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# EMDR: A Putative Neurobiological Mechanism of Action



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Numerous studies have provided evidence for the efficacy of eye movement desensitization and reprocessing therapy (EMDR) in the treatment of posttraumatic stress disorder (PTSD), including recent studies showing it to be more efficient than therapist-directed flooding. But few theoretical explanations of how EMDR might work have been offered. Shapiro, in her original description of EMDR, proposed that its directed eye movements mimic the saccades of rapid eye movement sleep (REM), but provided no clear explanation of how such mimicry might lead to clinical improvement. We now revisit her original proposal and present a complete model for how EMDR could lead to specific improvement in PTSD and related conditions. We propose that the repetitive redirecting of attention in EMDR induces a neurobiological state, similar to that of REM sleep, which is optimally configured to support the cortical integration of traumatic memories into general semantic networks. We suggest that this integration can then lead to a reduction in the strength of hippocampally mediated episodic memories of the traumatic event as well as the memories' associated, amygdala-dependent, negative affect. Experimental data in support of this model are reviewed and possible tests of the model are suggested. © 2002 John Wiley & Sons, Inc. *J Clin Psychol* 58: 61–75, 2002.

Keywords: EMDR; REM sleep; episodic memory; memory systems

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In 1989, Shapiro described her initial “walk in the park,” during which she noticed her eyes making spontaneous saccadic bursts to the upper right when thinking of disturbing thoughts. She proposed (Shapiro, 1989a) that these saccadic eye movements were linked to the alterations in information processing she perceived within her mind. In early EMDR treatments, Shapiro intentionally reproduced these saccadic eye movements, with patients instructed to shift their gaze back and forth between two fingers held up in front of the patient on either side of the midline or with extremely rapid contralateral movements (Shapiro, 1989a), which were most likely saccadic in nature (Welch, 1996).

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Because EMDR assigns a causal relationship between these eye movements and clinical improvement, their precise nature has come under considerable scrutiny. Whether they are true saccades or, alternatively, smooth pursuit eye movements has been the subject of several papers (Rosen, 1995, 1997; Welch, 1996), although the initial instructions called for 4 sweeps per second, a rate that would likely require saccadic movements. Although no quantitative studies have measured the actual eye movements of EMDR, the question, as noted by Welch (1996), has, in fact, become moot, since these presumably saccadic eye movements of the initial EMDR treatments are now often replaced with smooth pursuit eye movements, bilateral tones, and bilateral tapping (Shapiro, 1995, 1999), and many of the published studies have also utilized these smooth pursuit eye movements (e.g., Marcus, Marquis, & Sakai, 1997; Rothbaum, 1997; Scheck, Schaeffer, & Gillette, 1998; S.A. Wilson, Becker, & Tinker, 1997). Thus an explanatory model must explain how saccadic and smooth pursuit eye movements as well as bilateral stimulation in the auditory and tactile domains might facilitate the treatment of PTSD.

Before describing our model, we want to make several points clear. First, we do not intend to review the literature demonstrating the efficacy of the EMDR method (for reviews, see Servan-Schreiber, 2000; Shapiro, 1999) or, for that matter, the literature questioning its efficacy (Cahill, 2000; McNally, 1999). This is a rapidly growing literature, and we leave it to other articles in this issue to address this question. Second, we are not claiming that we have solid evidence for all of the links and interpretations in the train of logic presented here. We are not claiming to "prove" through a deductive scientific process that EMDR works. Instead, our goal is to demonstrate that there is a reasonable explanation of how EMDR works, which is consonant with modern neurobiology and cognitive neuroscience and which provides a basis for future studies of the mechanism of action of EMDR as well as of other approaches to PTSD, such as therapist-directed flooding (Steketee & Foa, 1987). For us, this represents the first step toward an understanding of the mechanism of action of EMDR in specific and, possibly, toward a greater understanding of PTSD in general.

The model is presented in three sections. The first describes the normal life of memories of everyday events and how this relates to PTSD. Sensory images pass through several representational systems in the brain, all of which appear capable of holding at least temporary memories of the images, before being encoded in the hippocampal complex, from whence recall can be readily triggered. How these various systems encode information, and how their relative impact changes over time, is the focus of this first section. The second section of the model describes possible roles of sleep in the slow changes in memories that occurs over days to years. The neurochemical and neurophysiological state changes that occur on entry into REM sleep are described, and their possible role in memory reprocessing described.

Finally, the third section proposes how EMDR might serve to bypass a PTSD-induced breakdown in sleep-dependent memory reprocessing. By inducing neurophysiological and neurochemical changes during the therapeutic session that mimic those seen in REM sleep, the effective integration of traumatic episodic memories into semantic memory networks is achieved. As a consequence, the hippocampal episodic memories and associated affect are believed to be weakened or eliminated, leading to the alleviation of the symptoms of PTSD.

### The Formation, Transfer, and Integration of Episodic Memories

#### *Formation of Memories*

We remember remarkably little of what we experience, surely only a fraction of a percent by the most generous of measures. If one takes an average 40-year-old, she will have had

approximately a quarter of a million hours of waking experience. But she clearly would not have even close to a thousand hours of memories recallable in the full detail of their original sensorimotor and emotional richness. Instead, her brain will have attempted to extract, abstract, and store the critically useful information contained in all of these hours of experience. Thus she remembers what  $5 + 5$  equals, and what  $6 \times 4$  equals, but not the hours and months she spent rehearsing these in school and at home. She might have a general memory of “learning math” in school, and even a specific memory of an event or two during her childhood schooling in math. But if you asked her where she was, and who was with her, and how she felt when she learned that  $3 + 3 = 6$ , she would rightly look at you baffled. We lose these episodic memories and retain only the “general knowledge” or “semantic content” of the experience. What has only recently become clear is that this semantic memory is not simply a degraded version of the original episodic memory. Instead, it is a separately stored memory that, over time, has been extracted and abstracted from the initial episodic memory.

The existence of multiple memory systems has only been described in the last 10 to 15 years (Schacter & Tulving, 1994). But it is now clear that there are separate and distinct memory systems that store information in different formats and in different parts of the brain. For our purposes here, there are three critical systems—the perceptual representation system (Schacter & Tulving, 1994), the episodic memory system (Squire, 1992), and the semantic memory system (Schacter & Tulving, 1994).

Information from the outside world passes first through unimodal sensory cortices that produce separate internal representations of a stimulus in each sensory modality. Visual, auditory, tactile, and olfactory inputs are each processed by their respective regions of unimodal sensory cortex, and then passed on to higher processing regions. By this time, conscious perception of the sensations has occurred. But even after the information is passed on, a residual “trace” of the information, a “perceptual memory,” is retained within the sensory cortex, and although normally short-lived and outside our conscious awareness, such memory traces can be probed and utilized for varying lengths of time after the initial sensory perception has ended (Schacter, Chiu, & Ochsner, 1993). This simple pathway, from sensation to perception, is shown across the top of Figure 1.

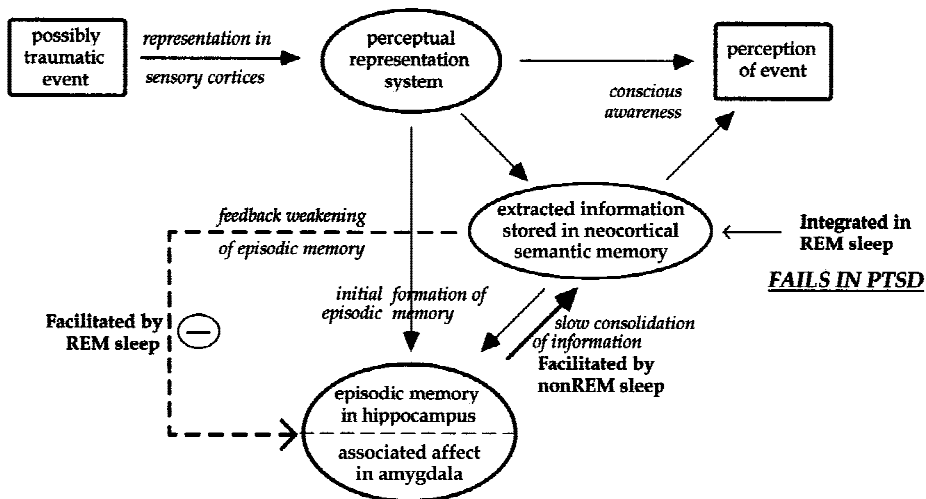


Figure 1. General model for sleep-dependent transfer and integration of episodic memories.

As the information flows through the perceptual representation system, a memory of the sensation is formed within it. In fact, these memories can form even with subliminal stimuli that never reach our conscious awareness. From the unimodal sensory cortices, information flows in complex and interwoven pathways. One set of pathways carries information to regions of association cortex where meaning is ascribed to the perception. For visual information, this branch follows the ventral “what” pathway to the temporal lobe, where an object is identified and then, with the help of language areas, named (Ishai, Ungerleider, Martin, Schouten, & Haxby, 1999). Similar pathways allow identification of sounds heard or objects touched.

Such identifications access neocortical semantic memory networks within which such words and concepts as “baby,” “car,” “blue,” and “loud” are stored. When a baby is seen, memories of both the word and the concept become activated, and the semantic meaning of “baby-ness” is recalled. Here again, the activation of these networks is not without lasting effects, and the concept of “baby” is imperceptibly altered to incorporate this new example of a baby (Figure 1, middle). Using these two systems, we consciously see and identify the baby. But by themselves, neither of these two memory “traces” is strong enough to permit their recall even five minutes later.

For long-term memories to form, the hippocampal complex is required. At the same time, information from both perceptual and semantic representations flow into the hippocampal complex. The hippocampus serves two major functions (McClelland, McNaughton, & O’Reilly, 1995). First, while the memory traces formed in the perceptual and semantic memory systems in the cortex are too weak to support direct recall, the hippocampus can form a memory of an event that supports its long-term recall. It is because of the creation of these “hippocampal memories” that we are able to recall the events of the day and to remember and subsequently recall phone numbers and names heard only once. Thus, in the minutes and hours after experiencing an event, only the hippocampal memory can initiate the intentional recall of the event.

But the details of the memory are not in the hippocampus. Instead, the hippocampus stores memories as a set of “pointers” to the information stored in other systems. These are thought to include links to all sensory modalities activated during the event, any semantic memories initially activated by the sensory input, and any emotional response to it. Thus a hippocampal memory is both strong and integrated. But these memories are more than just flashbulb images of our surroundings. They are memories of sensations and actions over time, a movie-like memory of an episode from our lives, linking together stored information not only across different memory storage systems, but over time as well.

### *Episodic Memories*

Of necessity, I have been somewhat loose in all of these descriptions, perhaps most importantly in relation to the concept of memories being “stored” in the hippocampal complex. In reality, the concept of memory storage is at best metaphorical. What occurs in reality when we “store a memory” is that we simply alter a system so that a certain pattern of brain activity and hence perception or thought is more likely to be reinstated in the future (McClelland et al., 1995). An analogy might be to say that as a forest develops game trails, it is “remembering” and “storing” memories of all the animals that have passed through it, whereas in fact it has simply been altered by the passage of animals in a way that increases the likelihood that future animals will follow the same paths. The conscious recollection of hippocampal memories occurs when the hippocampus facili-

tates the simultaneous reactivation of numerous weak cortical traces, some going to the perceptual representation system, some to the semantic memory system, and possibly others to the amygdala for associated affect. Thus, the hippocampal formation points one to a set of specific trails, mostly to the cortex, and whether the memory is “stored” in the hippocampus or the cortex or the amygdala may simply be a matter of where we choose to focus our interest. But without the contextual integration (Nadel & Moscovitch, 1998) provided by the hippocampus, recalling memories would be either impossible (as in amnesic patients; Scoville & Milner, 1957; Squire, 1992) or highly fragmented (as described in some PTSD patients; van der Kolk, 1994).

A second critical difference, both structurally and functionally, between the cortical and hippocampal memory systems is the density of memory storage. Within the hippocampus, memories are stored sparsely, with minimal overlap between the networks of cells encoding a single memory (McClelland et al., 1995). This has two functional consequences. First, it is feasible to form a strong, recallable memory trace with a single activation of the network. If one does not have to worry about other trails in the forest that must be preserved, one can be heavy handed in creating a new path. In contrast, if there is a dense network of preexisting paths, so that the new path has to cross hundreds or even thousands of preexisting game trails without destroying them, a slower, more complicated and delicate process must be used. Because of its sparse representations, the hippocampus can bulldoze new roads without significant side effects. The second functional consequence is related. When you can use a bulldozer to clear a path, it remains discrete and isolated. As a result, it is easy to follow and you do not confuse it with other trails elsewhere in the forest; when you start down a path, there is only one place you are likely to end up. Thus, episodic memories tend to be rapidly formed, strong, clear, and unambiguous (see Table 1) (McClelland et al., 1995).

### *Semantic Memories*

In the neocortex, memories are stored in dense, highly overlapping neural networks. The same neurons participate in large numbers of memory traces, and it is only the overall activation pattern that determines which particular memory is recalled (McClelland et al.,

Table 1  
*Characteristics of Episodic and Semantic Memory*

	Episodic Memories	Semantic Memories
Memory content	isolated memories of distinct events	general knowledge abstracted from episodic memories and integrated with other semantic memories
Brain localization	hippocampus, medial temporal lobe	neocortex
Storage density (overlap, ambiguity)	sparse	dense
Initial strength	high	low
Speed of consolidation	rapid	slow
Longevity	relatively short (but can be years)	relatively long (but can slowly disappear with disuse)

1995). By our previous analogy, new trails are formed in your “cortical forest” not by rapid bulldozing of a path, but by each nascent trail being gently passed over hundreds or thousands of times, while other, crossing trails are similarly retraced. This process permits each trail to be laid down without obscuring those that were already there and without blocking the formation of other new trails. In an analogous manner, episodic memories are slowly transferred to cortical, semantic memory. By a process that McClelland and his colleagues (1995) have referred to as “interleaved replay,” hippocampal memories are slowly and repetitively replayed from the hippocampal complex to the cortex, where the memories are eventually incorporated into your general semantic knowledge. Thus, cortical memories, in contrast to hippocampal memories, are slowly formed but are densely represented (see Table 1; McClelland et al., 1995). They are also highly interconnected. Starting down one path, it is easy to be distracted onto another, and in the end the most frequently followed “path” might in fact be constructed from pieces of several different original paths. It is this integration of separate, related memory traces that forms the basis for semantic knowledge, meaning, and understanding.

### *Integration of Memories*

Perhaps the most difficult computational task that the brain faces is that of integrating memories. This wide range of activities varies immensely in its scope. At the simplest level, the brain takes several views of a single object and classifies them as alternative representations of the same object, or it takes several images of similar objects and defines them as members of a single class of objects. Although not a trivial task, computers can perform these functions, learning to abstract the essence of tree-ness from pictures of multiple trees, or of dog-ness from many dogs. But at its most complex, the integration of disparate memories leads to artistic and scientific creativity—the associating of objects and concepts in new, unexpected, but meaningful patterns. In between these extremes, memory integration serves to create meaning and an understanding of oneself within the context of the larger world. These are tasks well beyond the capabilities of modern artificial intelligence software.

How does this integration occur? The details of these processes are poorly understood at best. But it clearly occurs within regions of association cortex, and involves the formation and strengthening of connections between networks encoding related memories. While the simpler components of this process can undoubtedly occur in “real time,” during acts of perception, the more complex components of this integrative process appear to occur most consistently when the brain/mind is “off-line,” during reminiscence and, as we shall show below, during sleep (Stickgold, 1998).

### *The Weakening of Episodic Memories*

The final step in the consolidation and integration of semantic memories in the neocortex is the least well understood. Amnesic patients show a pattern of retrograde amnesia, whereby events from immediately prior to the onset of the amnesia are completely lost, and there is then a graded amnesia that can go back to several years before the precipitating event (McClelland et al., 1995; Scoville & Milner, 1957; Squire, 1992). But when one looks further and further back, more and more memories are spared. Eventually, when cortical memory traces become sufficiently strong, we no longer need our hippocampal memories. Evidence from rat studies suggests that as hippocampal memories are transferred to and integrated into semantic memory networks, the hippocampal traces are

actually weakened (Poe, Skaggs, Barnes, & McNaughton, 1997), presumably weakening associated affect as well (Figure 1, dashed line). Thus we learn general lessons from specific instances and quickly forget the actual events. In this way, space is freed up in the hippocampus, permitting the maintenance of this system as a sparse representational network.

### *Memory and PTSD*

PTSD is, at its core, a consequence of failed memory processing, characterized in part by the prolonged and inappropriate dominance of specific episodic memories of traumatic events. We suggest that PTSD, as opposed to simple trauma, arises when the brain fails to appropriately consolidate and integrate the episodic memory into the semantic memory system and, as a result, associations between the event and other, related events fail to develop. The breakdown of this normal process of memory transfer and integration leads to the continued maintenance of the episodic memory and its affect in an inappropriately strong and affect-laden form.

### *Summary*

In summary, declarative memories, those which we can “remember,” are initially stored in the hippocampus and related limbic brain structures as “episodic memories.” In the case of emotional events, the amygdala links the episodic memory to these emotions. Thus, when an event is recalled, the original sensations and emotions are both replayed. Over time, relevant information about the event is extracted from this memory and transferred to semantic memory networks located in the brain’s neocortex. Here, the information is integrated into the individual’s store of general knowledge and becomes available for understanding events in the future. Once this transfer has been accomplished, the hippocampal memory is largely obsolete, and both the memory trace and its links to associated affect can be forgotten, freeing up memory for storing future episodic memories. (What information is stored in the amygdala, and how it evolves over time, is an unresolved question. For a different perspective, see Armony & LeDoux, 1997.)

Occasionally, however, the extraction process fails and information from the episodic memory fails to be extracted, transferred, and integrated into the neocortex. In such instances, the individual fails to “learn from the event,” and the weakening and elimination of the episodic memory and its associated affect that normally follows this transfer and integration also fails to occur. If the episodic memory is a traumatic one, the consequence is PTSD. Although the patient can minimize the consequences of this failure either by avoiding stimuli that would reactivate the memory (avoidance of stimuli) or by generally blocking emotional responses to stimuli (numbing of responses), true recovery requires the reestablishment of these failed processes of cortical memory consolidation and integration.

### Sleep and Memory Consolidation

When does this transfer and integration of episodic memories into the neocortex occur? Since these processes use the same brain regions normally used for processing incoming sensory information and creating new memories, it would make sense to run them during sleep, when sensory input is shut down. Numerous studies have shown that sleep can play a critical role in the process of memory consolidation, and we have reviewed this

literature in detail elsewhere (Stickgold, 1998; Stickgold, Hobson, Fosse, & Fosse, 2001). Several lines of evidence suggest that REM and non-REM sleep serve related but distinct functions in off-line memory reprocessing (Table 2). Non-REM sleep appears most critical for strengthening of hippocampal memories and REM sleep for neocortical memories (Plihal & Born, 1997). In rats, information flows out of the hippocampus and into the cortex during non-REM sleep, then reverses direction during REM sleep (Buzsáki, 1996). In addition, semantic memory in humans preferentially activates weak associations in REM sleep, but strong ones in non-REM sleep (Stickgold, Scott, Rittenhouse, & Hobson, 1999). Regional brain activation is also dramatically different in the two sleep states, with limbic and sensory cortices preferentially activated in REM sleep (Hobson, Stickgold, & Pace-Schott, 1998). Finally, the central brainstem neuromodulation of the brain shifts dramatically, from domination by serotonin and norepinephrine during non-REM sleep (Aston-Jones & Bloom, 1981; Portas et al., 1998) to domination by acetylcholine during REM sleep (Kametani & Kawamura, 1990; Marrosu et al., 1995).

### *Sleep, Dreams, and Memories*

Many of the distinctive characteristics of dreams can be understood in light of REM sleep physiology. The bizarre, hyperassociative nature of dreams may result from the preferential activation of weak semantic associates combined with the lack of input from the hippocampus. As a result of the preferential activation of weak associations, dream images unpredictably juxtapose only barely related objects, characters, and locations, proceeding in illogical sequences. Without the spatial and temporal coherence that the hippocampus normally provides to our thoughts and images, these bizarre hyperassociations often appear to float free of specific anchors in space and time. Hyperactivation of limbic cortices and the amygdala could similarly explain the hyper-emotional aspect of dreaming (Merritt,

Table 2  
*Characteristics of Waking, REM, and Non-REM Sleep*

	Waking	Non-REM Sleep	REM Sleep
Mental content	logical, progressive, based on external sensory input, episodic and semantic memory	logical, perseverative, based on semantic and episodic memory and strong associations	illogical, progressive, based on semantic memory and weak associations
Brain serotonin, norepinephrine	variably medium to high	medium to low	near zero
Brain acetylcholine	variably medium to high	low	very high
Hippocampal-neocortical communication	bidirectional	Hippocampus → neocortex	neocortex → hippocampus
Memory function	formation of episodic memory	transfer of episodic to semantic memory	integration of semantic memory, feedback to hippocampus
Circadian peak	day time	early night	late night
Activation of associative networks	strong > weak > 0	strong > weak ≈ 0	weak > strong ≈ 0



Stickgold, Pace-Schott, Williams, & Hobson, 1994). Finally, the cessation of inputs from the hippocampus would seem to necessitate the focus of dream content on symbols and meanings—these and perceptual memories are the only sources of information out of which the dreams can be constructed. All of these results point toward complex memory reprocessing and integration during REM sleep.

The effect of these shifts in cortical processing during REM sleep are profound. The preferential activation of weak associative links with the neocortex enhances the testing of semantic associations most likely to result in the “discovery” of valuable new relationships between older memories. In addition, while hippocampal outflow to the cortex during non-REM sleep may serve to reinforce old memories, blocking hippocampal outflow during REM will help prevent semantic associations from falling back into more predictable, over-learned patterns and will favor the formation of new associative links necessary for understanding the meaning of events in our lives.

### *Sleep and PTSD*

There is a striking parallel between these physiological processes and the psychological theories implemented by therapists in the treatment of PTSD. For example, the therapist knows that progress cannot be made with the PTSD patient until she is able to discuss the traumatic event without replaying the episodic memory with its sensory and affective intensity. Only when these images are no longer intrusive can she integrate the event into her life, come to understand it, discover what it means for her, and thereby come to terms with it. Thus, a therapist strives, over time, to help a patient carry out these processes in the office—exposure therapy, for example may so overstimulate the episodic memory as to leave it refractory, allowing the therapist to help the patient integrate the experience cortically without interference from hippocampal intrusions. But this equally well describes the physiological processes of REM sleep—integration of the extracted and abstracted core of episodic memories into cortical, semantic memory networks, unimpeded by intrusive, hippocampal replay of the episodic memories. In both models, it is this integration that subsequently leads to the reduction in symptoms. What we now propose is that sleep, and particularly REM sleep, have evolved to provide a privileged time during which the brain/mind is optimally tuned for exactly this type of memory transfer and integration.

When traumatic episodic memories are repetitively replayed in sleep, it is an indication that this system has broken down. Outside of PTSD, episodic memories are almost never replayed veridically in dreams (Stickgold et al., 2001). Although dreams contain “day residue,” this is usually in the form of factoids, not contextually accurate images or stories. Day residue enters our dreams as a character or phrase from the day, as an emotion or similar situation. But it does not appear as a replay of an actual event. For such a replay to occur would require the breakdown of the normal blockade of hippocampal outflow to the cortex, which, we propose, prevents the normal integration and subsequent weakening of the episodic memory. It is this sequence of events that we believe leads to PTSD.

### *The Physiology of REM Sleep and PTSD*

What could cause this disruption of sleep physiology? The sleep of PTSD sufferers is known to be more fragmented than normal and they appear to retain an inappropriate level of vigilance even while asleep (Mellman, Kumar, Kulick-Bell, & Nolan, 1995; Mellman, 1997). The amount of REM sleep may be reduced as well (Lavie, Hefez, Halperin, & Enoch, 1979; Hefez, Metz, & Lavie, 1987; Glaubman, Mikulincer, Porat,

Wasserman, & Birger, 1990; but see Ross et al., 1994; Mellman, Kulick-Bell, Ashlock, & Nolan, 1995). Both of these phenomena could be explained by increased release of adrenal adrenaline or brainstem norepinephrine (NE) sufficient to produce a hyperaroused and hypervigilant condition with disrupted sleep. But NE also regulates the REM–non-REM cycle. During REM sleep, activity in the locus coeruleus and dorsal Raphe nucleus, brainstem structures which control levels of NE and serotonin in the brain, normally ceases (Hobson, McCarley, & Wyzinski, 1975). Failure to shut down these systems would lead to a dissociated neuromodulatory state and incomplete entry into REM sleep. Under such circumstances, we would expect associative processes to shift toward stronger associations and away from the weak associates normally activated during REM. They could also lead to a breach of the blockade of information flow from the hippocampus to the cortex, permitting the replay of traumatic memories.

Thus, the appearance of PTSD may result at least in part from the inability of the brain to inhibit norepinephrine or serotonin release during REM sleep. There is considerable evidence for elevated norepinephrine levels in PTSD patients both during wake (Bremner, Southwick, & Charney, 1999) and sleep (Mellman, Kumar, et al., 1995). Such an increase during sleep would block the activation of weak associations in the cortex (Stickgold et al., 1999), necessary for the integration of atypical (e.g., traumatic) memories into normative association networks, and would also disinhibit the blockade of hippocampal outflow (Buzsáki, 1996), leading to recurring reenactments of traumatic memories. With integration blocked, there would be no feedback to the hippocampus, and hence the hippocampus would fail to be sent instructions to weaken the episodic memory of the traumas and its associated negative affect. The consequence of this chain of events would be the self-sustaining condition of PTSD.

Brain imaging studies provide additional support for the concept that PTSD and REM sleep share functional pathways in the brain. Brain imaging studies have shown alterations in the activity of the hippocampus, amygdala, anterior cingulate, and possibly orbital frontal cortex, and visual cortex when PTSD patients are provoked with script driven imagery (Rauch et al., 1996; Shin et al., 1997). Yet these are precisely the brain regions that imaging studies suggest are activated during REM sleep (Hobson et al., 1998). Thus, the specific brain regions affected by restimulation of traumatic imagery in PTSD are the same ones activated during REM sleep. These findings support both the concept that traumatic memories are reprocessed during REM sleep and the hypothesis that PTSD may alter the normal functioning of the brain during REM sleep.

#### The Mechanism of Action of EMDR

How might EMDR contribute to recovery from PTSD? If the particular physiological state encountered during REM sleep is supportive of the memory integration necessary for recovery, then it is not unreasonable to conclude that interventions which shift the brain toward this state likewise would be beneficial. The question then is how EMDR might produce such a shift. Repeated saccadic eye movements could arguably “push-start” brainstem REM-induction mechanisms (Nelson, McCarley, & Hobson, 1983) through the reciprocal pathways that normally lead to the generation of rapid eye movements during REM sleep. Indeed, human brain imaging studies (Hong, Gillin, Dow, Wu, & Buchsbaum, 1995) indicate that eye movements in both REM sleep and wakefulness activate similar cortical areas. But visual pursuit and bilateral auditory and tactile stimulation require a different mechanism.

What all these techniques have in common, including those that utilize saccadic eye movements, is a pattern of alternating, bilateral stimulation that forces the subject to

constantly shift her attention across the midline. It is this orienting response (Sokolov, 1990) that we propose induces a REM-like state, facilitating cortical integration of traumatic memories.

The reorienting of attention can be triggered either intentionally, as when you choose to look at an object, or automatically, when a sudden movement “catches” your attention. This reorienting of attention requires first a release of focus from one location in visual space, then its shift to a new location, and finally its refocusing on this new location (Posner, 1980).

In extreme cases, for example a sudden loud noise, the automatic shift of attention is accompanied by an autonomic startle reaction (Koch, 1999). This startle response is biphasic, with an initial cholinergic activation that slows the heart (Fendt & Koch, 1999) and may also cause an automatic release of attention from its current focus (Davidson, Cutrell, & Marrocco, 1999). A fraction of a second later, release of adrenaline and nor-epinephrine causes the more familiar increase in heart rate, sweating, and muscle tensing associated with the adrenergic fight-and-flight response. But the brainstem surge of nor-epinephrine release also serves to shift and refocus the just-released attention to the location of the startling sound (Clark, Geffen, & Geffen, 1987).

What is most striking, aside from the momentary cholinergic activation reminiscent of REM sleep, is that the brainstem initiates a burst of “pontogeniculooccipital” (PGO) waves (Callaway, Lydic, Baghdoyan, & Hobson, 1987) in concert with the startle response (Bowker & Morrison, 1976). The only other condition known to generate PGO waves is REM sleep itself (Brooks & Bizzi, 1963). In fact, the neuronal circuits in the pontine brainstem that initiate PGO waves appears to control the REM–non-REM cycle as well. Local injection of acetylcholine into this PGO-generator brain region in the resting cat induces immediate and long-lasting REM sleep (Baghdoyan, Lydic, Callaway, & Hobson, 1989). Thus, inducing a startle response leads to activation of brainstem circuits that can also initiate REM sleep.

Cognitive tests have suggested that distinct brain systems are responsible for the release, shift, and then refocusing of attention of the orienting response (Posner & Dehaene, 1994). Among regions postulated to be involved are the anterior cingulate, discussed above in relation to both sleep and PTSD, and the superior colliculus, which controls eye movements, and which is activated by the PGO waves of REM sleep (Cespuglio, Laurent, & Calvo, 1976; Nelson et al., 1983). In addition, evidence suggests that cholinergic increases or noradrenergic decreases facilitate the release of attention prior to the shift (Clark et al., 1987; Davidson et al., 1999). Thus it seems reasonable to suggest that having a subject repetitively reorient her attention from one location to another could produce shifts in regional brain activation and neuromodulation similar to those produced during REM sleep.

In support of this hypothesis, Levin, Lazrove, and van der Kolk (1999) saw activation of the anterior cingulate and left frontal cortex following EMDR treatment, and Wilson, Silver, Covi, and Foster (1996) found decreased galvanic skin responses, suggestive of reduced adrenergic drive, with EMDR. It is precisely such shifts that we propose permit the changes in memory processing by which EMDR facilitates treatment of and demonstrably speeds recovery from PTSD.

In summary, our model proposes that the constant reorienting of attention demanded by the alternating, bilateral visual, auditory, or tactile stimuli of EMDR automatically activates brain mechanisms which facilitate this reorienting. Activation of these systems simultaneously shifts the brain into a memory processing mode similar to that of REM sleep. This REM-like state permits the integration of traumatic memories into associative cortical networks without interference from hippocampally mediated episodic recall. From

a psychological perspective, the patient becomes able to see the significance and meaning of the event in terms of their overall life, and thereby to “come to terms” with the traumatic event. Once successfully integrated, corticohippocampal circuits induce the weakening of the traumatic episodic memory and its associated affect.

But EMDR can work even better than REM sleep for two specific reasons. First, unlike REM sleep, when frontal lobe activity is largely inhibited (Hobson et al., 1998), during EMDR treatment the patient can choose the material to hold in mind at the start of the bilateral stimulation, and thereby bias the information that will be processed. Although the selection of associations is largely automatic and unintentional, holding a specific image in mind at the start of the stimulation assures that the associations, however weak and tangential, will most likely be related to the original image. Second, through careful management by the therapist, levels of anxiety and fear during EMDR treatment largely can be maintained at low levels, enhancing the ability of the bilateral stimulation to produce the desired physiological and neurochemical shifts in the brain without interference from increased NE levels. In this manner, our model suggests that EMDR specifically reverses the breakdown of normal memory processing that initially leads to the development of PTSD. But, of course, this must be only a portion of the story.

In concluding, we wish to make clear that we are not suggesting that the breakdown in this memory processing pathway is either absolutely necessary or sufficient to produce and sustain PTSD, but rather that it plays an important role in the disease process, and, more importantly, that restoring the normal activity of this pathway can aid in recovery from PTSD. For example, breakdowns at other points in the process, such as the formation of the contextual pointers that encode the episodic memory, might explain the associated phenomenon of recall of traumatic memories through only a small number of sensory modalities (van der Kolk, 1999).

Nor are we the only ones to propose neurobiological or psychological models for the action of EMDR. Most obviously, this model bears a strong resemblance to that originally put forward by Shapiro (1989a, 1989b, 1995). She proposed a model of “accelerated information processing” (Shapiro, 1995, p. 28) that involved the formation of connections “to appropriate associations” (p. 29), and went on to suggest that the breakdown of this normal process might result from excessively high levels of norepinephrine and that the eye movements of EMDR might activate REM sleep mechanisms. Although our current model was developed independently, it may best be seen as narrowing the focus and deepening the physiological and cognitive neuroscience underpinnings of Shapiro’s model. Alternative, more psychologically defined models for a role of the orienting response in EMDR have been proposed by Lipke (1995, 2000) and by Armstrong and Vaughan (1996), and both Bergmann (1998) and Servan-Schreiber (2000) have recently proposed a model based on enhanced interhemispheric communication and synchronous neuronal firing patterns. Like ours, these models focus on the ability of EMDR to produce an altered mind-brain state in which effective processing of the traumatic memories can occur.

These theories are testable. Servan-Schreiber has begun tests of his model, and we are now looking at whether EMDR produces an enhancement of weak associations using the same cognitive test with which we showed this effect after REM sleep awakenings (Stickgold et al., 1999). Other studies can be imagined. We would predict, for example, that formal analysis of the dreams of PTSD patients would show increased overall incorporation of episodic memories into their dreams. We would similarly expect an alteration in the normal sleep onset replay of recent experiences (Stickgold, Malia, Maguire, Roddenberry, & O’Connor, 2000). Whether the results of such studies support or oppose these theories, they will move us forward in our understanding of the brain basis of EMDR.

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