NEUROPHYSIOLOGICAL COMPONENTS OF EMDR TREATMENT

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The research on Eye Movement Desensitization and Reprocessing (EMDR) has had a significant development in the last 10 years. The EMDR consists on a dual focalization activity (the use of eye movements or other forms of left/right rhythmical stimulation, while focusing on personal disturbing material). Many hypotheses have been made on how EMDR works and why the clinical results are so significant. One of the most possible reasons may regard the fact that there seems to be an innate information processing system that is physiologically configured to facilitate mental health in much the same way the rest of the body is designed to heal itself when injured (Shapiro, 1995). When operating appropriately, this system takes the perceptual and emotional information from a traumatic event to an adaptive resolution - useful information is stored with appropriate affect and is available for future use. The physiological and emotional arousal stemming from a traumatic event may disrupt the information processing mechanism. The blocked processing prevents the traumatic information from progressing through the normal steps of adaptive integration. The physiological stimulation appears to activate the innate information processing system and may be linked to the mechanisms inherent in memory storage. EMDR apparently intervenes in brain functions, especially in the limbic system and amygdala, which have been already identified as actively involved in traumatic experiences.
Key-words: *EMDR, Information Processing System, Neurophysiology.*

There seems to be an innate information processing system that is physiologically configured to facilitate mental health in much the same way the rest of the body is designed to heal itself when injured (Shapiro, 1995). When operating appropriately, this system takes the perceptual and emotional information from a traumatic event to an adaptive resolution - useful information is stored with appropriate affect and is available for future use. But sometimes, the physiological and emotional arousal stemming from a traumatic event may disrupt the information processing mechanism. This can result in the information taken in during the time of the trauma (e.g. disturbing images, thoughts, sensations, beliefs, and the like) becoming stored in disturbing, excitatory, state-specific form. The blocked processing prevents the traumatic information from progressing through the normal steps of adaptive integration. Nightmares, flashbacks, intrusive thoughts and sensory imagery, and other symptoms of PTSD may result from continual activation of this dysfunctionally stored information by internal or external stimuli, or perhaps because of repeated unsuccessful attempts of the information-processing mechanism to complete its own processing. Since the non-adaptively stored trauma is functionally compartmentalized from the appropriate information, the perceived event does not become integrated.

The researches of an der Kolk (1995) and LeDoux (1992) help us to understand the neurophysiology of disrupted information processing and PTSD. Sensory information enters the Central Nervous System via the sensory organs (e.g., eyes, ears, nose, skin). This

information is passed on to the thalamus, where some of it is integrated. The thalamus passes this raw sensory information on to both the amygdala and the prefrontal cortex for further evaluation, with some of the information arriving to the amygdala first, before it is passed on to the cortex. The amygdala interprets the emotional significance of the incoming information. LeDoux (1992) proposes that since input from the thalamus arrives to the amygdala before the information from the neocortex, this earlier arriving sensory information "prepares" the amygdala to process the information at a faster rate than the cortex (van der Kolk, 1995). Hence, the emotional evaluation of sensory input seems to precede the conscious experience.

The information evaluated by the amygdala is passed on to the hippocampus and pre-frontal cortex. The hippocampal system, which is anatomically adjacent to the amygdala, is involved in the evaluation of how incoming information is related in terms of time, space, and previously stored information. Thus, the thalamus, amygdala, hippocampus, and prefrontal cortex are all involved in the integration, interpretation, and storage of sensory information.

However, this integration seems to be disrupted by high levels of arousal. Neuroimaging studies indicate that trauma affects specific structures, e.g. the amygdala, corpus callosum, anterior cingulated and pre-frontal cortex. These are critical to working memory and to the ability to interpret the meaning of incoming stimuli (McFarlane, 1993; van der KoIk, 1997), functions involved in anxiety disorders. Traumatic events signal the amygdala, which responds to the perception of threat.

Where moderate activation of the amygdala enhances the declarative memory mediatedby the hippocampus, extreme arousal disrupts hippocampal functioning. Excess arousal in the amygdala generates emotional responses and sensory impressions that maybe based on fragments of information, rather than on complete perceptions of objects. Memories of these traumatic events, then, are stored as affective states or in sensorimotor modalities, as somatic sensations and visual images, and not integrated into semantic memory. Hence, the information is stored in state specific form, not able to be fully processed and integrated.

This research is consistent with the notion that the nature of a traumatic memory is to be dissociated and to be stored initially as sensory fragments that have poorly developed linguistic components. Memories of the trauma tend, at least initially, to be experienced as fragments of the sensory components of the event: as visual images, olfactory, auditory, or kinesthetic sensations; or intense waves of feelings that traumatized people usually claim to be representations of elements of the traumatic event (van der Kolk, 1995).

Neuroimaging studies add further understanding of how information processing getsdisrupted by trauma. Research found that when recalling a traumatic memory, there is heightened activity in the right hemisphere of the brain, in the areas that are most involved in emotional arousal – the parts of the limbic system most closely associated with the amygdala (van der Kolk, 1995). These are central sites for the experience of anxiety, and has been called the "worry circuit" (van der Kolk, 1995). When recalling a traumatic memory, the Broca's area, the part of the left hemisphere responsible for translating personal experiences into language, seems to be "turned off" (van der Kolk, 1995). Consequently, rather than being able to put the experience into words, traumatized people experience "speechless terror" (van der Kolk, 1995). As described above, the traumatic information is stored as sensory fragments, with emotions experienced as physical states rather than verbally coded experiences free of excessive affective load.

One can hypothesize that altered neurophysiology accounts for the heightened arousal states, like exaggerated startle response, generalized anxiety, intrusive imagery, avoidance, emotional numbness and other persistent psychological and behavioral symptoms which make it hard to achieve symptom remission in persons who have been traumatized.

Eye Movement Desensitization and Reprocessing (EMDR) is a treatment methodology that can facilitate the adaptive integration of traumatic information. Controlled studies of the effectiveness of EMDR on single-trauma PTSD demonstrated that after the equivalent of three 90 minute sessions (i.e., 4.5 hours), 84-100% of the single-trauma subjects no longer met criteria for PTSD (Marcus, et al., 1997; Rothbaum, 1997; Scheck, et al., 1998; Wilson, et al., 1995).

A recent research suggests that after effective treatment with EMDR, subjects show increased activity in the anterior cingulate gyrus and left frontal lobe as well as in changes in scales measuring PTSD symptoms (Levin, et al., 1999)

In fact, neuroimaging studies suggest EMDR treatment may result in neurobiological changes (Levin, et al., 1999). Six subjects with PTSD received three sessions of EMDR. Effects of three sessions of EMDR were evaluated using pre and post treatment Single Photon Emission Computed Tomography (SPECT). It was found that with four of the six subjects, two areas of the brain were more active after EMDR treatment relative to pretreatment: the anterior cingulate gyrus and the left frontal lobe. Subjects had decreased pre frontal lobe activation when they were exposed to their personalized trauma script prior to treatment.

Further, objective and subjective measures of PTSD also showed significant improvement. The authors speculate that the activation of the anterior cingulate and left prefrontal cortex helps the person distinguish between real threats and traumatic reminders that are no longer relevant to current experience. Further, the activation, functioning and increased metabolism of the prefrontal cortex may indicate the assignment of meaning to the emotions associated with traumatic memory, which would facilitate at the same time being able to make sense of incoming sensory stimulation. This increased frontal capacity appears to be reflected in the change in our subjects' accounts of their traumatic memories. Following EMDR treatment, their narratives of the trauma had a much more symbolic quality than before.

Thus, successful treatment seems to have led to an integration of the traumatic memory into the main stream of consciousness. Now the subjects could talk about their trauma without being emotionally overwhelmed and view the trauma as a past event.

Since data indicate that traumatic experiences appear to be initially stored as somatic sensations and affect states that may be inaccessible to semantic processing, treatments that primarily rely on semantic or analytical mental processes may be less effective. Traumatic memories seem to be primarily represented in the right hemisphere and in the limbic portion of the brain, which may make it difficult to process them with verbal therapies alone. Though the mechanisms underlying EMDR are not known, it is speculated that this method may result in the activation of parts of the brain that facilitate integration of traumatic material. It is highly speculative that the changes seen in the SPECT scan reflect any specific effects of EMDR as a therapeutic modality. The neurophysiological changes that resulted from EMDR may be an indication of the changes that would occur following any form of effective treatment. However, as Levin, Lazrove, and van der Kolk (1999) conclude, it is possible that EMDR may yield benefits that traditional insight-oriented therapies lack.

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