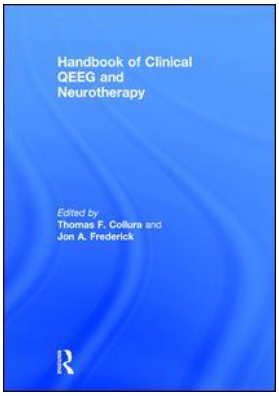

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sLORETA NEUROFEEDBACK AS A TREATMENT FOR PTSD

Nir Getter, Zeev Kaplan and Doron Todder

Abstract

In this chapter we will discuss the localized approach for neurofeedback practice using the standardized low resolution tomography algorithm as applied to patients suffering from post-traumatic syndrome. PTSD is a chronic, severe and disabling mental disorder resulting from the exposure to specific, prolonged or a series of threatening events in which the individual experiences intense anxiety due to immediate danger to one's self or exposure to other people's injury, suffering and death. Providing a relief for these people's suffering is a major concern in most of the modern citizen and military healthcare systems. In this chapter we will use the neuropsychological theoretical framework for the understanding of PTSD suggesting that these symptoms are the cognitive and behavioral consequences of post-traumatic altered functioning of the fronto-temporal limbic network. In our discussion we put an emphasis on the amygdale and the ventromedial prefrontal cortex (vmPFC) and their connectivity properties. We will suggest an improved approach to the traditional alpha-theta neurofeedback practice by a localized tomographic neurofeedback that is based on the advanced EEG standardized low resolution tomographic method (sLORETA). Having the patient practice the localized brain activity in the vmPFC at the theta band power we expected the resetting of the fronto-temporal limbic network to a more healthy state providing the patient with anticipated symptoms relief. We present the case of one patient suffering from PTSD resistant to other treatments who was practicing sLORETA neurofeedback treatment. This patient's intrusion symptoms were improved after 22 sLORETA neurofeedback meetings. Furthermore, we demonstrated executive function and memory tasks performance improvement after the last session compared to baseline.

Post-Traumatic Stress Disorder

Clinical Perspective

The Diagnostic and Statistical Manual of Mental Disorders 5 (DSM-5) define PTSD as a pathological response to an event in which one is exposed to a serious threat of injury, death and then experiences extreme fear, helplessness or horror. This disorder is characterized by three symptom clusters: (1) The re-experiencing symptom cluster is characterized by recurrent and intrusive recollections, dreams of the trauma and flashbacks. (2) The hyper arousal symptom cluster is characterized by an enhanced startle response, sleep and concentration difficulties, problems with anger management,

hyper-vigilance for danger and a sense of a foreshortened future. (3) The avoidance symptom cluster includes symptoms such as an inability to remember aspects of the event, extreme distress and avoidance of cues even remotely related to the actual trauma and emotional numbing with difficulties feeling positive emotions.

Neuronal Network Implicated in PTSD

Fear Conditioning

Fear conditioning is the process by which a previously neutral conditioned stimulus (such as a tone or visual stimulus) is presented immediately before an aversive stimulus (such as a shock) and predicts its onset. After repeated presentations, the conditioned stimulus can elicit a fear response, such as freezing, increased startle or increased skin conductance (SCR). For instance, an anxious individual may learn that a stimulus or situation is threatening, which may then lead to pathological reactions (e.g., phobias and post-traumatic stress disorder). Support for this view has emerged from both animal models for PTSD (H. Cohen & Richter-Levin, 2009) and neuroimaging studies in humans (Phan, Wager, Taylor & Liberzon, 2002).

The amygdala, previously found to be implicated in emotional behavior (Phelps & LeDoux, 2005), shows altered activity in animal PTSD models in comparison to controls. Different studies have shown that PTSD patients show exaggerated activation in the amygdala in response to both traumatic reminders (e.g., pictures from the traumatic scene) and more general predictors of threat (e.g., fearful facial expressions). In addition, a positive relationship between symptom severity and amygdala activation have been reported (Pissiota, Orjan, Fernandez, Fischer & Fredrikson, 2000; Shin et al., 2004).

The amygdala's activity is modulated by input from the ventromedial prefrontal cortex (vmPFC), which is thought to inhibit expression of conditioned fear following extinction training (Phelps & LeDoux, 2005). Activation in the vmPFC demonstrates a role for this region in the automatic regulation of fear prior to fear extinction. Increased vmPFC activity was observed primarily in low trait anxious individuals and was inversely correlated with SCRs (Indovina, Robbins, Núñez-Elizalde, Dunn & Bishop, 2011).

Results from a prospective functional magnetic resonance imaging (fMRI) study have established the formation of a physiological coupling between the right ventromedial prefrontal cortex (vmPFC) and the right amygdala after exposure to highly stressful events (Admon et al., 2009). The strength of this coupling had a positive correlation with the magnitude of the reported post-traumatic symptoms. Interestingly, a unique lesion study (Koenigs et al., 2008) compared the size of the vmPFC lesion in brain injured trauma survivors and the risk for PTSD. Koenigs et al. concluded that the vmPFC plays a significant role in contributing to the development of chronic PTSD. Low activity or omission of this area due to physical damage might serve as a protective factor against the development of PTSD (Koenigs et al., 2008).

These two studies present contradictory evidence regarding vmPFC's role in the formation and maintenance of PTSD. One suggests that the absence or a reduction in vmPFC activity might protect from the symptomatology. The other suggests that a reduction in vmPFC activity might contribute to symptom severity (Admon et al., 2009; Koenigs & Grafman, 2009; Koenigs et al., 2008).

A model suggested by Rainnie and Ressler (2009) can explain this contradiction. The model emphasizes the different roles of the vmPFC in various processes of fear conditioning (i.e., acquisition and retaining of fear responses). Neural activity in the amygdala and the vmPFC may usually be under mutual inhibitory control. These physiological connections may cognitively reflect executive "top down" control of the vmPFC on the amygdala. A breakdown of such process could contribute to the psychopathology of PTSD by means of retention of a hyper-vigilant state. Therefore, hypoactive vmPFC may contribute to the retention of PTSD symptoms.

Alternatively, the absence of vmPFC activity immediately following a traumatic event may prevent an individual from developing PTSD by means of interfering with the process of fear conditioning. Fear conditioning is normally formed at the time of the traumatic stressor and requires both the amygdala and the vmPFC to form the fear acquisition. Therefore, damage to the vmPFC will protect the individual from developing PTSD for no acquired fear conditioning is formed.

VmPFC's role in the modulation of fear is further emphasized by Etkin, Egner, and Kalisch (2011). This line of research strongly suggests that dorsal mPFC structures are implicated in threat appraisal and the expression of fear whereas mPFC structures are involved in the inhibition of conditioned fear (Etkin et al., 2011). Failure to activate the appropriate ventromedial prefrontal cortex structures among PTSD individuals may contribute to the retention of hyper-vigilance by blocking extinction.

Further data reviewed by Etkin et al. (2011) suggest a controlled conscious top down regulation, like emotional conflict regulation, uses structures at the ventral PFC (such as vmPFC) to inhibit negative emotional processing in the amygdala. The vmPFC might thus perform a generic negative emotional inhibitory function that can be triggered/elicited by other regions (e.g. dorsal ACC and mPFC and lateral PFC) when there is a need to suppress limbic reactivity. One might speculate that a dysfunction in this circuit might contribute to the PTSD dysfunction in regulating negative emotional conflict. Unable to regulate and suppress the emotional arousal associated to trauma relevant information, the PTSD affected individual might choose to avoid trauma related arousal eliciting stimuli altogether. The limbic reactivity might contribute to hyper-vigilant responses, and affect memory processes leading to an intrusion, as well. Dysfunction in the ventromedial PFC structures and their connections may contribute to all symptom clusters.

To summarize, according to the fear acquisition theory in PTSD, the hyper-responsiveness of the amygdala combined with hypoactivity observed in the vmPFC can explain the individual's hypersensitivity to threatening stimuli. The question of whether increased activity in the vmPFC will result in down-regulation of amygdala activity or PTSD symptoms is still to be answered. This perspective focuses on the hyper-vigilance cluster of symptoms and therefore covers only part of the PTSD phenomenology.

Neurocognitive Perspective

In a neurocognitive perspective, it is commonly assumed that the symptom clusters of re-experiencing and avoidance are in effect a manifestation of memory impairments. These impairments can be the result of the exposure to trauma but can also present prior to the traumatic event and therefore serve as a pre-traumatic risk factor. Specifically, the autobiographical memory is hypothesized to be compromised among PTSD diagnosed population, therefore eliciting the above symptoms.

Autobiographical memory is a general term describing an array of cognitive mechanisms dedicated for the encoding, retention and retrieval of information from personally experienced events. The data processed by these mechanisms includes sensory and perceptual data from the event together with feelings and thoughts activated at the time of the event. According to the encoding specificity principle suggested by Tulving and Thomson (1973), a key property of these data is their contextual relevance at the time of encoding and the time of retrieval. Therefore, perceptual and higher cognitive information that does not fit correctly with the context of the experienced event will have lesser probability to be encoded. Moreover, recollection of details from a past event will be more probable when the context of the situation is congruent with the context of the retrieved event. Recent findings from animal studies (H. Cohen, Liberzon & Richter-Levin, 2009; Je ek et al., 2010) support a relationship between stress exposure and the utilization of contextual cues in both the encoding

phase and retrieval phase. Failing to use context while retrieving episodic memory had been further supported by the results obtained from a study comparing memory performance of PTSD patients with controls. Guez et al. (2011) compared performance in a context sensitive recall task (a pair association recall) and a free, non-context sensitive recall task (free recall). The results have indicated that memory for the paired items remained intact while memory for the pair's arrangement was compromised. These findings were interpreted as representing the PTSD group's difficulties in using context for retrieving an existing memory (Guez et al., 2011). Together with other findings from neuroimaging studies (Acheson, Gresack & Risbrough, 2012; Werner et al., 2009), it is compelling to attribute intrusion and avoidance symptoms of PTSD to the difficulties of this population in retrieving memory by using its context. In PTSD, paradoxically the alterations in memory for the traumatic event can take the form of both intrusive recollections and difficulties with intentionally retrieving aspects of the traumatic event. Furthermore, individuals with PTSD are often described as showing fear responses to trauma reminders outside of contexts in which these cues would reasonably predict danger (Acheson et al., 2012).

The structure most attributed to autobiographical memory dysfunction is the hippocampus (Cipolotti & Bird, 2006). It has been suggested that emotion plays a key role in the quality of memory integration preformed at the hippocampus (Dere, Pause & Pietrowsky, 2010). It has been shown that the strength of neuronal activity in the hippocampus and amygdala is correlated both during the encoding and the retrieval of emotional information (Kensinger & Corkin, 2004). Imaging studies in PTSD suggested compromised network of limbic structures including the hippocampus, the vmPFC and the amygdala (Nutt & Malizia, 2004). Hippocampal volume decreases after psychological stress (Woon, Sood & Hedges, 2010) and recent studies in animal models implicate a key role to the connectivity between the hippocampus and vmPFC specifically in the integration of context into the stored and retrieved memory (Je ek et al., 2010; van Kesteren, Fernández, Norris & Hermans, 2010). Together with the pairing of amygdala and vmPFC, an fMRI study in healthy medicine corps cadets by Admon et al. (2009) also documented a relationship between the hippocampus and the vmPFC formed after stressful events. There seems to be a correlation between the magnitude of post-traumatic symptoms as reported by the subjects and strength of the relationship between the amygdala and vmPFC. In another longitude study PTSD patients underwent two fMRI scans, 6–9 months apart, while viewing fearful and neutral faces in preparation for a memory test (administered outside the scanner). At the end of the protocol, symptom levels correlated positively with memory-related fMRI activity in the amygdala and ventromedial prefrontal cortex (vmPFC) (Dickie, Brunet, Akerib, & Armony, 2011).

Taken together, findings from neuroimaging studies suggest a specific network compromised in PTSD with the amygdala, the vmPFC and the hippocampus as key neural structures. We believe that the vmPFC has a significant role in the functions of contextual encoding and retrieving of the autobiographical memory.

Intermediate Summary

The fear acquisition theory and the autobiographical theory are two proposed complementary perspectives for the symptomatology of PTSD. Fear acquisition is attributed to hyperactivation of the amygdala and to the exaggerated sensitivity of PTSD individuals to threatening signals. Intrusion symptoms such as flashbacks need no threatening signals to appear and might be explained by the autobiographical model for PTSD as a failure in fitting the retrieved memory to the current context. Investigating both perspectives using different approaches has pointed to the same functional network with the medial prefrontal cortex, the amygdala and the hippocampus as its central interconnected structures. This implies that an intervention in the activity of one structure can affect the whole network with a reduction in symptoms outcome.

Neurofeedback Treatments for PTSD

Traditionally PTSD was considered to be a type of anxiety disorder. Neurofeedback treatments for anxiety aimed primarily at increasing brain activity in the alpha band. Hardt and Kamiya (1978) assigned 16 students to high and low trait anxiety groups by means of MMPI scores and trained them to increase and decrease their alpha band activity. The results suggested a link between alpha changes and anxiety rating changes in the high anxiety group but not in the low anxiety group. Since this original study other studies have also demonstrated the efficacy of neurofeedback, specifically alpha upregulation, as an anxiety treatment.

The most documented approach for the treatment of PTSD via neurofeedback training is the protocol named alpha-theta. This neurofeedback protocol was initially developed for the treatment of alcoholism (Peniston & Kulkosky, 1989) and later adopted for the treatment of PTSD (Peniston & Kulkosky, 1991). Patients participating in this protocol had a surface electrode attached to their scalp at a posterior midline location (Pz in the 10/20 electrode positioning system). Both alpha band (8–12Hz) and theta band (4–7Hz) are filtered from the EEG signal, and each signal band power is marked by a different tone. The patient task is twofold: their main task is to upregulate both alpha and theta activity by increasing both types of tone prevalence. The secondary goal is to maintain a relatively equal frequency of "alpha tone" compared to "theta tone." To achieve the second goal a "cross-over" pattern has to emerge in which theta waves gradually increase, and the alpha waves gradually decrease. This pattern is a marker for a state of consciousness believed to increase the probability for the release of repressed imagery content.

PTSD treatment by means of alpha-theta protocol was documented in a hallmark study published by Peniston and Kulkosky (1991). Twenty-nine male Vietnam combat veterans with a comorbid diagnosis of PTSD and alcohol abuse were assigned to either neurofeedback protocol or a control group. The neurofeedback protocol combined the alpha-theta practice with a relaxation program. The participants in the control group received psychotropic and behavioral therapy. All participants continued their regimen of psychotropic drugs during this study. Results indicated a reduction in the consumption of psychoactive medication by the end of the treatment compared to control. All participants from the experimental group reduced their consumption of drugs compared to only one participant from the control group. Also, by comparing MMPI questionnaire profiles of participants before and after treatment, both groups showed decreases on the schizophrenia scale, but only the experimental group showed reductions in hypochondriasis, depression, hysteria, psychopathic deviation, paranoia, psychasthenia, hypomania, introversion and the PTSD subscales. A follow-up study indicated low relapse rates in the study group compared to control. Only three of the 20 original cohorts had relapsed to alcohol by 26 months after (Peniston & Kulkosky, 1991).

Graap and Freides (1998) raised two questions regarding the work of Peniston. First, are different published articles reporting independent samples? Second, what was the clinical status of the patients prior to treatment and what is the mechanism underlying the alpha-theta protocol? At least the last question can be, for some extent, answered by the work of Egner and Gruzelier (2009) on healthy high performing musicians, actors and people suffering social anxiety disorder. According to these authors, while the main benefit of alpha training is a relaxation, the alpha-theta protocol helps the participant to induce a "hypnogogic conscious state." This served as a state for re-experiencing and reprocessing past traumatic events. "It is as though the patient was capable of integrating past traumatic experiences by coping with previously unresolved conflicts represented in the essential anxiety-free images and memories generated during the theta state of consciousness" (Gruzelier, 2009, p. 103). Neuroanatomically, these authors propose enhanced circuit connectivity, especially of frontal structures to more limbic and meso-limbic structures after alpha-theta training. Taken together that alpha-theta enhance frontal-limbic networks connectivity and the biological approach to PTSD symptomatology described above, it is reasonable to view the mechanism behind alpha-theta therapy

for PTSD as a reactivation of the broken inhibitory connection between the frontal structures such as the ACC or vmPFC and the amygdala and hippocampus.

Low Resolution Tomography Neurofeedback for the Treatment of PTSD

A major shortcoming of traditional neurofeedback, such as the alpha-theta protocol described, relates to the limited information provided by a single electrode placed on the scalp. In conventional neurofeedback, electroencephalographic (EEG) activity is recorded at a particular scalp location. This electrical activity recorded by a single electrode represents not only below the cortical area but the sum of all neuronal activity that is detectable by any given electrode. Therefore, at the single scalp site of recording there is a weighted accumulation of all electrical signals in the brain sphere that cannot be separated.

This limitation can be overcome by using more than a single electrode recording and a low resolution electromagnetic tomography (LORETA), which is a mathematical process to extract the source of the recorded data. LORETA is widespread linear, discrete, instantaneous, full-volume family of an inverse solution proximity for brain electromagnetic measurements (Pascual-Marqui, Esslen, Kochi & Lehmann, 2002). Whereas EEG is a measure of electric potential variations on a two dimensional surface, LORETA estimates the current density in a three dimensional space that results in the potential divergence on the scalp. sLORETA is an evolutionary development of the original LORETA algorithm. This novel algorithm suggests more reliable and zero localization errors compared to the old one (Pascual-Marqui, 2002).

EEG tomography directed biofeedback correlates the physiological signal with a constant feedback signal; however, the physiological signal is defined as the current density in a specified region of interest (ROI) calculated by means of sLORETA algorithm. This allows the continuous feedback signal to become a function of the intracranial current density and to co-vary with it.

Congedo, Lubar and Joffe (2004) established a method for extracting and providing feedback on intracranial current density, and carried out an experimental study to ascertain the ability of the participant to drive their own EEG power in a desired direction by means of this tomographic EEG biofeedback. The authors demonstrated that healthy participants have the ability to drive the current density of their own Anterior Cingulate Cortex (ACC) (a subregion of the medial prefrontal cortex; mPFC) in a desired direction using LORETA directed biofeedback.

Other studies used tomographic EEG biofeedback in clinical populations such as antisocial personality disorder (Surmeli & Ertem, 2009) and chronic pain (Ozier, 2010). These studies reported successful alteration of the brain activity in the targeted anatomical location with no side effects.

A neural network that includes the amygdala, the hippocampus and the medial prefrontal cortex is responsible for emotion regulation and emotional behavior among healthy population. A deficit in this network serves as a general neurophysiological account to both behavioral and cognitive deficits in PTSD. This research proposal focuses on the activity of the ventral structures of the medial prefrontal cortex and its relation to amygdala activity.

We propose that, among PTSD patients, the activity of this network is shifted to a novel, inflexible homeostatic state in which the medial prefrontal cortex is mainly hypoactive, and the amygdala is hyperactive.

The intrusions can be explained by a failure to control the memory retrieval. We will adopt the autobiographical memory retrieval failure hypothesis for explaining the intrusion symptoms cluster. The medial prefrontal cortex is thought to be implicated in executive functions and as such the initiator of a "top down" control over lower cognitive mechanisms. Using a context for the appropriate selection of memory representation for retrieval is considered one of the "top down" processes affected in PTSD. We will hypothesize that alleviating activity in the vmPFC will improve the subjects' functioning in contextual retrieving tasks (Guez et al., 2011) together with mitigation in the intrusion symptoms.

sLORETA Neurofeedback in PTSD

A compromised neural network responsible for emotional behavior serves as a general neurophysiological explanation for both behavioral and cognitive deficits. This network includes the amygdala, the hippocampus and the medial prefrontal cortex. According to the classical fear conditioning model for PTSD, amygdala hyperactivity can explain the symptoms from the hyper-vigilance and the avoidance cluster. Following this model, increasing vmPFC activity will inhibit the activity of the amygdala and will result in symptom reduction together with an observable reduction in the sensitivity to threatening stimuli. Changes in the vmPFC following treatment and symptom reduction have been documented (Dickie et al., 2011). Following this, LORETA neurofeedback (LNF) can be attempted to alter neural activity of vmPFC regions in a chronic PTSD population.

In the suggested protocol, our objective was to practice an increase in brain activity at the theta (4–8Hz) band. An increase in theta activity is related to an increase in the fronto-limbic network connectivity (Denham & Borisyuk, 2000). Specifically, increase in theta activity reflects an enhanced ability to encode new information (Klimesch, 1999), the conscious feeling of knowing and the later accurate retrieval of these memories (Klimesch et al., 2001). Enhancement in anterior theta activity also correlates with an increase in hypnotic susceptibility (Brady & Stevens, 2000). In another study, differences in theta activity were observed when comparing a normal sample and a PTSD sample in response to watching emotional pictures or neutral pictures (J. E. Cohen et al., 2013). As mentioned previously, PTSD symptoms can be explained both by the fear acquisition theory and the neurocognitive model. Briefly summarizing, according to the neurocognitive model, exposure to a traumatic event form a recurrently retrieval of its memory causing an emotional distress. According to the fear acquisition theory in PTSD, the hyper-responsiveness of the amygdala combined with vmPFC hypoactivity may explain the individual's hyper-sensitivity to threatening stimuli. Given the possible therapeutic effects of increasing theta in PTSD patients, we elected to train these patients via neurofeedback to increase theta band activity in vmPFC.

Participants

Five PTSD diagnosed patients (4 male) from the PTSD clinic at the Mental Health Institute in Beer Sheva were referred to the study by their attending physician. Participants with closed or open brain injury were excluded. Four patients had above high school education. Traumatic event varied between the patients (car accidents, military actions and terrorist attack).

Procedure

Participants watched a sitcom episode while wearing a 19 electrode EEG cap attached to an EEG-200 amplifier. The signal was fed into a computer running the software Brain Tuner that online calculated the sLORETA source distribution of the participant's brain electrical activity. The software then isolated the current density in the bilateral vmPFC, at the theta band. The vmPFC defined by the sLORETA software (Pascual-Marqui, 2002) was the combination of voxels from the "medial frontal gyrus" (Brodmann area 11; 30 voxels) and Orbital gyrus (Brodmann areas 18, 13; 31 voxels); therefore our definition for vmPFC includes 61 sLORETA voxels.

The LNF practice had two phases. In the first period, the treatment goal was determined by recording 3 minutes of the vmPFC current density fluctuations and taking the 80th percentile of the current density distribution of this baseline as the participant's goal for this session. When the goal had been fixed, the practice phase immediately begun. The video quality of the television display was manipulated so that for current density higher than the goal, the video quality was sharp. But when the activity dropped under the goal, the video quality was degraded in correlation with the amount of discrepancy of the theta activity from its goal.

Results

Three minutes of open-eye EEG (sampled at 256 Hz and digitally bandpass-filtered to between 1 and 40 Hz) was recorded before and after every LNF session. Using the paired nonparametric voxel-wise statistics implemented in the LORETA-KEY statistical software package, we analyzed the average difference of the source distribution at baseline, before the beginning of the LNF sessions and after the last LNF session. A statistically significant increase in vmPFC activity was recorded ($t = 3.89$, $p < 0.05$) at resting time with eyes open. Therefore, we could demonstrate a specific effect of LNF on the targeted brain structure. When analyzing differences in beta band activity we found that the most significant difference in the source distribution is in Brodmann area 40, but other less significant differences were distributed on other Brodmann areas. This result can be interpreted as a nonspecific effect of the LNF procedure.

We also investigated the effect of LNF on connectivity between the frontal region that the participants trained and other regions, specifically limbic regions. Using the Neuroguide statistical package we set the orbitofrontal cortex (an overlap region with vmPFC) as a seed and calculated its current density correlations to other regions. In a paired t-test for the difference in correlation coefficients before compared to after NBF, we found a significant increase in correlation between the orbitofrontal cortex and other brain regions only in the theta band and not in other frequencies. This correlation increase can be interpreted as an increase in connectivity after one session of LNF, specifically in the theta activity that was the target of this protocol.

This finding represents the first attempt to use LNF as treatment for severe chronic PTSD patients. We demonstrated the validity of LNF and presented proof of concept for the ability to alter the network underlying PTSD symptoms as described previously. The effect of this LNF protocol on patients' PTSD symptoms still needs to be evaluated. In the next section, we present a case study that will demonstrate the effect of LNF treatment on one of this study participants.

Case Study—Alona

Alona, age 55, married with 2 children, lives in the southern city Beer Sheva. On August 31, 2004, Alona was on her way to work when a suicide bomber blew up the bus she was on. Alona was saved from serious injury only because she bent down to pick up her bag at the exact moment the explosion occurred. Seven years later at the time of the research (2013), she reports a severe re-experiencing of the moments after the explosion both in frequent nightmares and during waking hours. She cannot get on a bus and specifically cannot get on the bus line that was bombed. When she tries to face her fears by attempting to go on this bus line, she suffers a severe physiological reaction when the bus approaches the location where the bomb exploded and has to leave the bus. She suffers from concentration problems, mood swings, feeling always alert and easily startled, always on the edge of outburst. She avoids going to any social encounters and had decreased her participation in family gatherings.

Given her intense reaction to the event, Alona was diagnosed with PTSD and started treatment at the Post Trauma Clinic in the Mental Health Center of Beer Sheva. In the seven years following the event Alona received a behavioral cognitive therapy (Prolonged Exposure protocols) as well as insight-oriented psychotherapy and was pharmacologically treated with a variety of antidepressant. In spite of all the efforts, the post-traumatic symptoms continued to impair Alona's life, and her PTSD was estimated to be resistant and not responsive to currently practiced therapies. We invited Alona to participate in a clinical study aimed at reducing her symptoms by teaching her to control specific brain activity in the vmPFC.

Alona was seated in a comfortable armchair in a noise attenuated room while her brain's electrical activity was recorded by means of a 19 electrode EEG cap. Data attained from the recording was processed online, and the current density (CD) in the bilateral vmPFC was calculated by means of the sLORETA algorithm. A video and audio clarity was altered by means of a device connected to the

video and audio input of a television set. Alona watched a 30-minute video with natural emotional valence. Reward for fulfilling the current density goals of the vmPFC was applied by means of a clearer picture (less distorted) and clearer sound (with less white noise). This session protocol was repeated twice a week for a total of 22 neurofeedback sessions.

To measure the clinical treatment outcome, a trained psychiatrist completed with Alona the CAPS interview (Blake et al., 1990) before the first neurofeedback session and after the last neurofeedback session. Alona also completed a neurocognitive test battery that included memory, visual perception, problem solving and executive function tests, before and after the neurofeedback set. Figure 16.1 shows neuropsychological measurements before compared to after the neurofeedback set. Alona's executive function, attention and verbal performance improved dramatically while no change was found in other cognitive domains such as memory functions, visual spatial and motor functions. Furthermore, Table 16.1 compares Alona's baseline CAPS score to her score at the end of the treatment protocol. Alona reported a marked reduction in the frequency of intrusion symptoms (from score 14 to 4) as well as a reduction in avoidance scores (from 12 to 9). These changes are demonstrated by two anecdotal reports from Alona. First, after approximately 15 neurofeedback sessions, Alona was able to get to the hospital by the bus line that was the target for the terrorist attack. Prior to the neurofeedback treatment, she was unable to travel on this line past the junction where the terrorist detonated the bomb. Second, Alona reported a marked reduction in agitation at a fireworks display on the Israeli Independence Day. Previously she could not stay near the square where the fireworks display took place.

We believe that these results show that elevation of vmPFC activity by means of sLORETA neurofeedback training resulted in a reduction of some of Alona's PTSD symptoms.

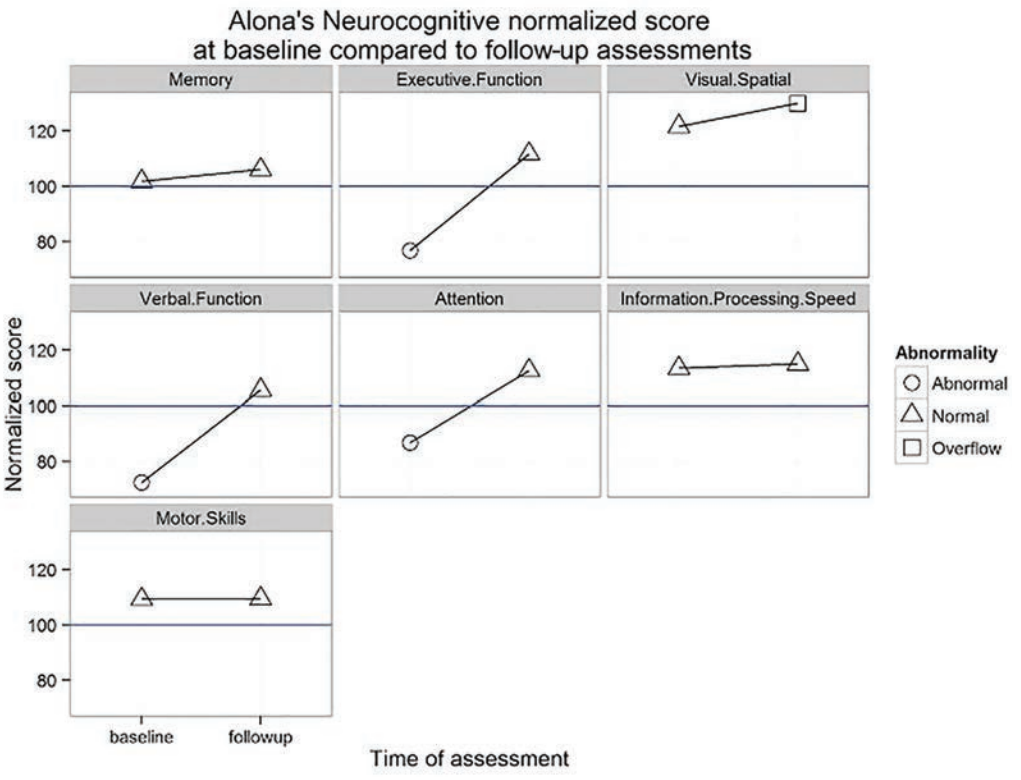

Figure 16.1 Alona's neurocognitive normalized score at baseline compared to follow-up assessments. Higher scores reflects better performance. Normal population score is 100.

Table 16.1 Alona's CAPS score at baseline (Pre) compared to follow-up assessment (Post). Higher score mean more severe and frequent PTSD symptoms.

Symptoms Group	*Frequency/Severity*	*Pre*	*Post*
Intrusion	F	14	4
	S	10	10
Hyper-vigilance	F	3	2
	S	3	2
Avoidance	F	12	9
	S	10	9

Summary

In this chapter, we discussed two converging models for the explanation of PTSD phenomena. Fear conditioning serves as a behavioral explanation of PTSD while the autobiographical deficit model is a cognitive related account. Both describe the same anatomical network of connected frontal and limbic structures. The dysregulation and decreased connectivity in the fronto-limbic network is, therefore, an appropriate target for intervention by means of neurofeedback techniques. The alpha-theta protocol is discussed as the first protocol tested specifically on PTSD population and gained a good support for its effectiveness. This efficacy on reducing PTSD symptoms is explained as the result of an integration of fronto-limbic network and the release of repressed memories in a secure environment. Nevertheless, we suggested LORETA neurofeedback as a more specific protocol for PTSD intervention. This protocol is based on findings supporting both vmPFC and theta activity dysregulation among patients suffering from PTSD compared to normal samples. Our discussed results suggest both a specific and unspecific effect of this treatment on theta and beta activity together with an improvement in CAPS scores as described in the presented case report.

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