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A HYPOTHETICAL MECHANISM OF ACTION OF EMDR: THE ROLE OF SLOW WAVE SLEEP

Marco Pagani, Sara Carletto

Abstract

EMDR is now considered as an elective treatment for PTSD and its efficacy is being proved in several other psychological conditions.

Nevertheless, the EMDR underlying mechanisms of action have not yet been fully clarified. At the moment being, different theories have been proposed, such as the orienting response and the working memory hypothesis, which have been supported by various clinical and neurophysiological researches. This paper discusses a hypothesis which focuses on the similarity between the typical EMDR Eye Movements and delta and beta waves occurring during the Slow Wave Sleep.

SWS appears to have a key role in memory consolidation and in the reorganization of distant functional networks, as well as Eye Movements seems to lead to a weakening of traumatic episodic memory and a reconsolidation of new associated information. SWS hypothesis may represent another important step toward the understanding on how EMDR works.

Key words: EMDR, mechanism of action, eye movements, sleep, slow wave sleep, REM, orienting response, working memory

Declaration of interest: MP and SC have been invited speakers in national and international EMDR conferences

Marco Pagani[1], Sara Carletto[2]

[1] Institute of Cognitive Sciences and Technologies – CNR, Rome, Italy
[2] Clinical and Biological Sciences Department, University of Turin, Turin, Italy

Corresponding author
Marco Pagani
E-mail: marco.pagani@istc.cnr.it

Eye Movement Desensitization and Reprocessing (EMDR) has been proven to successfully desensitize subjects' traumatic memories and decrease anxiety levels improving up to 3 years follow up (Högberg et al. 2008).

Research focused on EMDR has dramatically grown and neuroimaging represents a powerful tool to investigate its neurobiological correlates. The impact of EMDR on cortical and sub-cortical brain regions involved in Post-Traumatic Stress Disorder (PTSD) has been proven by several investigations demonstrating a clear association between symptoms disappearance and normalization of cortical functional changes (Lamprecht et al. 2004, Lansing et al. 2005, Oh and Choi 2007, Pagani et al. 2007, Harper et al. 2009, Ohtani et al. 2009, Nardo et al. 2010, Pagani et al. 2012). Furthermore, patients non-responding to EMDR showed peculiar patterns of neuronal density distribution in limbic regions (Nardo et al. 2010). EEG real time monitoring of cortical activations during bilateral ocular desensitization made EMDR the first psychotherapy in which neurobiological correlates have been depicted in real time during therapy sessions (Pagani et al. 2011, 2012, 2015).

In the recent past EMDR has been successfully applied in several diseases and it has been proposed to reduce anxiety and depressive symptoms in several psychological and psychiatric disorders (Chen et al. 2014, Shapiro 2014) and also possibly impact on the quality of life in different medical conditions (Grant and Threlfo 2002, Friedberg 2004, Schneider et al. 2008, Arabia et al. 2011, Capezzani et al. 2013, Carletto et al. 2016).

Although EMDR is now considered an established treatment for PTSD and other disorders, there is still a debate about its functioning. Despite Maxfield (2008) remark that EMDR is a lot more than Eye Movements (EMs), a large part of this ongoing controversy is about whether EMs are an active treatment component and whether the mechanisms responsible for its efficacy differ substantially from those operating in trauma-focused cognitive behavioural therapy and standard exposure. Although conflicting account remain as to the mechanism of action of EMDR, the evidence that EMDR facilitates the desensitization of traumatic memories called for the search of a rationale of its effects and several hypotheses about EMDR mechanism of action have been put forward.

The orienting and relaxation response (OR) hypothesis offers a theoretical framework explaining the relaxation produced after bilateral stimulation. The OR is based on a natural attentional reflex to any novel stimulus associated with an increased response to potential danger with autonomic response. Moreover, the feeling of relaxation in absence of danger desensitizes traumatic memories and favours rapid extinction (Wilson et al. 1996, Barrowcliff et al. 2003, 2004).

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The working memory account (Hornsveld et al. 2010, 2011; Van den Hout et al. 2010, 2011, 2012, 2013), suggests that both EMs and visual imagery draw on the limited-capacity of the visuospatial sketchpad and the central executive working memory resources. The competition in resources created by the EMs and the dual task will impair imagery, such that the disturbing images become less emotional and vivid. However, most of the evidence is based on inferences from experimental findings in non-clinical subjects rather than from clinical populations and so are not supported by neurobiological evidence.

Some studies suggest that EMDR causes changes in interhemispheric connectivity enhancing episodic retrieval through increased interhemispheric communication via the corpus callosum.

This idea is consistent with both the theoretical framework of EMDR (Adaptive Information Processing Model) and with patients' reports of increased autobiographical memory retrieval during therapy. Anyhow, recent findings cast doubt on the interhemispheric interaction hypothesis (Samara et al. 2011).

In general, these studies assign an important role to eye movements. EMs seem to be not only the distinctive characteristic of EMDR but also the factor accounting for the faster response in EMDR therapy as compared to other psychotherapies. A recent meta-analysis of 26 randomized control trials reported a moderate but significant additive effect size of the EMs to treatment gains (Lee and Cuijpers 2013). Both the OR and the working memory account hypotheses ascribe to bilateral stimulation the putative role of EMDR underlying mechanism. Thus, the neurobiological added value of the EMs might be to complement traumatic memory extinction, but the underlying neurobiological mechanisms are yet to be uncovered.

Several neurobiological theories of mechanism of action have been proposed to explain EMDR therapy's diverse effects. Since the very first case report (Levin et al. 1999) and studies performed with a limited number of subjects (Lansing et al. 2005, Oh and Choi 2007), functional neuroimaging has contributed to the awareness that EMDR impacts directly on the same cortical and sub-cortical brain areas involved in the pathophysiology of PTSD (Pagani 2013). In this respect in 2007 a relatively large study has demonstrated that in patients responding to EMDR cerebral blood flow normalized in regions hyper- and hypoactivated when symptoms were clinically evident whereas this did not occur in non-responders (Pagani et al. 2007). These findings were confirmed by a recent functional study performed by a different technique (fMRI) in bipolar disorder patients (Landin-Romero et al. 2013) showing the normalization of the default mode network following successful EMDR therapy. On the other hand, structural neuroimaging (MRI) has proven that the neuronal density before EMDR therapy might have an impact on its outcome since non-responders showed a significant reduced grey matter volume in the regions target of EMDR (Nardo 2010). However, direct evidence of the impact of EMDR on brain pathophysiology was limited to studies performed pre- or post- desensitization itself and the neurobiological bases of its direct effect on the brain were still unknown. To overcome this limitation Pagani and colleagues (Pagani et al. 2011, 2012, 2015) in a series of electroencephalographic (EEG) investigations reported that the combination of EMs and trauma recall during the symptomatic phase caused a prefrontal cortex hyperactivation not found in control subjects upon the same EMDR session conditions. After successful EMDR therapy no prevalent frontal activation was found in clients and cortical firing moved to regions with cognitive valence. The same occurred in a group of traumatised children in which EEG showed post-EMDR therapy a significant activation in temporo-parietal association cortex (Trentini 2015). These results suggest that EMDR's end point is a transfer of traumatic memory from an implicit hyperemotional subcortical status to cortical regions properly processing traumatic experiences and integrating them into one's semantic memory

In the light of the latter findings we can speculate about a possible link between these neurobiological evidences and rapid eye movement (REM) and slow wave sleep hypotheses.

Patients with PTSD show high rates of sleep disruption (Roszell et al. 1991, Neylan et al. 1998). Insomnia and nightmares are among the most common responses to psychological trauma (Harvey et al. 2003) while long-lasting sleep disturbances are prevalent symptoms in PTSD (Ohayon and Shapiro 2000, Leskin et al. 2002) for which DSM-5 classifies nightmares as intrusion symptoms (cluster B) and insomnia as arousal/reactivity symptom (cluster E).

Sleep facilitates emotional processing and sleep disturbances represent a physiological stressor (Harvey et al. 2003, Maher et al. 2006) intensifying diurnal PTSD symptoms and worsening patients' ability to properly elaborate previous psychological traumas. A poor sleep quality predicts PTSD symptoms severity (Germain et al. 2004) and sleep disturbances also have a negative impact on memory. In fact, chronic insomnia and relative chronic stress induce reduction of hippocampal volume and neurogenesiscausing in turn impairment in the functions processed by such limbic structure, above all long-term and working memories.

Sleep disturbances have also been found to be associated with significantly reduced grey matter volume in amygdala and hippocampus by a structural magnetic resonance investigation (Nardo et al. 2015), underscoring the role of these structures in the neurophysiopathology of the disorder.

Converging evidence supports the significance of sleep in learning and memory reprocessing. During physiological sleep, rapid eye movement (REM) and non-REM slow-wave-sleep (SWS) alternate cyclically with a predominance of REM sleep early in the morning. At EEG, SWS shows synchronous brain electrical activity in the range of typical delta waves (0.5-4 cycles/sec, i.e. 0.5-4 Hertz) while during REM sleep synchronous theta waves (4-8 Hertz) are recorded.

Non-REM SWS appears to have a key role in memory consolidation. SWS also facilitates information transfer from hippocampus to neocortex and the reorganization of distant functional networks. The "dialog" between hippocampus and neo-cortex favours memory encoding. During Rapid Eye Movement (REM) sleep a decreased activity from hippocampus to neo-cortex occurs, suggesting a more intense memory consolidation. In this phase, new associations of emotional events mediated by limbic structures take place.

To summarize, during wakefulness autobiographical, emotional and potentially traumatic events are represented in the sensorimotor cortex, the anterior-superior part of parietal lobe. From such perceptual representation system information are transferred to subcortical limbic structures as hippocampus (episodic) and amygdala (associated affect) where an initial formation of memory occurs. At this stage, hippocampal and prefrontal cortex activation facilitate memory encoding by potentiating

newly acquired information. During SWS, due to the peculiar electrophysiological and biochemical conditions, global synaptic weakening along with slow consolidation of information takes place. Relevant memory circuits reactivate and long-term potentiation is induced in the SWS up-states. During REM sleep, a further potentiation of the reactivated connections and memory network rearrangement occurs, favoured by feedback weakening of episodic memory in subcortical structures. At this stage memory strengthening is associated to increased cholinergic modulation and decreased aminergic modulation as well as to decreased glucocorticoid secretion. After such sequence of events, variably lasting one to several nights, during the wake state information is better retrieved and it is properly positioned into semantic memory.

Stickgold (2002) already proposed a link between EMDR and REM-sleep, proposing that Alternate Bilateral Stimulations typical of EMDR shift the brain into a memory processing mode similar to that of REM sleep.

In 2009 Harper and colleagues (Harper et al. 2009) reported that, upon eye movements, EEG recorded in the delta range (1.5 Hertz) waves very similar to the ones registered during SWS. Such delta waves also paced beta waves (frequency of 13.5 Hertz), speaking in favour of a general resonance in brain electric activity during bilateral stimulation consonant with eye movements.

More recently, Pagani et al. in two separate investigations (Pagani et al. 2011, 2012) reported during the bilateral stimulation phase of EMDR therapy an EEG pattern similar to the one described by Harper and colleagues, suggesting that one of the several neurophysiological effects of EMDR, upon reliving the traumatic memory in association with eye movements, might be a close similarity of the electrical activity to the delta waves seen during SWS.

Fragmented episodic and traumatic memories are stored in hippocampus or amygdala without contextual integration. Memory integration needs the encoding in association cortex to create an understanding in a larger context. Memories temporarily stored in the hippocampal-amygdala complex are transferred to neocortex, replayed, consolidated into semantic associative memory networks and information is integrated to create meaning and "learn from the event". Such transfer might occur during slow-wave-sleep (1-3 Hz) and definitive memory consolidation during REM sleep (about 4-6 Hz). The traumatic episodic memory is weakened and then removed from hippocampus. If this does not happen the lack of available working memory space may lead to cognitive deficits and memory impairment (3° PTSD criterium).

The straightforward implication of such phenomenon is that trough EMDR therapy the client/patients experiences a brain activation state corresponding to the one seen during SWS, the phase in which episodic and emotional memories are supposed to move from subcortical structures and be transferred to neocortex.

Interestingly, such speculation is consonant with the working memory account hypothesizing that bilateral eye movements decrease the vividness of emotional memories promoting their elaboration. The claim of working memory account is that both tasks simultaneously draw on the limited-capacity of the visuospatial sketch pad, a working memory subsystem, and on the central executive working memory resources. This competition created by the dual task would impair imagery, such that the disturbing images become less emotional and vivid. Combining the two hypotheses, it can be speculated that such decrease of vividness might also be related to the added simultaneous effect of SWS-like neurophysiological conditions reproduced by bilateral stimulation during EMDR, favorable to weaken and extinct the traumatic memory at hippocampal level and integrating episodic memory in associative neocortex.

In this respect, a low-frequency stimulation (5 Hertz) was found to cause in the rats'amygdala a depotentiation of AMPA receptors responsible for traumatic memory retention (Mao 2006). During EMDR sessions therapists administer bilateral stimulation at about 1-2 cycles/sec (1-2 Hz). The neurobiological mechanism underlying therapy might be related to the slowing of the depolarization rate of neurons in the limbic system elicited by bilateral stimulation resulting in the emotional memories pathologically confined in the amygdala to move to higher brain centers and being fully processed. Furthermore, such condition resembles the one occurring during SWS in which delta waves of similar frequency facilitate episodic memory, both autobiographical and traumatic in nature, to be transferred to neocortex. It is worth noting that SWS occurs 3 to 5 times during night while bilateral stimulation is performed 25 to 30 times upon each EMDR session. This might account for the very fast processing of bad memories experienced by clients in a single or in a few EMDR sessions.

Further studies are needed to better investigate this hypothesis, and also to explore possible links between SWS and other theories and neurobiological findings already proposed, in order to foster a better understand of the mechanisms of action of EMDR.

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