does not place much value on extensive exposure to the original traumatic event(s). While activation of these memories would be critical for initiating their processing, the continuous reactivation of these memories would imply that the REM sleep mechanism has not been activated and that effective processing of the traumatic memories has failed. Thus, the amount of exposure should correlate *negatively* with outcome. In contrast, if EMDR’s efficacy depends on shared properties with exposure therapy, then EMDR should produce as much exposure, and arguably at the same density, as does exposure therapy, and outcome should correlate *positively* with exposure. It seems unlikely that EMDR produces as much exposure, both because of the relatively low number of EMDR sessions used for single-trauma PTSD treatment and because of the absence of the between-session “homework” incorporated in exposure therapy.

But these are all empirically testable hypotheses. One can review audio- or videotapes of complete sets of treatment sessions in both protocols, using a predetermined set of criteria that define what constitutes exposure, and standard instruments for measuring treatment outcome. Ideally, this would be a prospective study, with therapists who routinely use either EMDR or exposure therapy and with patients randomly assigned to one condition or the other. Patients could record their homework time and homework exposure time in logs. Treatment would

continue until a preset level of reduced symptoms was achieved or a maximum treatment time was reached. Exposure time averages for the two treatment protocols could then be compared for patients with equivalent outcomes, or outcome variables could be regressed against exposure time to test the hypothesis that EMDR requires significantly less classic exposure to produce benefits equivalent to those seen with exposure therapy. It is possible that such treatment tapes already exist, although an unbiased selection of tapes (i.e., not selected on the basis of how successful treatment was) would be critical, and ancillary information, such as exposure logs or standardized outcome measures, might not be available. Nevertheless, such a retrospective study could provide important information.

Prospective studies could be further enhanced by recording autonomic nervous system activity during sessions. For example, one might expect dramatically higher levels of sympathetic activation in exposure therapy than in EMDR. Wilson and colleagues have presented support for decreases in sympathetic arousal during EMDR (e.g., Wilson, Silver, Covi, & Foster, 1996), and comparing these to changes seen in exposure therapy treatment could provide important differentiation between the two therapies.

How Much Intrusion of Weakly Related Memories Is There in EMDR? Our REM sleep mimicry model suggests that the frequency and magnitude of intrusions of weakly related memories during EMDR sessions is a measure of the extent to which REM sleep mechanism are being activated and, hence, should correlate with treatment outcome. In addition, the model predicts that these intrusions should occur at much higher frequencies during periods of bilateral stimulation than during more conventional talk therapy between stimulus sets. In addition, the model predicts that intrusion frequency during periods of bilateral stimulation would greatly exceed that observed during exposure therapy sessions. Although the model is neutral on whether the frequency seen between EMDR stimulus sets would be greater than that seen during exposure therapy, we predict that it would be, based on the EMDR model being more accepting of such intrusions. It would also be worth examining whether there is any correlation, positive or negative, between such intrusions and outcome in exposure therapy. These, again, are testable hypotheses and questions, and the experimental designs described above for evaluating the extent of exposure could also be used to test the extent of intrusion by weakly related memories. If carefully designed, both questions could be answered with the same set of

treatment tapes. Methods would need to be developed for evaluating the degree of shift when new images, thoughts, or feelings arise in a session, but examples of such techniques exist (see, for example, Stickgold, Rittenhouse, & Hobson, 1994).

How Much Improvement Does Exposure Therapy Produce When Treatment Time Is Matched to That of EMDR Treatment? If EMDR's efficacy depends on shared properties with exposure therapy, then one would also predict that restricting exposure therapy to three 90-minute sessions with no homework would produce as much symptom improvement as an equivalent amount of EMDR. Again, a prospective study would be most powerful. (Treatment could continue after completion of the experimental protocol as appropriate.) But even using existing tapes to compare outcomes after three sessions each of EMDR and exposure therapy could provide definitive findings.

What Benefit Is Derived From the Eye Movements?

This, obviously, is the key question raised by EMDR, and it has been approached from various directions. Here I suggest two approaches.

How Do the Eye Movements of EMDR Affect Treatment and Treatment Outcome?

Several studies have attempted to look at how eye movements affect treatment outcome from a variety of perspectives. But to our knowledge, none have looked in detail at how the treatment itself is dependent on eye movements. For example, does the amount of exposure increase when bilateral eye movements are removed? Does the amount of intrusion of weakly related memories *decrease*? Both would be consistent with the REM sleep mimicry model.

In such studies, the design of the control condition is critical. Comparisons have been made to vertical eye movements and fixed-eyes conditions or to other forms of stimulation. But I would argue that none of these is an appropriate control. Most, and arguably all, proposed mechanisms of action of EMDR hypothesize that the bilateral stimulation results in an altered brain/mind state in which trauma processing is enhanced. Models may even differ on whether the stimulation necessarily must be bilateral. But obviously no one would propose that it is the contraction of the lateral and medial rectus muscles in the eye—the muscles responsible for the actual eye movements—that enhances trauma processing. Rather, these eye movements are presumed to trigger more global changes in the brain/mind state, which are in turn responsible

for the treatment benefits. This is why the issue of a control condition is so critical. For example, maintaining eye fixation for 30 seconds appears to produce a shift in mental state, a fact that can be confirmed by simply holding one's eyes fixed on any single word in this article for 30 seconds. If such a state shift also facilitated trauma processing, then its use as a control would reveal no relative benefit for bilateral movements, leading to a false rejection of their efficacy. Equally likely, the brain/mind state triggered by eye fixation might actively impair trauma processing, producing a false positive benefit for bilateral movements when compared to this control condition. Similar concerns surround the use of vertical eye movements. We would propose that the correct control conditions would be the absence of any intentional eye movements or nonmovements. Simply giving patients the standard instructions regarding how to hold an image while letting it change, and then using the classic "stay with that" instruction across processing sets, but giving no instructions regarding eye movements and giving no bilateral stimulation would arguably result in the cleanest excision of the eye movement component from the control condition. If such a control condition produced outcomes equivalent to those of standard eye movement treatment, we would be hard pressed to argue for a unique benefit of EMDR. But if a clear difference were seen, it would be equally difficult to argue against such a benefit.

Using such a protocol would produce two important confounds that would need to be carefully considered. First, one would want to match the duration of sets in the two conditions. One method would be to use a predetermined number of eye movements or an equivalent time without eye movements for all sets. But the end of each set is normally determined by watching the client's body language—for example, flushing in the face or changes in muscle tension or breathing. One could use such changes to mark the end of sets in both conditions, along with minimum and maximum durations. Although this would in many ways be preferable, it could lead to significant differences in average duration between the two groups, which would remain as a confound.

The second problem is potential treatment bias. Arguably, therapists could be unconsciously biased in the control, no-eye movement condition and provide less effective treatment, failing to optimally implement the other features of the EMDR protocol. Controlling for this would be a difficult task. But trained clinicians, unaware of the nature of the study, could be asked to review audiotapes of treatment sessions and rate treatment fidelity using fidelity

scales developed for the standard EMDR protocol. Minor edits of the tapes could remove references to eye movements and other features that might reveal the nature of the study or distinguish between treatment conditions.

How Do Bilateral Eye Movements Affect Memory Processing Outside of the EMDR Protocol?

We have argued that PTSD is fundamentally a memory disorder; the normal processing of traumatic memories, which leads over time to a reduction in and then elimination of the trauma-related characteristics of the memory, breaks down, and the memory retains its traumatic nature over extended periods. While some models of EMDR action (e.g., Wilson et al., 1996) predict that the impact of bilateral stimulation concurrent with memory reactivation might be limited to memories associated with strong autonomic activation, especially traumatic memories, others (e.g., Stickgold, 2002) predict that all memories should be affected. Christman, Propper, and colleagues have begun to examine the impact of bilateral eye movements on retention of simple verbal memory (Christman, Garvey, Propper, & Phaneuf, 2003) and have reported enhanced memory recall following horizontal saccadic eye movements.

More studies of this nature are needed. Similar studies could be conducted to examine whether such eye movements enhance activation of weak associative networks using the semantic priming (Stickgold et al., 1999) or anagrams (Walker et al., 2002) tests described above for studies conducted with REM and non-REM awakenings.

Other studies could look at the impact of eye movements on more complex forms of memory processing, such as those described earlier in this article that are also thought to be enhanced by REM sleep. If, as we have argued (Stickgold, 2002), trauma processing depends critically on the integration of the episodic memories of the traumatic event(s) into larger semantic memory networks, allowing the elaboration of an accurate personal meaning for the traumatic event(s) within the patient's sense of self, then looking at the impact of bilateral eye movements on memory tasks that require the integration of episodic memories into larger networks, such as the extraction of gist from complex memories or the recognition of patterns within large sets of stimuli, could further explicate the role of eye movements in EMDR and aid in the development and refinement of models that attempt to explain the physiological basis of EMDR's effectiveness.

Conclusion

Clinical practice has historically developed new stratagems for disease treatment far in advance of scientific understanding of their mechanisms. Perhaps most famously, the discovery of penicillin's antibiotic power by Fleming in 1928 preceded the 1965 report of its mechanism of action by almost 40 years. From that perspective, it is not surprising that we are still in the early stages of seeking the detailed mechanism of action of EMDR. But this does not diminish the need for, or value of, such an understanding. The clarification of the mechanism of action of penicillin led to the development of entirely new classes of antibiotics, and one might expect similar advances once EMDR's underlying mechanisms are similarly understood.

Finding EMDR's mechanism of action requires a multipronged approach. One approach is to seek possible mechanisms from a theoretical perspective. Given what we know about trauma, memory, and EMDR, what might explain the effects of EMDR? A second approach is to critically test the predictions of a specific model; are those of the REM sleep mimicry model confirmed by experiment? A third approach is the top-down dismantling of EMDR, to find what components provide the unique benefits of EMDR. And a fourth approach is a bottom-up investigation of the impact of eye movements or other forms of bilateral stimulation on basic neurophysiological systems and memory processing to identify the basic building blocks that might combine with other aspects of EMDR treatment to produce its remarkable efficaciousness. By combining these varied approaches, and by being patient, the brain and body mechanisms underlying EMDR will eventually be identified.

References

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington DC: Author.

Brewin, C. R., Andrews, B., & Valentine, J. D. (2000). Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. *Journal of Consulting and Clinical Psychology, 68*(5), 748–766.

Cartwright, R., Baehr, E., Kirkby, J., Pandi-Perumal, S. R., & Kabat, J. (2003). REM sleep reduction, mood regulation and remission in untreated depression. *Psychiatry Research, 121*(2), 159–167.

Cartwright, R., Luten, A., Young, M., Mercer, P., & Bears, M. (1998). Role of REM sleep and dream affect in overnight mood regulation: A study of normal volunteers. *Psychiatry Research, 81*(1), 1–8.

Christman, S. D., Garvey, K. J., Propper, R. E., & Phaneuf, K. A. (2003). Bilateral eye movements enhance the retrieval of episodic memories. *Neuropsychology, 17*(2), 221–229.

Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin, 99*(1), 20–35.

Loftus, E. F., Loftus, G. R., & Messo, J. (1987). Some facts about “weapon focus.” *Law and Human Behavior, 11*, 55–62.

Marks, I. (1979). Exposure therapy for phobias and obsessive-compulsive disorders. *Hospital Practice, 14*, 101–108.

Payne, J. D., Propper, R., Walker, M. P., & Stickgold, R. (2006). Sleep increases false recall of semantically related words in the Deese-Roediger-McDermott memory task. *Sleep, 29*, A373.

Payne, J. D., Stickgold, R., Swanberg, K., & Kensinger, E. A. (2008). Sleep preferentially enhances memory for emotional components of scenes. *Psychological Science, 19*, 781–786.

Rasch, B., Buchel, C., Gais, S., & Born, J. (2007). Odor cues during slow-wave sleep prompt declarative memory consolidation. *Science, 315*(5817), 1426–1429.

Rogers, S., & Silver, S. M. (2002). Is EMDR an exposure therapy? A review of trauma protocols. *Journal of Clinical Psychology, 58*, 43–59.

Rosen, G. M. (1999). Treatment fidelity and research on eye movement desensitization and reprocessing (EMDR). *Journal of Anxiety Disorders, 13*(1–2), 173–184.

Shapiro, F. (1995). *Eye movement desensitization and reprocessing: Basic principles, protocols, and procedures*. New York: Guilford Press.

Smith, C., & Smith, D. (2003). Ingestion of ethanol just prior to sleep onset impairs memory for procedural but not declarative tasks. *Sleep, 26*(2), 185–191.

Stickgold, R. (2002). EMDR: A putative neurobiological mechanism of action. *Journal of Clinical Psychology, 58*, 61–75.

Stickgold, R. (2005). Sleep-dependent memory consolidation. *Nature, 437*, 1272–1278.

Stickgold, R. (2007). Of sleep, memories and trauma. *Nature Neuroscience, 10*(5), 540–542.

Stickgold, R., Rittenhouse, C. D., & Hobson, J. A. (1994). Dream splicing: A new technique for assessing thematic coherence in subjective reports of mental activity. *Consciousness and Cognition, 3*, 114–128.

Stickgold, R., Scott, L., Rittenhouse, C., & Hobson, J. A. (1999). Sleep induced changes in associative memory. *Journal of Cognitive Neuroscience, 11*, 182–193.

van der Kolk, B. A. (1994). The body keeps the score: Memory and the evolving psychobiology of posttraumatic stress. *Harvard Review of Psychiatry, 1*, 253–265.

Wagner, U., Gais, S., Haider, H., Verleger, R., & Born, J. (2004). Sleep inspires insight. *Nature, 427*(6972), 352–355.

Walker, M. P., Liston, C., Hobson, J. A., & Stickgold, R. (2002). Cognitive flexibility across the sleep-wake cycle: REM-sleep enhancement of anagram problem solving. *Cognitive Brain Research, 14*, 317–324.

Walker, M. P., & Stickgold, R. (2006). Sleep, memory, and plasticity. *Annual Reviews in Psychology*, *57*, 139–166.

Williams, J., Merritt, J., Rittenhouse, C., & Hobson, J. A. (1992). Bizarreness in dreams and fantasies: Implications for the activation-synthesis hypothesis. *Consciousness and Cognition*, *1*, 172–185.

Wilson, D. L., Silver, S. M., Covi, W. G., & Foster, S. (1996). Eye movement desensitization and reprocess-

ing: Effectiveness and autonomic correlates. *Journal of Behavior Therapy and Experimental Psychiatry*, *27*(3), 219–229.

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