A Model for the Flash Technique Based on Working Memory and Neuroscience Research

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Research has shown that the Flash Technique (FT) appears to reduce memory-related disturbance and may reduce symptoms of posttraumatic stress disorder. This paper discusses the connections between FT and eye movement desensitization and reprocessing (EMDR) therapy. In FT, clients remind themselves of a traumatic memory without dwelling on it and focus instead on a positive engaging focus and then blink their eyes when prompted. This paper summarizes numerous models describing how the brain processes traumatic material and presents a model for how FT may work in the brain. It proposes that during the blinking, the patient's periaqueductal gray (PAG) may take over, sensing the reminder of the traumatic memory and reflexively triggering the amygdala. In Porges's neuroception model, the PAG assesses danger without going through the conscious brain. Recent fMRI data show that for patients with posttraumatic stress disorder, there is enhanced connectivity from the amygdala to the left hippocampus. Thus, triggering the amygdala may, in turn, activate the left hippocampus, which may then provide a brief access to the traumatic memory. Given the brief access, there is insufficient time for the amygdala to go into overactivation. The client remains calm while accessing the traumatic memory, thus setting up the prediction error necessary for possible memory reconsolidation. This process is repeated during blinking in FT allowing memory reconsolidation to proceed. This model requires experimental confirmation.

Keywords: flash technique; eye movement desensitization and reprocessing; neuroscience; memory reconsolidation; amygdala-prefrontal cortex circuit; subliminal access to traumatic memory

In this article we make the case that the deliberate eye-blinking in the Flash Technique (FT) may provide very brief access to the traumatic memory to be processed, similar to subliminal messaging. Flash Technique was developed in 2017 with the intention to quickly reduce the client's emotional response to a traumatic memory so that eye movement desensitization and reprocessing (EMDR) can proceed. In this article, we will first give a brief overview of EMDR therapy and FT. We will then review relevant concepts from neuroscience and working memory theory to build our case and offer our thoughts on testing our proposed model for FT with functional magnetic resonance imaging (fMRI).

Overview of EMDR

Eye movement desensitization and reprocessing (EMDR) therapy was first introduced more than 30 years ago. Since then, it has been developed into an eight-phased protocol including history taking, preparation, assessment, desensitization, installation, body scan, closure, and reassessment (F. Shapiro, 2001). Theoretically, EMDR is based on Adaptive Information Processing (AIP; F. Shapiro, 2001; Solomon & Shapiro, 2008). The AIP posits that, in normal circumstances, the brain is predisposed to process an experience to an adaptive resolution. However, "a particularly distressing incident may become stored in

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state-specific form, meaning frozen in time in its own neural network, unable to connect with other memory networks that hold adaptive information" and can be "triggered by a variety of internal and external stimuli" (Solomon & Shapiro, 2008, p. 316). In EMDR, the therapist helps the client to access the unprocessed information and, through bilateral eye movement and other standard EMDR procedures, allows innate AIP to occur. As a result, the previously isolated memory network can be connected to the larger adaptive memory network, and the traumatic memory can be stored in a more adaptive form (Solomon & Shapiro, 2008).

EMDR and Memory Reconsolidation Theory

EMDR effects are consistent with memory reconsolidation (MR) (Ecker et al., 2012; Solomon & Shapiro, 2008). Memory reconsolidation research has shown that when reactivation of a long-term, consolidated memory is accompanied by some violation of what is expected according to the memory, the neural encoding of the memory can become destabilized and labile, allowing memory contents to be revised by new learning during the period of lability. In EMDR, the client accesses the traumatic memory while doing bilateral stimulation, typically eye movement but also biaural sounds or bilateral tapping. According to Ecker, from the perspective of MR, the dual-attention focus in EMDR

> *. . . keeps the client's consciousness anchored and positioned in a safe context outside the memory while attending to the memory's contents. In this state of unmerged attending, the client's other self-states and knowings remain accessible, and existing knowledge that is contrary to the target memory can readily activate into foreground awareness due the brain's automatic detection of mismatches, a background process always scanning current conscious experience. That activation of a contrary knowing in response to the target learning creates the juxtaposition, noted just above, that drives the counter-learning needed for unlearning, nullification and erasure to occur. (Ecker, 2018, pp. 77-78)*

EMDR and Working Memory Theory

EMDR therapy can also be viewed from the perspective of working memory theory (Van den Hout & Engelhard, 2012). Working memory is defined as the brain system responsible for temporarily storing and manipulating information. It is required for all complex cognitive tasks, such as learning, reasoning, problem-solving, and comprehension. The capacity of working memory is limited. When an individual is asked to complete two tasks simultaneously, this taxing of working memory can result in a degradation of performance. During EMDR therapy, when the client is asked to simultaneously attend to the past traumatic memory and eye movements, "there is a competition for the limited working memory resources, producing a deterioration in the quality and vividness of the memory image and related components" (Maxfield et al., 2008, p. 258). Working memory effects such as decreased emotion and memory vividness have been found in many laboratory studies (Landin-Romero et al., 2018). However, no clinical study providing full treatment to a diagnosed sample has evaluated whether working memory effects are related to EMDR's clinical outcomes.

EMDR's Neurobiological Effects

EMDR has also been explored from the perspective of neuroscience in recent years. Rousseau et al. (2019) have shown that, post-EMDR, there were changes in the fear circuit (amygdala and left hippocampus) as well as the right inferior frontal gyrus, the right frontal eye field (FEF), and insula. In addition, there were changes in connectivity between brain structures. There was increased connectivity between left amygdala and the left posterior division of the inferior temporal gyrus, a part of the temporal pole. Furthermore, there was a decrease in connectivity between the left hippocampus and the left superior parietal lobule as well as decreased connectivity between the right insula and the right ventral entorhinal cortex. Rousseau et al. interpreted the changes in the activation of the brain structures, such as the insula, the temporal pole, and the right inferior frontal gyrus, and changes in connectivity, such as between the amygdala and the temporal pole, as related to improvements in fear conditioning post-EMDR. Changes in brain connectivity was also reported by Santarnecchi et al. (2019) for posttraumatic stress disorder (PTSD) subjects post-EMDR and (post trauma-focused cognitive behavioral therapy (TF-CBT) treatment. Specifically, changes in Clinician-Administered PTSD Scale (CAPS) in the subjects across both samples correlated with an increase in connectivity between the bilateral superior

medial frontal gyrus and right temporal pole, and a decrease in connectivity between left cuneus and left temporal pole.

The Flash Technique

In traditional EMDR, clients are asked to access their traumatic memory while doing bilateral eye movement or forms of bilateral stimulation, such as bilateral tapping and bilateral sounds. However, in the case of severe trauma, clients may find the memory too traumatic and may go into an abreaction such as dissociation. In 2017, Manfield et al. (2017) developed the FT to help clients bring down their distress level quickly so that EMDR can proceed. In the FT, circa 2017, clients only need to identify the memory to be processed and then focus on a positive experience. At the prompt "flash," clients recall the memory and then return to the positive memory so quickly that they are not aware of the content or the emotions from the memory. Manfield et al. used the metaphor of moving a finger quickly over a flame. In early 2018 Wong, working with a group of dissociation-prone substance abusers, simplified the process by having clients visualize putting the traumatic memory inside a book and then looking quickly at the book three times when prompted. Wong's work was later published in 2019. Subsequent to the publication of their article (and Wong's FT group work), Manfield et al. further simplified the FT protocol by having clients simply blink their eyes three times when prompted instead of trying to consciously recall the memory (Manfield & Engel, 2018).

The FT generated substantial interest in the EMDR community after its first publication in 2017. Since then, it has been presented at the EMDR Annual Conference in both 2018 and 2019. Currently, it is being offered as part of an online training under the auspices of EMDRIA (Manfield, 2020).

Flash Technique Research

A small random control trial (RCT; Konuk, 2021) evaluated a group procedure using FT, called the Flash Group Protocol. It was compared with the Group Traumatic Episode Protocol (G-TEP, Shapiro & Moench, 2018) and waitlist. Participants had PTSD symptoms related to a recent earthquake, and those in the group conditions received two 120-minute sessions on subsequent days. Results found that the Flash Group Protocol produced a significant decrease in PTSD symptoms at 1 week posttreatment, with effects maintained at one month follow-up. G-TEP also had a significant improvement at 1 month, although not at one week. Both G-TEP and Flash Group Protocol were significantly better than waitlist at 1 month on PTSD symptoms and on a measure of resilience.

A large case study involving 175 healthcare providers impacted by COVID-19-related stress indicated that FT appears to be an effective low-intensity group intervention, significantly reducing memory-related disturbance (Manfield et al., 2021). In their study, Manfield et al. demonstrated a significant reduction in the participants' mean subjective unit of disturbance (SUD) from 7.34 to 3.19, after a 15-minute FT group intervention, within the context of a 1-hour online program. At the end of the 15-minute intervention, 45% of the participants (79 out of 175) reported a posttreatment SUD level of 0, 1, or 2, indicating that the traumatic memory was minimally disturbing or not disturbing at all. In addition, preliminary evidence has shown the possibility that FT may be effective in the treatment of dissociative patients, as evidenced in two case studies (Shebini, 2019; Wong, 2019).

Concepts From Neuroscience and Working Memory Research

Flash Technique is still in its infancy. There are various speculations regarding the putative mechanism for the observed rapid effects. Manfield (2017) suggested that FT might be similar to subliminal messaging, with the client accessing the memory so briefly that he or she was not consciously aware of the details of the memory to be processed. However, it is not clear how the current practice of FT, with the blinking, can be related to subliminal messaging. In addition, while we ask clients to do bilateral tapping in FT, in Wong's FT group for dissociation-prone substance abusers, some clients reported memory processing without doing bilateral tapping (Wong, 2019). The role of blinking and bilateral tapping in FT are issues that need be clarified in order to further the practice of FT.

In this article, we suggest a plausible scenario that the deliberate blinking in FT may provide very brief access to the traumatic memory identified by the client. The model is based on known concepts from working model research and from our current understanding of neuroscience. From neuroscience, we draw upon the operation of the amygdala-prefrontal cortex (PFC) circuit in the brain, as well as the role of the periaqueductal gray (PAG) in the midbrain and abnormal connectivity in brain structures in PTSD

patients. From working memory research, we draw upon the brain's bias towards the most emotionally charged memory. If the deliberate blinking in FT can indeed provide very brief access to the traumatic memory, then some of the published works on treatment of phobia using subliminal access can offer us guidance in the workings of FT. From that perspective, our model can be tested using fMRI on PTSD patients undergoing FT.

The Amygdala-PFC Circuit in the Brain

In the first two paragraphs of this section, we will draw from the work of Arsten et al. (2015) to summarize the basics of the neuroscience of fear response. The amygdala and the PFC play key roles in the brain's response to stress and fear. The amygdala is the brain's threat detector while the PFC, in particular the ventral medial PFC (vmPFC), is the brain's "brake" on the amygdala, regulating emotion and modulating the amygdala's fear response.

The amygdala-PFC circuit is mediated by catecholamines (e.g., epinephrine, norepinephrine, and dopamine). At initial presentation of threat, moderate levels of catecholamines are released which strengthen the PFC and weaken the amygdala to allow regulation of the fear response, allowing the person to have the presence of mind to assess the threat. However, with increasing threat, the level of catecholamine release can rapidly increase to high levels. High levels of catecholamines impair the top-down cognitive functions of the PFC, especially those of the ventromedial prefrontal cortex (vmPFC), while strengthening the emotional and habitual responses of the amygdala and basal ganglia. With high levels of catecholamines, the PFC cannot regulate the amygdala and the brain goes into fear response: fight, flight, or freeze.

It should be noted that it takes time to release high levels of catecholamines in the brain. Thus, in short exposures, for example, in subliminal exposures to a fear trigger, the brain may stay at low or moderate levels of catecholamines, allowing the PFC to maintain emotion regulation over the amygdala during the brief exposure.

Catecholamine levels will drop after the threat is passed, allowing the PFC to regain emotion regulation of the amygdala. However, in the case of overwhelming and uncontrollable threat, such as rape or threat to one's life, a person may develop PTSD, and a meta-analysis of published research had identified elevated levels of norepinephrine, but not dopamine or epinephrine, in PTSD patients (Pan et al., 2018). Published fMRI data have also demonstrated that instead of top-down regulation of the amygdala by the PFC, the brain of a PTSD patient tends to be in a bottom-up mode, with an overactive PAG in the midbrain driving the amygdala towards hypervigilance and exaggerated startle response (Terpou, Densmore, Thome, et al., 2019).

The Reflexive PAG and Innate Alarm System

The PAG is located in the midbrain and is part of the innate alarm system (IAS) in the brain. According to Terpou, Densmore, Thome, et al. (2019, p. 1), "The innate alarm system, a network of interconnected midbrain, other brainstem, and thalamic structures, serves to rapidly detect stimuli in the environment prior to the onset of conscious awareness. This system is sensitive to threatening stimuli and has evolved to process these stimuli subliminally for hastened responding." Porges (2009) suggested that the PAG might be part of the neural network, including the viscera, that provides neuroception for rapid and subconscious assessment of threat.

The work of Luo et al. (2010) demonstrates the presence of a reflexive IAS. Using magnoencephalography and an advanced beamformer source localization technique, Luo et al. found that, with a 300 ms exposure of stimulus, amygdala showed two responses: an early response (40–140 ms) was unaffected by attentional load, and a later response (280–410 ms), subsequent to frontoparietal cortex activity, was modulated by attentional load. The early and late amygdala responses are consistent with a dual neuropathway model. The first, fast pathway is a reflexive but coarse pathway via subcortical structures, that is independent of the attentional load. This pathway is consistent with the IAS described by Terpou, Densmore, Thome, et al. (2019). The second, slower response from the amygdala corresponds to a pathway via cortical structures, the frontal and parietal regions, such that the stimulus is evaluated for its emotional content and thus is affected by attentional load. It should be noted that the 300 ms presentation of stimulus is nine times longer than the 33.4 ms used in the experiment of Siegal et al. (2017) that we will discuss later in this article. With very brief exposures (VBEs), it is not clear whether there will be a second cortical response if there is no conscious emotional awareness of the threat. Regardless, Luo's early response data is consistent with the fast, reflexive action of the IAS based on subcortical structures in the brain.

Abnormal Brain Activation and Connectivity in PTSD Patients

There is evidence from fMRI studies on 21 participants with PTSD that the PTSD patients may have enhanced connectivity between different brain structures: between the PAG and the amygdala and between the amygdala and the left hippocampus (Nicholson et al., 2016). In addition, based on a group of 26 PTSD participants and a group of 20 healthy individuals, fMRI studies showed enhanced connectivity between the PAG and the middle frontal and middle temporal gyrus in the default mode network (DMN) for PTSD participants (Terpou, Densmore, Theberge, et al., 2019). Regression analyses showed a positive correlation between the PAG-right middle frontal gyrus connectivity and state dissociation scores (CADSS). Moreover, regression analyses also showed a positive correlation between the PAG-left temporal gyrus connectivity and avoidance symptom scores (CAPS criterion C). Furthermore, based on the same PTSD and control groups, fMRI studies showed that the PAG may be over-activated in PTSD patients (Terpou, Densmore, Thome, et al., 2019).

It should be noted that the enhanced connectivity in the PTSD brain is consistent with the symptoms of PTSD. With the enhanced connectivity between an overactivated PAG and the amygdala, the amygdala may be on high alert constantly, resulting in hypervigilance and hyperarousal. The left hippocampus provides access to episodic and autobiographical memory. Thus, the enhanced connectivity between the amygdala and the left hippocampus may lead to enhanced access to episodic and autobiographical memory leading to flashbacks and reexperiencing the past. Also, as discussed in the previous paragraph, the enhanced connectivity between the PAG and the right middle frontal gyrus, and the enhanced connectivity between the PAG and the left middle temporal gyrus, are positively correlated to dissociation and avoidance respectively. Thus, enhanced connectivity between the PAG and those two regions may lead to dissociation and avoidance symptoms in PTSD patients (Terpou, Densmore, Theberge, et al. 2019).

Nicholson et al.'s data (2016) also showed that the abnormal connectivity in brain structures could be alleviated even with one session of neurofeedback training: the enhanced connectivity between the PAG and the amygdala and between the amygdala and the left hippocampus. Instead, postneurofeedback training showed increased connectivity between the PFC and the amygdala, suggesting top-down emotion regulation of the amygdala. In addition, Nicholson et al. also found that, consistent with previous research (Sadeh et al., 2014), decreased PTSD symptoms correlated with decreased amygdala-hippocampus connectivity, even though no quantitative data was presented in the article. It is important to note that while changes in brain connectivity can be observed in one session and this may support the use of neurofeedback as an adjunct therapy, it typically takes 20–30 sessions to stabilize/maximize symptom reduction.

In addition, fMRI studies have demonstrated that the PAG, the amygdala and the left parahippocampal area, and the left hippocampus can all be activated via subliminal presentation of threat (Sakamoto et al., 2005; Terpou, Densmore, Thome, et al., 2019; Whalen et al., 1998). As discussed previously, the left hippocampus, including the left parahippocampal area, is associated with episodic and autobiographical memory. In addition, there is an enhanced connectivity from the PAG to the amygdala and then to the left hippocampus in PTSD patients (Nicholson et al., 2016). Thus, a PTSD patient may be able to briefly access the full traumatic memory, via the enhanced PAG-amygdala-left hippocampus connectivity, when the overactivated PAG senses a subliminal trigger.

Neuroscience of Subliminal Presentation of Threat

In this section, we summarize the results of the work by Paul Siegal and his collaborators on the effect of repeated subliminal (33.4 milliseconds) presentation of threat (pictures of spiders masked in pictures of flowers) to a group of spider-phobic subjects (Siegal & Warren, 2013; Siegal et al., 2017; Siegal & Weinberger, 2012). Compared to a similar group of spider-phobic subjects with clearly visible exposures (CVE), there was more reduction in phobic symptoms with the VBE group and the results held after a year (Siegal et al., 2012, 2013).

As expected, fMRI data from the CVE group showed that regions of the brain associated with vision, the occipital and parietal areas, were activated, that is, the spider-phobic subjects could see the spiders. In addition, the subcortical structures associated with emotion/fear, including the amygdala, thalamus, parahippocampal gyri, and hippocampus, were activated. Furthermore, the parts of PFC that support emotion regulation, the ventral medial and ventral lateral prefrontal cortices, were deactivated. Activation of the amygdala and other subcortical structure and the deactivation of the PFC showed that the subjects

were in the fear response, with the PFC unable to provide emotion regulation over the amygdala.

For the VBE group, fMRI data showed that the occipital and parietal regions were also activated. In contrast to the CVE group, *both* the PFC and the subcortical structures associated with emotion/fear, including the amygdala, thalamus, parahippocampal gyri, and hippocampus, were activated. In addition, the activation of the amygdala decreased over time. Thus, for the VBE group, the fMRI data showed that the brain registered the subliminal threat. However, during the VBE, the amygdala did not have enough time to go into overactivation. Instead, as the amygdala responded to the threat, the PFC remained activated, thus maintaining top-down emotion regulation over the amygdala during the very brief presentation of threat. Over time, the PFC might be able to process the spider-phobia to reduce its emotional reactivity.

Siegal's work on subliminal exposure of photos of spiders to spider-phobic subjects may offer some guidance on the use of fMRI for the FT. In his 2017 article, Manfield suggested that FT might be similar to subliminal messaging. If FT involves subliminal access to traumatic memory, we can expect relevant brain structures, for example, the left hippocampus, to be activated. Processing of the traumatic memory may also involve activation of the PFC during the subliminal access to traumatic memory, to provide top-down emotion regulation of the amygdala.

Concepts From Working Memory Research

The working memory is a concept to describe how the brain can choose, retrieve, maintain, and manipulate information from the long-term memory for goal-oriented tasks (D'Esposito & Postle, 2015). Extensive research has been done, both from the psychological and from the neurocognitive perspective, over the past 50 years (see, e.g., Chai et al., 2018; D'Esposito & Postle, 2015). Rather than going into the details of different models, we will highlight a few concepts from the current understanding of working memory that contribute to our model for the FT. Please note that here we are drawing from the large body of working memory research, and are not referring to the previously mentioned work on working memory taxation proposed for EMDR, which represents only a small aspect of working memory research.

In this paragraph, the relevant concepts from working memory research are summarized from D'Esposito and Postle's review (2015). First, in order to work on goal-oriented tasks, the working memory includes an executive control center, possibly the PFC, to retrieve relevant information from long-term memory, to make manipulations on the information to achieve the goal of the task, and to prioritize the information held in the working memory. Secondly, the working memory has the capacity to hold different pieces of information for a task but can focus more attention on the one piece of information to be worked on while paying less attention to other pieces of information, yet holding them within the working memory. Thus, different pieces of information are held in the working memory in different states of activation established by the executive control center through allocation of attention. Thirdly, the brain can switch from one task to a second task while still holding information from the first task in the working memory for some time. In other words, the information previously used is held in some activated state for some time during the interim period (D'Esposito & Postle, 2015; Fuster & Alexander, 1971).

Fourthly, there is the concept of salience. The brain has "biased attentional processing toward emotionally stimulating material content attended by increased sensory responses" (Tyng et al., 2017). Thus, we can expect the brain will tend to seek out and focus on emotionally charged contents in the working memory.

The Central Executive, Default Mode, and Salience Networks

From a neuroscience perspective, the brain has three large networks of connected structures (Lanius et al., 2015): the Central Executive Network (CEN), the DMN, and the Salience Network (SN) (see also Fisher, 2020; Lanius, 2020), The CEN is crucial to verbal learning and executive functioning. It is the main network for goal-oriented tasks, and provides the attention and cognitive capability to focus and work on a task in the working memory. The DMN, consisting of the anterior and posterior medial cortices and the parietal lobes, is the network which takes over when the brain is off-task, that is, when the CEN is not working on a task. It is responsible for self-referential processes, autobiographical memories, and social cognition. The SN is responsible for salience detection, directing the brain toward the most pertinent action, and it integrates input from the amygdala and the PAG. The anterior insula of the SN is crucial to the engagement of the CEN and the disengagement of the DMN, and vice versa, and mediates "the dynamic interplay between externally- and internally-focused attention and cognitive-affective processing" (Lanius et al. 2015).

The work by Nakano et al. (2013) illustrated the action of the SN directing the brain from the CEN to the DMN literally in the blink of the eye. Nakano's data showed, using fMRI, that subjects can switch their attention momentarily with eye-blinking. In their research, subjects watched a funny video and fMRI data showed activation in the subjects' dorsal attention network, which is part of the CEN. However, during spontaneous eye-blinking, event-related analyses of fMRI data showed momentary decrease in the activation of the dorsal attention network: in the FEF and the superior parietal lobe (SPL), and instead, an increase in activation in the subjects' DMN: the medial visual area, the anterior cingulate cortex (ACC), the posterior cingulate cortex (PCC), the angular gyrus (AG), and the insular/secondary somatosensory (SII) cortex. The research of Nakano et al. showed that the SN can switch attention during spontaneous eye-blinking from one task in the working memory to another and then back to the first task, *so quickly that the person has no awareness of the switching and break in attention.*

A Model for the Processing of Trauma With FT

Because of its emotional content, a traumatic memory will be the most salient item when we bring it into working memory, that is, the brain will naturally give the traumatic memory the highest priority and more attention than any other piece of information. In FT, by identifying a traumatic memory without going into details, we consciously put a *reminder* of the memory into the working memory, but also by setting it aside, we consciously give it lower priority and less attention. In addition, by using a positive engaging focus (PEF), we consciously focus the brain on a positive memory or activity, even though the PEF may not be the most salient item in the working memory. Then the PEF is interrupted by a verbal cue such as "flash." The interruption can be purposeful, as when the client is asked to think about the memory very briefly, without even being aware of the emotions or the details of the memory (Manfield et al., 2017), or when the client is asked to briefly recall a symbolic representation of the memory, looking at the book three times (Wong, 2019). In both cases, the client may briefly access the traumatic memory directly or via a trigger/representation of the memory, and then quickly returns to the PEF. The interruption can also be non-purposeful, just by not paying attention to the PEF by blinking three times, as in the current practice of FT. The work of Nakano et al. (2013) has already shown that the SN can momentarily switch from the CEN with its focus on the engaging video to the DMN, during spontaneous blinking and then back to the engaging video, out of the awareness of the person. We hypothesize that, with a reminder of a traumatic memory in the working memory, the SN, which includes the overactivated and reflexive PAG, switches from the PEF to the traumatic memory during the deliberate blinking. On a brain structure level, PAG may sense the reminder of the traumatic memory and reflexively activates the amygdala. In turn, with the enhanced connection between the amygdala and the left hippocampus in patients with PTSD, the brain rapidly activates the left hippocampus (Terpou, Densmore, Theberge, et al. 2019) and briefly accesses the traumatic memory before it goes back to the PEF. During the brief access, the amygdala does not have time to go into overactivation but remains regulated by the PFC. The juxtaposition of the traumatic memory and an activated amygdala regulated by an activated PFC, that is, fear under control in the face of overwhelming threat during this brief access, may provide the prediction error for MR. Repeated very brief access to the trauma memory may enhance the connection between the PFC and the amygdala and may simultaneously allow the MR process to proceed.

Discussion

Our model draws from the experimental work of Nicholson et al. (2016), Siegal et al. (2012, 2013, 2017) and Terpou, Densmore, Theberge, et al. (2019), Terpou, Densmore, Thome, et al. (2019). While we make a plausible scenario for a reflexive access to the traumatic memory via the amygdala and left hippocampus, we will have to confirm this model with fMRI data using procedures similar to the aforementioned authors.

In our model, we emphasize the enhanced connectivity among the PAG, the amygdala, and the left hippocampus, which leads to the activation of the left hippocampus when the PAG senses a reminder of the traumatic memory. On the other hand, other brain structures such as the SN (e.g., the anterior insula and anterior cingulate cortex) and the motor control regions (e.g., putamen and caudate and premotor regions) may also be affected (Siegal, 2017). Changes in the activation in those regions may support the interpretation that the brain has a brief access to the traumatic memory.

Data from Siegal et al. (2017) showed that the amygdala can be "toggled" repeatedly by masked photos of spiders and able to return to a baseline condition and not continue to escalate to a hyperactive state. We

argue that the same is happening in FT as the amygdala is triggered reflexively by the PAG "prior to the onset of conscious awareness," and accessing the traumatic memory (Terpou, Densmore, Thome, et al., 2019, p. 1). Also, clinically, clients do not feel the emotion associated with the traumatic memories during FT. If somehow the brief access results in clients "locking" onto their traumatic memories, then they will feel the emotion. On the other hand, while we hypothesize that processing of the memory during the brief access, there may be other processes that continue in other networks in the brain, after the brief access. This is an area that needs further exploration.

In our model, the role of the PEF is to provide a positive and engaging focus to keep the brain in a calm place initially. Even though the PAG may be overactivated, we hypothesize that it will likely not be triggered by a pleasant focus, and the amygdala will not be start from an over-activated state as the brain access the traumatic memory reflexively. This may explain why we can have so many different choices for PEFs, including body scan, happy memories, funny videos, talking about something of personal interest to the patient, music, and even dancing, as long as patients find them positive and engaging.

Furthermore, in this model, the deliberate blinking is a brief interruption from a pleasant PEF, allowing the brain to briefly and reflexively access the traumatic memory on its own via the PAG-amygdala-hippocampus connection, outside of the consciousness of the client. Since most people blink spontaneously a few times a minute, based on this model, the same process may also occur during spontaneous blinking. This can be an area for further exploration.

It should also be noted that FT, by not focusing on the traumatic memory, may seem at odds with published works on taxation of working memory which showed that recall of an unpleasant memory during working memory taxation resulted in reduction in memory vividness and emotionality (e.g., Cuperus et al., 2016; Van Veen et al., 2016). However, *our model is based on the brain over-activation and enhanced connectivity in PTSD subjects* (Terpou, Densmore, Theberge, et al., 2019; Terpou, Densmore, Thome, et al., 2019), which is absent in healthy subjects. Thus, the results of working memory taxation research, using healthy subjects, may not be germane to our model for FT.

There is also research showing that noradrenaline facilitates MR and that a noradrenergic β-blocker, propanolol may degrade MR in EMDR (Littel et al., 2017). This may provide a test for whether MR occurs with FT. fMRI measurements on two matched groups of PTSD clients doing FT, one group taking and the other group not taking propanolol, may show different results. Assuming that MR occurs in FT, then there will be reduction in over-activation and connectivity in the no-propanolol case and no change in over-activation and connectivity in the propanolol group.

Using our model, we can explore the role of bilateral stimulation in FT. In our model, it is the brief interruption in attention that sets up the brief access to the traumatic memory. While we encourage clients to do bilateral tapping during FT, the tapping does not play a part in the brief access to the traumatic memory. This may explain why some clients can process their traumatic memories without bilateral tapping (Wong, 2019).

Our discussion on MR and fMRI data begs the question of how MR relates to neuroscience in the area of trauma/PTSD. Specifically, are there a core group of brain structures whose abnormal activation and/or connectivity account for the core PTSD symptoms? If so, what is this core group of brain structures? Conversely, does trauma recovery result just by bringing the activation and connectivity of this core group of brain structures back within normal limits? If so, MR would be a result of changes in brain activation and connectivity. On the other hand, what is the interplay between changes in the structure of memory and changes of activation and connectivity in brain structures? Trauma involves learning from a painful experience, resulting in changes in brain structures. Memory reconsolidation involves new learning, a juxtaposition of the painful memory with contradictory information/experience. From this perspective, new learning drive changes in brain activation and connectivity. The question of how MR relates to neuroscience in the area of trauma/PTSD is an interesting and important one but is beyond the scope of this article.

Limitations

This model is an attempt to understand the basic mechanism of the FT. It extrapolated from research that used fMRI data from clients with PTSD as well as that from experiments using VBEs to images of threat. While the model is based on existing fMRI data, it needs confirmation by fMRI data from patients with PTSD undergoing FT.

Future Research

In our model, we suggested that the deliberate blinking, together with the brain's bias towards the most emotionallycharged memory, the hypervigilance

from an overactivated PAG, and the enhanced connection between the amygdala and the hippocampus, may set up a very brief access to the traumatic memory. In addition, during this brief access, the PFC remains activated, providing top-down emotion regulation of the amygdala. These two points can both be collaborated with fMRI data from patients with PTSD undergoing FT.

Firstly, we need to explore the connectivity in the brain, pre-FT session, using fMRI, to confirm the pre-FT condition of the patient's brain. Based on Nicholson et al.'s work (2016), we expect to see enhanced connectivity between the PAG and the amygdala and between the amygdala and the left hippocampus in our PTSD subjects before treatment, consistent with hypervigilance and enhanced access to the traumatic memory. Confirmation of enhanced connectivity will support our hypothesis that enhanced connectivity is present as an initial condition, and it may set up a very brief access to the traumatic memory during deliberate blinking.

Secondly, we need to further explore the very brief access to the traumatic memory with fMRI measurements while PTSD patients are undergoing FT. While the fMRI measurements cannot confirm the brief access, they may be able to show brain activation consistent with a brief access to the traumatic memory, similar to the data from Siegal et al. (2017). In addition, we can do similar fMRI measurements while patients with PTSD are presented with subliminal triggers of their traumatic memories, similar to Siegal's work with spiders. Consistency between these two sets of fMRI measurement may put this model on a stronger experimental footing.

Thirdly, we can do post-FT fMRI measurements and collect clinical data, such as SUD scores for reactivity to traumatic memories as well as established PTSD symptom measures. This can be done over a series of FT sessions, while patients with PTSD process multiple traumatic memories. While these measurements do not directly address the brief-access tenet of our model, it will add to our knowledge base on the relationship between symptom reduction and changes in brain activation and connectivity in PTSD patients.

Conclusion

With this model, we offer a plausible mechanism whereby clients can very briefly access their trauma memory while an activated amygdala remains regulated by an activated PFC. We propose the juxtaposition of a trauma memory with an amygdala regulated by an activated PFC sets up the prediction error for reconsolidation for the traumatic memory. The model was based on recent published fMRI data from experiments with VBE to photos of spiders for spider-phobic subject. The model also draws from fMRI data showing abnormal brain connectivity data in patients with PTSD. As such, this model can be confirmed with fMRI data from patients with PTSD undergoing FT. We hope that this model can offer a rough roadmap for experiments that can further our understanding of FT as it relates to neuroscience. It is hoped that more FT/trauma research using fMRI can shed more light on the process of recovery in highly traumatized patients.

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