Psychology Research and Behavior Management

Dovepress

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Open Access Full Text Article

REVIEW

Potential of eye movement desensitization and reprocessing therapy in the treatment of post-traumatic stress disorder

This article was published in the following Dove Press journal:
Psychology Research and Behavior Management
26 September 2014
Number of times this article has been viewed

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Abstract: Post-traumatic stress disorder (PTSD) continues to attract both empirical and clinical interest due to its complex symptom profile and the underlying processes involved. Recently, research attention has been focused on the types of memory processes involved in PTSD and hypothesized neurobiological processes. Complicating this exploration, and the treatment of PTSD, are underlying comorbid disorders, such as depression, anxiety, and substance use disorders. Treatment of PTSD has undergone further reviews with the introduction of eye movement desensitization and reprocessing (EMDR). EMDR has been empirically demonstrated to be as efficacious as other specific PTSD treatments, such as trauma-focused cognitive behavioral therapy. There is emerging evidence that there are different processes underlying these two types of trauma treatment and some evidence that EMDR might have an efficiency advantage. Current research and understanding regarding the processes of EMDR and the future direction of EMDR is presented.

Keywords: post-traumatic stress disorder, eye movement desensitization, neurobiological, symptoms, treatment, comorbid

Introduction

Post-traumatic stress disorder (PTSD) presents with a complex and diverse set of symptoms involving a mixture of social, biological, and psychological processes. Adding to the complexity are several comorbid disorders, including mood, anxiety, and substance use disorders, traumatic brain injury, grief, and chronic pain. Comorbid disorders complicate the identification of predisposing and perpetuating factors, assessment, clarity of primary diagnosis, and selection of treatment plans. Here we present a broad overview of PTSD, including its intricate neurobiological and psychological symptom profile and common comorbid disorders. Evidence for the effectiveness of eye movement desensitization reprocessing (EMDR) is then presented, as well as its possible advantages, controversies, and key processes. Finally, the future possibilities for EMDR are discussed.

Psychological symptom profile of PTSD

The recently released DSM-5 (*Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition) identifies a more expansive symptom profile for PTSD than previous definitions. The symptom criteria now include re-experiencing, avoidance, negative alterations in cognitions and mood, and negative alterations of arousal and reactivity. In contrast with the DSM-IV, the type of event that can result in symptoms of PTSD

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has been extended, and differing presentations in children and adolescents have been acknowledged.

Perhaps a distinguishing hallmark of PTSD is the intrusive experiences, consisting of a sense of reliving the event in vivid visual images with physiological responses, and auditory and olfactory aspects, all of which occur with the same intense feeling of panic and fear as experienced in the distressing situation.[1–3] An internal or external reminder of the event triggers these intrusive experiences, resulting in a sense of lack of control over their appearance. Combined with the vivid yet fragmented and disorganized presentation of such memories, the intrusive symptoms are both distressing and confusing, resulting often in fear of the symptom itself.[4,5] The fragmented, and sometimes inhibited, storage of memories related to the distressing event is another defining feature of PTSD.[4] An inability to place the event in a coherent, sequential time line, or to remember elements of the event at all, is a distinctive symptom that again causes both distress and frustration.

The cycle of fear and retraumatization caused by the intrusive symptoms often results in avoidance strategies. Avoidance of external and internal stimuli (thoughts and feelings) directly associated with the traumatic event can be explained as a direct learning experience; however, individuals often avoid unrelated situations.[3,6] Research has demonstrated that avoidance not only results in an increase in symptomatology but also results in diminished interest in participating in activities, leading to withdrawal and feelings of detachment and estrangement from others.[3,7] In addition, individuals can respond to the distressing event and intrusive symptoms with an intense need to self-protect. This manifests as symptoms of hyperarousal, including sleep difficulties, hypervigilance, and lack of concentration. These heightened levels of arousal both feed into and result from a fear response cycle, thereby resulting in a constant state of hyperarousal.[3] This combination of symptoms can ultimately result in irritability, aggression and, at times, self-destructive behaviors.

PTSD continues to be listed as an anxiety disorder in the DSM-5, although the centrality of dissociative symptoms has been argued.[8] Dissociative responses, such as derealization and depersonalization, at times referred to as defensive responses, and constricted affect or emotional numbing, are features of PTSD.[6,8] Such symptoms have been shown to correlate with the severity of the trauma, fear of death, and feelings of helplessness during the traumatic event.[8] These symptoms can emerge from defense mechanisms that initially protect individuals from the reality of their current experiences, but later evolve into maladaptive strategies that do not enable consolidation of past experiences. Altogether, PTSD symptoms are debilitating and, as time passes, can generalize to experiences unrelated to the distressing event. This can result in isolation and cognitive distortions affecting one's core sense of self.

Neurobiological symptom profile of PTSD

Shalev[3] proposed that the complexity of PTSD symptoms is best understood as the co-occurrence of several processes including: an alteration of neurobiological processes; the acquisition of conditioned fear responses to trauma-related stimuli; and altered cognitive schemata and social apprehension. Neuroimaging technology has enabled researchers to explore the neurobiological basis of PTSD. The research to date presents contrasting results partially attributed to methodological differences including age range, trauma type, imaging methods, analysis of imaging methods, symptom duration, and inclusion of comorbid disorders and medications. Overall, there is building evidence that symptomatology following experiences of traumatic stress may derive from effects on brain function and structure. Less clarity surrounds the exact areas involved and whether the differences are predisposing or perpetuating factors of PTSD.

Research has demonstrated that following a distressing event there is an interruption of the brain's normal mode of processing information.[9,10] This includes a failure to create a coherent memory of the experience, as all aspects of the memory, sensory, thought, and emotionality appear unable to be categorized and integrated with other experiences.[10] This may be due to deficits in declarative and non-declarative memories that appear to be implicated in PTSD symptoms through their impact on conditioned responses, reliving of traumatic memories, and fragmentation of memories (both autobiographic and trauma-related).[9,11]

Non-declarative (also known as implicit or procedural) memory refers to memories of skills and habits, emotional responses, reflexive actions, and classically conditioned responses (for example, memories that cannot be willfully brought into the conscious mind such as riding a bike).[9] Declarative (also known as explicit) memory refers to conscious awareness of facts or events that have happened to the individual (for example, remembering facts or lists).[12] In relation to PTSD, two types of declarative memories appear to be of importance, ie, episodic and semantic memories. Episodic memories are isolated memories of distinct events that form rapidly, with strong and vivid clarity, are rich in

sensory detail, and are unambiguous.[13] Semantic memories are stored as more general knowledge; these are abstracted from episodic memories and integrated with other semantic memories, creating meaning and understanding of oneself in context of the world.[13] PTSD symptoms are proposed to occur due to a failure of the brain to consolidate and integrate episodic memories into the semantic system, resulting in prolonged and inappropriate resurfacing of episodic memories of traumatic events with no association to other memories.[13]

Situationally accessible memory is a long-term, image-based perceptual representation of the incident that is accessed automatically, whereas verbally accessible memory incorporates representations that reflect more conscious attention when being encoded, are accessible, verbalizable, and interact with other information in the autographical memory. Brewin[1] proposes that intrusive flashbacks occur when encoding of the event into situationally accessible memory is enhanced, and encoding or re-encoding into verbally accessible memory is diminished, resulting in the memory never being re-encoded into episodic memory. Under this hypothesis, a large amount of sensory information can be stored and encoded over long periods due to the situationally accessible memory system capturing sensory images whereas episodic memory is downregulated while under extreme stress.

Each of these implicit memory systems is associated with particular areas in the central nervous system. The hippocampus and the amygdala have been identified as playing roles in the storage and integration of memories.[14,15] The hippocampus plays a role in new learning formation, consolidation of information from short-term memory to encoding, and retrieval of long-term memories.[10,11] For example, declarative memories are initially stored in the hippocampus and associated limbic structures as episodic memories; when integrated into the semantic system, it is hypothesized that hippocampal memory traces are weakened, resulting in a decrease in associated affect and PTSD symptoms.[13]

A deficit in the capacity for new learning and memory becomes critical to the stress response in regard to being able to assess potential threats as well as the response to current and future situations.[9,11] MRI scans have shown a reduction in volume of the hippocampus in individuals with PTSD; however, other evidence suggests a decreased hippocampal volume may predispose individuals to PTSD.[14,16] Both animal and human studies identify stress, particularly chronic stress, as a potential cause of neural damage to the hippocampus.[14,16–18] Neural damage may occur in the hippocampus due to increased levels of glucocorticoids, eg, cortisol and corticosterone, which are stress hormones aiding the fight and flight response and recovery from stressors,[19] but inhibit neurogenesis and decrease levels of brain-derived neurotropic factor during and following exposure to a stressful event.[17,20]

The hippocampus has a high concentration of glucocorticoid receptors, which can remain at high levels for days following the stress response. This appears to evoke a decrease in dendritic branching, alterations in synaptic terminal structure, loss of neurons, and inhibition of neuronal regeneration, resulting in hippocampal atrophy.[17,20,21] As the hippocampus mediates memory functions, hippocampal atrophy may lead to memory deficits and result in memories being experienced as timeless and fragmented.[10,22]

The amygdala stores the emotional content linking episodic memories to emotions, resulting in the original sensations and emotions replaying when memories are recalled.[13] The amygdala has been identified as playing a critical role in stress responses, including fear acquisition, emotional regulation, and learning, conditioning, generalization, and extinction processes of the fear response mechanism.[23–26] Alterations in fear response are proposed to lead to intrusive memories, flashbacks, and automatic hyperarousal, with avoidance and emotional numbing reactions acting as coping strategies for such symptoms.[23,24] Many subregions of the amygdala are proposed to influence the fear response involved in the symptomatology of PTSD (see Table 1), and other systems project to these amygdala subregions. One of these systems is the medial prefrontal cortex. The medial prefrontal cortex is understood to provide a system of negative feedback to the amygdala, that regulates activation of amygdala during fearful experiences.[27] Studies by Semple et al[28] and Shin et al[27] have found a decrease in medial prefrontal cortex activity and associated hyperactivation of the amygdala in individuals with PTSD. Amygdala hyperactivity, or an exaggerated response, has been proposed to result in a failure of extinction to fearful stimuli.[23,27,29]

Chronic stress that causes dendritic atrophy in the hippocampus elicits hypertrophy in subregions of the amygdala, increasing amygdala volume.[26,30] Unlike the hippocampus that demonstrates relatively rapid plasticity, animal research indicates that the hypertrophy in the amygdala remains for a much longer duration, and may result in heightened levels of anxiety over time.[31] Hyperactivity of the amygdala in PTSD may also be due to the exaggerated response of the fear circuitry, thereby explaining hypervigilance and hyperarousal in PTSD.

Table 1 Brain structures and their proposed function in PTSD symptomatology

Brain structure	Proposed function
BLA and ITC (GABAergic neurons lying between the BLA and CeA)	Suggested to be responsible for both initiating and inhibiting fear responses.[32] The BLA plays a role in sensory integration by sending information to both the CeA and the bed nucleus of the stria terminalis. These structures are thought to communicate with the hypothalamic and brainstem areas which are involved in fear and stress responses.[33]
Anterior cingulate gyrus	May play a role in filtering and aiding integration of emotional and cognitive components, possibly aiding modulation of fight/flight reactions to perceived threat.[10]
CeA	The main output center for responses to fearful stimuli.[23,34] The CeA mediates the initiation of fear responses; when this area is removed in animals, the fear response is nonexistent.[32]
Corpus callosum	An area of interest due to its involvement in the transfer of information across both hemispheres, integrating emotions and cognitive responses.[10,35]
Frontal lobes	Act as a supervisory system for integration of information.[36]
HPA	A hormonal flow traveling from the hypothalamus to the adrenal glands, via the pituitary gland. The hippocampus is thought to have inhibitory effects on the HPA axis, while the amygdala is thought to regulate the HPA via excitatory signals.[32]
Parahippocampal gyrus region	May have heightened influence from the amygdala during an emotionally arousing learning situation.[14]

Abbreviations: BLA, anterior basolateral nuclei; CeA, central nucleus of the amygdala; HPA, hypothalamic-pituitary adrenal axis; ITC, intercalated cells; PTSD, post-traumatic stress disorder.

Comorbid disorders

Ensuring that neurobiological research is methodologically sound is difficult, partly due to the presence of comorbid disorders in individuals with PTSD. Research suggests that comorbidity with PTSD is the rule rather than an exception. For example 63% of (veterans) who developed PTSD following a traumatic event also had a diagnosis of major depressive disorder, panic attacks, generalized anxiety disorder, or substance use disorder.[32] In comparison these disorders were only present in 9% of those who did not develop PTSD. Teasing apart the symptomatology of comorbid disorders has proven complicated because there is mixed understanding as to whether they are independent disorders or part of one construct.[33] Features traversing PTSD, major depressive disorder, generalized anxiety disorder, and substance use disorder include anhedonia, sleep disturbances, concentration difficulties, irritability and fatigue, low mood, agitation, guilt, withdrawal, and loss of enjoyment in activities.[3,34]

On a neurobiological level, a number of similar structures have been identified as possible underlying factors for several comorbid disorders and PTSD. Smaller hippocampal volumes have been found in individuals with major depressive disorder, anxiety, and alcohol abuse,[35] with research suggesting prolonged glucocorticoid exposure as a possible cause.[36] Thus, the development of major depressive disorder, generalized anxiety disorder, and PTSD may be influenced by pre-existing, overlapping, or common vulnerabilities, such as neuroticism, a history of pre-existing mood or anxiety disorder, a history of trauma, or neurobiological differences. However, these disorders have a unique constellation of symptomatology, with PTSD requiring a distressing event to meet diagnosis and studies suggesting that PTSD is distinguishable due to features such as reliving/flashbacks, fragmentation of memories, and dissociative features, enabling a separate diagnosis.[34,37,38]

The prevalence of substance abuse disorders among individuals diagnosed with PTSD has been shown to be as high as 40% in samples of those seeking treatment for substance abuse.[39] Substance use can follow PTSD, and this behavior has been described as "self-medicating".[40] PTSD with a comorbid substance use disorder has been shown to increase the complexity of the clinical presentation through increased severity, increased risk of anxiety and personality disorders, high-risk behaviors resulting in risk of exposure to traumatic experiences, high treatment attrition rates, and less engagement with aftercare.[40–42] If PTSD is untreated, the prognosis for substance use disorder is poor and the probability of exposure to future traumatic events is higher.[43] Once a comorbid diagnosis is established, treatment models incorporating both PTSD treatment and relapse prevention models are most successful.[44,45]

Traumatic brain injury, physical injury, and chronic pain are also commonly associated with PTSD. Corresponding symptoms of PTSD and traumatic brain injury include sleep disturbance, irritability, fragmented memories, difficulties concentrating, and haziness (appearing like dissociation).[46,47] From a therapeutic perspective, accurate diagnosis is often difficult as traumatic brain injury can be challenging to

detect, being a normal response in some circumstances; however, if present, it can interfere with the ability to process the traumatic event during treatment. Clearly identifying the presence of traumatic brain injury is important to ensure successful treatment.

The literature indicates chronic pain and PTSD comorbidity rates of 20%–50%.[48,49] Chronic pain and PTSD present with overlapping symptoms of fatigue, cognitive distortions, anxiety sensitivity, experiential avoidance, hypervigilance, and sleep disturbance.[48,50] Such symptoms may be both predisposing and maintaining for each disorder. A cyclical fear-avoidance process often occurs with chronic pain in which the fear of pain produces a catastrophic misinterpretation of bodily sensations and cognitive distortions/catastrophic appraisals.[48] This then leads to avoidance behaviors and inactivity, resulting in disability and an increase of focus on pain intensity, perpetuating further avoidance and anxiety.[48] Pedler and Sterling[51] found an interaction between pain, disability, PTSD symptoms, and sensory hypersensitivity that resulted in the pain cycle being triggered continually. Pain can act as a trigger for intrusive symptoms of PTSD, resulting in increased emotional distress, more intense feelings of pain, and heightened avoidance strategies and immobility.[51] On a neurobiological level, it has been hypothesized that pain and PTSD have similar neurobiological mechanisms, such as an abnormal stress response and hypothalamic-pituitary-adrenal axis deregulation.[48]

Treatments of PTSD

The complexities in the profile of PTSD are evident at both a psychological and neurobiological level. These same complexities influence treatment models for PTSD. Current literature suggests that trauma-focused cognitive behavioral therapy, exposure therapies, and EMDR are the most efficacious treatments. However, almost half of the individuals treated for PTSD do not fully recover.[52,53] Understanding the processes involved in PTSD and at work during treatment aids our ability to improve the rate of recovery. Early understandings of PTSD processes was driven by behavioral theories that were based on conditioning and learning principles. It was thought that preventing avoidance of fear triggers would result in habituation and extinction, thereby alleviating PTSD symptoms.[54,55] However, it has since been suggested that this model does not consider non-fear elements, such as shame, guilt, and anger.[56] Research has now evolved beyond the fear response to the way distressing memories are processed, integrated, and represented.[1,55]

Information-processing theories hypothesize that processing memories so that resolution of the meaning of the event takes place is a more successful theoretical and subsequent treatment model for PTSD than models based on learning theory.[54,57] Emotional processing, cognitive models, dual representation, and adaptive information processing all fall under the banner of information-processing theories.[55] More specifically, EMDR evolved under the adaptive information-processing theory.[58] EMDR was built on the understanding that processing the meaning of the event through integration of memories into an individual's autobiographical memory would help to alleviate PTSD symptoms.[58] EMDR has been verified as an effective treatment for PTSD and meets criteria for evidence-based practice in the UK by the National Institute for Clinical Excellence (2005), in Australia by the Australian Centre for Posttraumatic Mental Health (2013), and in the Netherlands by the Dutch National Steering Committee for Guidelines for Mental Health Care (2003). EMDR is also listed in the World Health Organization guidelines for PTSD.

Research suggests that EMDR, although equal in achieving overall symptom reduction, may be superior to other treatment models in terms of treatment efficiency.[59–62] Some studies have found that EMDR results in more rapid symptom reductions than other comparable treatment models, which in turn results in fewer treatment sessions required for the same outcome in comparison with other types of therapy.[63–66] The rapid reduction in symptoms may be due to a large drop in intrusive symptoms in EMDR. Lee et al[60] and Vaughan et al[62] found a greater decrease in intrusive symptoms in comparison with stress inoculation training with prolonged exposure, imaginal exposure, or applied muscle relaxation.

Ironson et al[59] reported a significant difference in reduction of Subjective Units of Distress Scale scores following the first active session of EMDR in comparison with prolonged exposure. It was suggested in this study that prolonged exposure was more distressing, particularly in the first session, than EMDR. Several studies have shown fewer dropouts in EMDR groups, leading to the proposition that EMDR is both better tolerated and a more efficient treatment model.[59,61,67] EMDR has been shown to be effective without the prescription of several hours of homework which is most often required in exposure-based treatments, again contributing to the view that it is better tolerated and more efficient.[59,67,68] Nevertheless, there is some difficulty in interpreting research on treatment efficiency due to the differences in the way efficiency has been operationalized (for example, number of sessions taken to reduce symptoms, reduction in Subjective Units of Distress Scale scores in the first session, attrition rates, and number of treatment hours required). Thus, more

extensive research is required to clarify the advantages of EMDR in terms of treatment efficiency.

Studies have shown that complex trauma cases require more sessions for symptom reduction than single case traumas; single case traumas have been shown to result in no longer meeting a diagnosis of PTSD after just a few sessions of EMDR.[59] There are currently few randomized controlled trials involving individuals with complex histories of trauma. Hence, the efficacy and efficiency of symptom reduction in a complex population both immediately following and after long-term use of EMDR is unclear. Lee et al[63] suggested that when comorbidity and chronicity are part of the treatment presentation, avoidance strategies or larger more entrenched memories may render fear networks more resistant to modification. With the rate of comorbid disorders being high in PTSD, and complex histories often emerging in a clinical setting, continued research is required in this complex trauma population to clarify the effectiveness and efficiency of EMDR.

The role of psychopharmacological treatment in PTSD is limited based on current evidence, with current guidelines indicating drug treatment to be second-line after trauma-focused psychotherapy.[69] Selective serotonin reuptake inhibitors, including sertraline and paroxetine, are most commonly prescribed to reduce PTSD symptoms and prevent relapse, although current evidence indicates these medications alone result in only modest effect.[70] There is some evidence that other classes of antidepressants, such as tricyclics and monoamine oxidase inhibitors, can be effective in the treatment of PTSD; however, the adverse effects/benefit ratio is generally seen to favor selective serotonin reuptake inhibitors.[71]

There is very little evidence to support the use of mood stabilizers (such as lithium or sodium valproate) or benzodiazepines in the treatment of PTSD. Prazosin and clonidine, which decrease adrenergic activity in the central nervous system, have been shown to reduce night-time and daytime PTSD symptoms, and there is some evidence that the more sedating atypical antipsychotic agents, such as olanzapine, quetiapine, and risperidone, can help as adjunctive treatments in reducing symptoms of hyperarousal.[70] Medications have been seen to reduce symptoms of PTSD; however, the literature identifies they should not replace evidence-based psychotherapies as a primary treatment unless patients are unable or unwilling to engage in therapy.[72]

EMDR processes

As in other therapies, the precise mechanisms of change are unknown in EMDR. There have been various arguments in the literature as to the differences between EMDR and trauma-focused cognitive behavioral therapy or exposure-based models of treatment. Both models can be viewed in the light of information-processing theories; both address the individual's troubling memories and personal meanings of the event and its consequences through having the client focus on the distressing event.[52,73] Both models adhere to theories of fear structures as they both activate the fear memory network through presentation of information that matches elements of the fear structure and introduce corrective information incompatible with these elements.[74,75]

Although EMDR and traditional models of exposure show some common elements, there are clear differences. Imaginal exposure guides the individual to repetitively relive the traumatic experience as vividly as possible without moving to other memories or associations.[74] This "flooding" approach is based on the theory that anxiety is caused by conditioned fear and reinforced by avoidance.[67,74] In contrast, EMDR progresses through chains of associations that appear to be linked to shared sensory, cognitive, or emotional states in a non-directive way. The individual is encouraged to "let whatever happens happen and just notice" when freely associated memories enter into the mind though imaginal exposure in short bursts.[66,76] According to traditional conditioning theories, promoting attention to the fear-relevant information facilitates activation, habituation, and modification of the fear structure; treatments that distract from this should be ineffective.[77,78] Nevertheless, EMDR is effective.

During EMDR, the therapist often accesses only brief details of the trauma memory, and encourages image distortion/distancing which, according to traditional theories, should result in cognitive avoidance.[74] EMDR treatment encourages distancing effects that are considered effective processing of the memory rather than cognitive avoidance.[74] This process may contribute to individuals reporting EMDR as less confronting and may explain why it is better tolerated.[67]

EMDR encompasses the complex emotional responses following a distressing event by looking at affect, physical sensations, cognitions, and emotions and beliefs concurrently.[74] The cognitive shifts that EMDR evokes show that the client can access corrective information and link it to the trauma memory and other associated memory networks with little, if any, guidance.[74] It seems that the integration of both positive and negative material that occurs spontaneously during the desensitization process of EMDR resembles assimilation into cognitive structures (in line with adaptive information-processing theory) such as world views, values, beliefs, and self-appraisals.[55,75]

Overall, EMDR has challenged the theoretical grounds and understandings of historical treatment models for PTSD. The eye movement component has drawn a large amount of debate as this seems to be the component that differentiates EMDR from trauma-focused cognitive behavioral therapy and exposure-based treatments. However, it has been suggested that eye movements are not necessary, with the research being mixed as to whether other bilateral stimulation (auditory or tactile) or no eye movements produce equivalent results.[79–82] Based on fear extinction models, the eye movements should cause distraction and decrease habituation. Lee and Cuijpers[83] conducted a meta-analysis to determine the efficacy of eye movements when processing emotional memories. Their results supported the inclusion of eye movement in both treatment and laboratory environments, and demonstrated the importance of treatment fidelity when implementing EMDR.[83,84]

The proposed advantages of the extra task of eye movements in EMDR are distancing and a reduction of the vividness and emotionality of the memory.[85] Based on the theory that PTSD symptoms result from a failure to process episodic memories, it is suggested that the bilateral eye movements may facilitate interhemispheric interaction, resulting in improved processing of the memory.[86] Research indicates that episodic memory processing is bilateral, whereas semantic memory processing is conducted in the left hemisphere of the brain.[86,87] Horizontal eye movement may enforce an increase in activation of both hemispheres, increasing communication between them and promoting processing by boosting the capacity to recall all elements of the event from episodic and semantic memories.[86] Conflicting research, such as that produced by Propper et al,[88] showed that engaging in bilateral eye movements during EMDR resulted in decreased interhemispheric gamma electroencephalogram coherence (which is associated with episodic memory processing).

Other proposed theoretical models are based on rapid eye movement (REM) sleep and orienting response models. Research suggests that integration of episodic memories to semantic memories occurs during sleep.[13] Brain research has demonstrated specific brain regions that are affected by the restimulation of traumatic memories in PTSD; these are the same regions activated in the REM phase of sleep.[13] The repeated bilateral eye movements are thought to activate the brain stem into a REM sleep state, thus supporting memory integration and a reduction in PTSD symptoms.[13,89]

The same repetitive bilateral stimulation reorienting attention from one side to another is also proposed to activate a similar neurological mechanism to REM sleep through an orienting response.[10] Activation of these mechanisms shifts the brain into a "memory processing mode" similar to REM sleep, permitting the integration of traumatic memories.[10,13,90] It has also been proposed that eye movements trigger the orienting response by activating an "investigatory reflex" that is firstly an alert response and then a reflective pause that produces dearousal if there is no real threat.[91] This reflex response results in heightened alertness which is proposed to permit exploratory behaviors where cognitive processes become more flexible and efficient, allowing the traumatic memory to be integrated.[92] The eye movements are proposed to create a relaxation response, facilitating reprocessing of memories by decreasing distress.[55,93]

Under a working memory model it has been hypothesized that the positive effects from EMDR result from the eye movements creating a dual attention task. In line with the working memory model proposed by Baddley,[94] working memory has limited capacity. When dual attention is required, the quality of the trauma image deteriorates, resulting in it being pushed out of working memory and integrated into long-term (semantic) memory where vividness and emotionality are reduced.[80,82,95] The dual task of holding the emotion in mind while focusing on bilateral eye movements may disrupt the storage of traumatic memories, decreasing the episodic quality of the memory and therefore decreasing the symptoms of PTSD.[58,85] More specific exploration by Gunter and Bodner[80] resulted in the proposition that memories held in the visuospatial sketchpad (a subsystem of working memory) decrease in vividness when eye movements deplete processing resources. Research has shown that a reduction in vividness of the memory, proposed to be due to eye movements, may result in a subsequent decrease in emotionality around the memory and a corresponding decrease in PTSD symptoms.[85] More research is required to verify a causal relationship between the level of vividness and consequent changes in emotionality.

Overall, key processes underlying EMDR mechanisms are complex in line with the treatment structure, which involves components of mindfulness, cognitive restructuring, exposure to the memory, and a sense of personal mastery.[75] A few studies have assessed neurobiological changes pre and post EMDR treatment to begin to establish structural and functional changes that may occur. Lansing et al[96] found a decrease in the left and right occipital lobe, left parietal lobe, and right precentral lobe, and increased perfusion in the left inferior frontal gyrus for participants following an average of 10.25 hours of EMDR. Similarly, single-photon emission computed tomography results showed normalization of

cerebral blood flow in the parieto-occipital, visual cortex, and hippocampus, and an increased cerebral blood flow in the prefrontal cortex in participants receiving EMDR.[97]

Electroencephalography has been used to monitor neuronal activation while actively engaged with bilateral eye movements.[98] Participants in the study by Pagani et al were found to have activation in the prefrontal regions, limbic regions, fusiform and visual cortex, ie, areas that have been associated with decreases in negative emotional experiences.[98] This study supports the hypothesis that EMDR works by moving the memory from an implicit subcortical to an explicit status where different cortical regions work to process the experience.

Lower gray matter density has been found in individuals with symptomatic PTSD (n=21) compared with individuals who have asymptomatic PTSD (n=22) in the left posterior cingulate (possibly influencing retrieval of autobiographical memories and relation to self) and left posterior parahippocampal gyrus.[99] In a second part of this study, 15 of the symptomatic participants received EMDR. The results showed that participants who responded well to EMDR (n=10) exhibited a proportional increase in gray matter density in the bilateral posterior cingulate as well as anterior insula and right anterior parahippocampal gyrus when compared with non-responders. Nardo et al[99] proposed that structural changes in these areas may influence the ability to cognitively reappraise and to integrate emotions and body sensation, resulting in non-response to EMDR.

Future directions and conclusion

PTSD is a complex disorder, and underlying mechanisms continue to be explored and understood. Neurobiological research is in its infancy; although it is already adding to the understanding of processes driving PTSD, there is mixed evidence supporting any one theory or neurobiological site. Future research exploring pre- and posttreatment structural and functional changes is required to support the hypotheses currently being suggested, and to explore specific areas of the amygdala, hippocampus, and the prefrontal cortex. It is vital that a methodological benchmark be set for such studies, because the various methods used are producing inconsistencies and some confusion regarding already complicated research models.

EMDR has demonstrated effectiveness and efficiency in the treatment of PTSD, and future research and directions for EMDR are abundant. The evidence to date suggests that traditional exposure-based therapy models and EMDR have different processes, both theoretically and when reviewing the processes of each treatment model. Theories and research looking at the function and structure of the memory system in relation to its role in PTSD and in EMDR continue to evolve. Recent areas of interest are the visuospatial sketchpad (processing visuospatial information) and the phonological loop (processing verbal information), which may lead to further clarification of the mechanisms of EMDR.[80,82] Further exploration of the role of eye movements in EMDR, and what mechanisms they activate when contributing to symptom reduction, is necessary.

Further research on outcomes research looking at treatment efficiency, particularly in a population that presents with complex trauma, is necessary. Efficiency is imperative in clinical settings where government and private funding models only allow a minimum number of sessions, and attrition rates are high in many settings. Hence, the potential of EMDR as a superior treatment model based on efficacy and efficiency is of critical importance and requires methodologically strong research to ensure that clinicians can make a clear choice in the treatment model they use. Research in children and adolescents is sparse, with comparative outcome studies required to determine the effectiveness of EMDR in this population.

EMDR is also in the early stages of being identified as a type of treatment for attachment disorders, grief, nightmares, other anxiety disorders, and substance disorders. Research to date is limited, but the results available indicate that comorbid disorders may also respond to EMDR. It is important to explore the potential of EMDR to provide clinicians with a treatment model that can traverse many symptom presentations in an efficient manner.

Disclosure

The authors report no conflicts of interest in this work.

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