Eye Movement Desensitization and Reprocessing for Post-Traumatic Stress Disorder from the Perspective of Three-Dimensional Model of the Experiential Selfhood

Andrew A. Fingelkurts a,*, Alexander A. Fingelkurts a

a BM-Science – Brain and Mind Technologies Research Centre, Espoo, Finland

Abstract:
Eye Movement Desensitization and Reprocessing (EMDR) therapy is included in many international trauma treatment guidelines and is also shortlisted as an evidence-based practice for the treatment of psychological trauma and Post-Traumatic Stress Disorder (PTSD). However, its neurobiological mechanisms have not yet been fully understood. In this brief article we propose a hypothesis that a recently introduced neurophysiologically based three-dimensional construct model for experiential selfhood may help to fill this gap by providing the necessary neurobiological rationale of EMDR. In support of this proposal we briefly overview the neurophysiology of eye movements and the triad selfhood components, as well as EMDR therapy neuroimaging studies.

Keywords:
EMDR therapy, PTSD, trauma, Self-Me-I, triad model of selfhood, EEG.

Abbreviations: AIP, Adaptive Information Processing; DSM-5, Diagnostic and Statistical Manual of Mental Disorders; EEG, electroencephalogram; EMDR, Eye Movement Desensitization and Reprocessing; fMRI, functional Magnetic Resonance Imaging; OM, operational module; PTSD, Post-Traumatic Stress Disorder; SPECT, Single Photon Emission Computed Tomography.
Introduction

Eye Movement Desensitization and Reprocessing (EMDR) therapy is an integrated psychotherapy treatment that was originally designed to alleviate the distress associated with traumatic memories [89,92,93]. During EMDR therapy the patient focuses on emotionally disturbing events/feelings in brief sequential periods while simultaneously executing lateral eye movements directed by the therapist [90]. After each period patients give feedback to the therapist about their feelings until the traumatic memory is desensitized and reprocessed in a constructive and adaptive way. Further, EMDR also includes working with anxiety brought on by present situations that link back and trigger previous traumatic experience(s). Treatment is concluded when patients imagine themselves in the future situations where they face the same distressful triggers but feel no emotional discomfort, having instead a complete positive self-experience. As soon as such past, present, and future issues related to traumatic events are desensitized and reprocessed, post-traumatic symptoms abate [91].

The efficacy of EMDR in trauma treatment has been documented in a large number of controlled clinical studies [110,66,13,20,46,54,59,78,100,47,83,109]. Due to such demonstrable efficacy, EMDR is nowadays a recognized and recommended official treatment for acute trauma and posttraumatic stress disorder in most international guidelines (e.g., United Kingdom Department of Health [104], Dutch National Steering Committee Guidelines Mental Health Care [19], American Psychiatric Association [2], French National Institute of Health and Medical Research [41], National Institute for Health and Clinical Excellence Clinical Guidelines [69], World Health Organization [115]).

The neurobiological underpinnings of EMDR’s treatment effects are currently unknown; however, Shapiro proposed a theoretical Adaptive Information Processing (AIP) model [94] according to which EMDR therapy facilitates access to and processing of traumatic memories and/or other adverse life experiences by bringing them to an adaptive resolution. After successful treatment with EMDR therapy, affective distress is relieved, negative beliefs are reformulated, and physiological/bodily arousal is reduced [91].

While the AIP model is helpful in conceptualising EMDR therapy, it lacks overall neurobiological detail and because of that could not provide the needed neurophysiological mechanisms for the observed positive effects. To date several other theoretical models have been also proposed to explain how EMDR therapy works [56]. They fall under three broad classes: The Working Memory Models, Psychophysiological Models, and Sleep Models. These models have been thoroughly reviewed elsewhere [56], thus a summary of their main tenets is outside the scope of the present paper. Suffice to say, while these models were useful for understanding EMDR, they all fall short of explaining the totality of EMDR effects, therefore highlighting need for a more comprehensive theoretical model.
We hypothesise that a recently proposed neurophysiologically based three-dimensional construct model for the complex experiential selfhood [37,38] may help to fill this gap.

The triad model of selfhood

Based on current advances in studying the neurophysiology of the self-referential brain network (also known as default mode network [44,72,10,28,102]) and accumulated empirical knowledge on the functional-topographical specialization of three subnets (i.e. operational modules) of this network under normal/healthy conditions [103,4,5,16,28,116] and during pathological conditions accompanied by diminished or lost self-consciousness [39,40,32], a three-dimensional construct model for the complex experiential selfhood has been proposed [37,38]. Practically, the three brain operational modules (OMs) can be easily and reliably estimated by applying operational analysis to the electroencephalogram (EEG) signal [26,30]. The proposed triad model of selfhood supports the view that the nature of self-awareness is multifaceted [42,61,68,81] and offers a practically useful “tool” to study separate but related brain OMs characterizing three different qualities of self-referential processing, which together form a unified sense of the self [28] (for a similar view see [43]).

According to this triad model of selfhood [37,38] the frontal module of the self-referential brain network mediates the first-person perspective and the sense of agency, and could be conceptualised as the “witnessing observer” or simply the “Self.” The right posterior module of the self-referential brain network supports the experience of self as a localized embodied entity (through interoceptive and exteroceptive bodily sensory processing), emotion-related thoughts, and autobiographical memories, and is conceptualised as the “representational-emotional agency” or simply “Me.” Finally, the left posterior module of the self-referential brain network accompanies the experience of thinking about and reflecting upon oneself, including momentary narrative thoughts and inner speech, and is conceptualised as the “reflective agency” or simply “I.” Every module of this triad is irreducible into one another, and can be enhanced or weakened depending on the current physiologic and mental state [28], voluntary training [37,38] or pathology [39,40,32,33], including the Post-Traumatic Stress Disorder [34].

Self-Me-I in the Post-Traumatic Stress Disorder

In the study examining functional integrity (by means of EEG operational synchrony) within three modules of the self-referential brain network related to three aspects of selfhood in individuals with Post-Traumatic Stress Disorder (PTSD) symptoms in comparison with healthy control volunteers,
it has been found that persons with PTSD symptoms exhibited a pattern with increased integrity of anterior OM (‘Self’-component) and increased integrity of right posterior OM (‘Me’-component) alongside with decreased integrity of left posterior OM (‘I’-component) [34]. Such ‘Self-Me-I’ dynamics help to explain the traumatic experience in PTSD sufferers, which is highlighted by (a) enhanced vigilance to self and surroundings [108,112] – increased ‘Self’-component, (b) enhanced emotional, sensory and bodily states that tend to persistently reoccur as intrusive memories [106,84,55,7] – increased ‘Me’-component, and (c) decreased narration, verbal representation and lack of linguistic/contextual information, often leading to detachment and dissociation [107,21,7] – decreased ‘I’-component. Indeed, correlation analysis [34] reveals a significant correspondence of Self-Me-I components with the four diagnostic criteria under the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) [15]: increased ‘Self’-component is associated with hyperactivity symptoms (criterion E), increased ‘Me’-component is associated with thought intrusion and mood symptoms (criteria B and D, respectively), and reduced ‘I’-component is associated with greater avoidance (criterion C).

Capitalizing on the three-dimensional model of complex selfhood in PTSD and considering the neurophysiological basis for the Self-Me-I components and eyes movements, as well as recent EMDR therapy neuroimaging studies, a neurophysiological rationale for EMDR efficacy in treating traumatic memories and PTSD readily emerges.

The triad model of selfhood as neurobiological rationale for EMDR therapy effectiveness

The triad model of selfhood provides “a novel potential aetiological account of PTSD symptoms grouped under DSM-5 in four clusters (hyperarousal, persistent bodily re-experiencing of the trauma, avoidance of trauma-related stimuli, and decreased narration), and also explains distinct contributions (related to three major aspects of selfhood: first-person agency, representational-emotional agency, and reflective/narrative agency) that every module of the self-referential brain network makes to PTSD” ([34]; p. 49). Based on this study, a three-component therapy for PTSD and traumatic memories was proposed [34]: increasing functional synchrony within the (i) left posterior OM, and decreasing functional synchrony within the (ii) anterior OM and (iii) right posterior OM.

The proposed therapy is hypothesized to normalise the main symptomatic clusters of the disorder: increasing functional synchrony within the left posterior OM (‘I’-component) supposed to reverse the situation when traumatized persons experience ‘speechless terror’ [108], with traumatic memories reduced to some fragmented moments lacking story and narration [7]. The reduction of the functional synchrony within the frontal OM (‘Self’-component) expected to decrease the observed in PTSD
sufferers hypervigilance and exaggerated self-focus [112], as well as associated with it anger, aggression [87], and self-destructive behaviour [99]. Lastly decreasing functional synchrony within the right posterior OM (‘Me’-component) supposed to revert the traumatic experience in PTSD sufferers that is dominated by highly detailed bodily sensations accompanied by fear, sweating, shaking, trembling, shivering, and palpitations [107,55,7]. It seems that EMDR therapy does exactly this. While some may argue that such changes are not specific to EMDR and similar effects could be achieved by other forms of trauma therapy, this is not exactly the case. To understand the uniqueness of EMDR therapy from the perspective of the triad model of selfhood, we have to discuss the neurophysiology of eyes movements (EMs) and OMs triad first.

Neurophysiology of EMs

The use of rhythmic, bilateral, saccadic EMs is perhaps one of the most distinctive elements of EMDR [56]. Initially the EMs were proposed as the "crucial component" of EMDR [48], which was subsequently denied by later some researchers [12,14]. In recent years, the accumulated evidence has re-instated the unique contribution of EMs (and to a smaller degree, other bilateral stimulations) to EMDR therapy success (for several thorough reviews see [49,58,56]). EMs account for quicker EMDR therapy response when compared to other psychotherapies [70] as well as contributing a significant additive effect size to treatment gains [58].

Considering the leading role EMs play in the EMDR therapy to reduce the PTSD symptoms, we argue here that its neurophysiology is uniquely suited to bring about the observed effects of EMDR therapy through modulation of triad modules of the self-referential brain network that are related to three aspects of selfhood.

EMs are generated by the brain and intimately related to many brain processes [80]. Studies have shown that neurons in fronto-parietal network (areas overlapping with the three OMs of the self-referential brain network) anticipate upcoming eye movements and reposition their receptive fields to account for changes produced by the saccades [18,96], thus resulting in complex changes in both local and global neural processing during saccadic EMs [96,67,80]. These EM-related neural activity changes (as opposed to the ‘ocular’ activity also termed as ‘eye movement artifacts’ [45]) predominantly occur within the alpha frequency range [17], which is easily distinguished as 7-13 Hz oscillations in the EEG signal.

The amplitude of ongoing EEG activity, particularly in the alpha band (7–13 Hz), is known to be related to fluctuations in attentional response [86,101,11], orienting reaction (OR) [95,8], memory operations with synchronised alpha activity (large amplitudes in the scalp EEG) during the retention of
a memory scanning task [51], enhanced recognition of relevant information whilst supressing irrelevant content [51], and general psychophysiological state of a person whereas increased alpha is linked to decreased vigilance and anxious arousal, lowered heart rate and skin conductance levels, and general feeling of relaxation [22,73,114,88], indicating a parasympathetic shift. From this brief overview one may easily see how alpha rhythm that is increased by horizontal EMs could represent an extremely useful mechanism to explain the whole totality of various positive effects of EMDR therapy as well as bridge different theoretical accounts of EMDR: (i) holding a traumatic image in mind (retention) while performing EMs decreases vividness and emotionality of disturbing memories [3,50,105] resulting from a discrepancy between the control of EMs and deployment of attention on the retention [71]; (ii) reconsolidation of memory structures through mechanism of active inhibition [51]; (iii) EMs trigger an orienting response that facilitates access to the traumatic memory without avoidance, causing subsequent rapid relaxation after registering no actual immediate threats [6]; (iv) decrease of heart rate and galvanic skin response as a function of EMs/EMDR [113,24]; (v) increased recognition of accurate information and attentional flexibility [23]; (vi) spontaneous generation of positive insight [56]; (vii) reduced anxious arousal when associated with the traumatic memory [93] similar to processes of memory consolidation via the integration of emotionally charged autobiographical memories into general semantic networks during rapid eye movement (REM) sleep [97,98].

**Neurophysiology of Self-Referential Brain Network OMs Triad**

Research has found accumulating evidence that it is exactly EEG alpha rhythm which has a significant positive correlation with self-referential brain network in comparison to other frequency bands [75,64,48,52,53], and it dominates the EEG of humans during mind-wandering and spontaneous thought [77,9,27,29].

As previously discussed in the section ‘The triad model of selfhood’, the self-referential brain network is composed of at least three spatially separable though functionally interacting OMs each consisting of brain regions showing tight “operational synchrony” within every OM in the alpha frequency range [28,37,38]. The operational synchrony estimation requires several consecutive steps of data processing [26,30]. Briefly, using an adaptive segmentation procedure each local EEG signal recorded from a concrete brain location is first reduced to a temporally organized sequence of quasi-stationary (nearly stationary) segments of various duration, where every segment is demarcated by intrinsic points of ‘gluing’ – rapid transitional periods (RTPs). An RTP is defined as an abrupt change in the analytical amplitude of the EEG signal above a particular threshold using statistical procedures. It has been proposed that each homogeneous segment in the local EEG signal corresponds to a
temporary stable microstate – an operation executed by a neuronal assembly [35]. The temporal coupling (synchronization) of such segments among several spatially distributed local EEG recordings then, reflects the synchronization of operations (i.e. operational synchrony), produced by different neuronal assemblies (located in different cortex regions) into integrated and unified patterns responsible for complex mental operations [35].

Neurophysiologically, a single RTP represents a loss of constraints among neurons that constitute a neuronal assembly, followed by rapid arrival to a new configuration, leading the new neuronal assembly to self-present a new simple operation [36]. Cognitively, RTPs could be interpreted as the breakpoints of involuntary (bottom-up) attention leading to attentional disengagement, shift, and allocation to a new operation [31]. In this sense it could be interpreted as a self-organized innate attentional mechanism [79] that is ‘used’ by the brain to place self-presented entities of available information in relation to one another [65]. Neurons within the neuronal assembly continue to fire for some period of time if they are properly biased (or preferentially primed) by another source of subthreshold excitatory input [31]. We argue that EMs (by means of alpha rhythm) provide such an input by affecting the duration of simple operations (modifying the sequences of RTPs) that are executed by local transient neuronal assemblies in the triad of OMs that correspond to Self-Me-I selfhood components (indeed, alpha oscillations have been associated with propagation of activity throughout the brain resulting in control of communication among and within occipital, parietal-temporal, and frontal regions [85]). In this context, by virtue of EEG alpha rhythm properties, EMs activate autonomic self-regulation (similar to autogenic neutralization), whereby excessive functional activity is dampened and diminished functional activity on the contrary is upregulated [62,63]. Thus, EMDR is hypothesized to normalise functioning of the OMs triad by (i) increasing functional integrity of the hypoactive left posterior OM (‘I’-component), (ii) decreasing functional integrity of the hyperactive frontal OM (‘Self’-component) and (iii) decreasing functional integrity of the hyperactive right posterior OM (‘Me’-component). These then are the proposed mechanisms through which PTSD symptoms are reverted.

Neuroimaging studies provide additional further support to this hypothesis. While results vary [56], the cumulative evidence has shown that comparing the pre-to-post EMDR treatment, the following tentative pattern emerges: the successful EMDR treatment of PTSD resulted in the loss of activity (measured by functional Magnetic Resonance Imaging (fMRI)) in the right brain hemisphere alongside with increase of activity in the left [82]. Activation of the left hemisphere as a function of EMDR therapy has also been documented in Single Photon Emission Computed Tomography (SPECT) studies [60] and EEG [74,76]. Since both brain hemispheres function as a functional unit, increased activation in one usually results in inhibition of the other [1,111]. Furthermore, it has been shown that
the most relevant functional correlate of successful EMDR therapy – attenuating the negative emotional responses to traumatic memories – is a decrease in the frontal activity [75,76]. The net effect of these neurobiological changes within the self-referential brain network was hypothesized to be responsible for the desensitization and reprocessing of traumatic memories, integrating them into a larger narrative story, and decreased self-focus, thus contributing to symptom reduction and remission [25].

In summary, it seems that the three-dimensional model of experiential selfhood may provide a broad neurophysiological rationale for EMDR therapy. However, experimental verification of this hypothesis is needed.

Acknowledgements

We thank Dmitry Skarin for English editing.

Conflict of interest

The authors have no potential conflicts of interest to declare with respect to the research, authorship, and publication of the article. The authors received no financial support or funding for the research, authorship, and publication of this article.

References


[106] van der Kolk BA. The body keeps the score: Memory and the evolving psychobiology of posttraumatic stress. Harv Rev Psychiatry 1994;1(5);253-65.
