# Neurotherapy of Traumatic Brain Injury/Posttraumatic Stress Symptoms in OEF/OIF Veterans

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*The Flexyx Neurotherapy System (FNS), a novel variant of EEG biofeedback, was adapted for intervention with seven treatment-refractory Afghanistan/Iraq war veterans, and brought about significant decreases in bothersome neurobehavioral and posttraumatic stress symptoms. FNS may help ameliorate mixed trauma spectrum syndromes.*

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An emerging legacy of the Afghanistan and Iraq Wars is a new generation of veterans with traumatic brain injury (TBI). Indeed, TBI, particularly due to blasts from improvised explosive devices, has been called the "signature injury" of those deployed to Operation Enduring Freedom (OEF) and Operation Iraqi Freedom (OIF).[1] Prevalence estimates range as high as 22% of wounded soldiers with TBI in clinical reports.[2,3] Although the more severely injured receive immediate care on the battlefield and are transferred to military hospitals, many return to duty after a brief period of light duty.[4,5] Many significant mild head injuries (concussions) are likely to be overlooked during continued duty in conflict. Despite the trend for improvement over time that is frequently noted with mild TBI (mTBI), it is also apparent that, after discharge, many veterans continue to report significant postconcussive symptoms.[4]

The actual mechanism(s) of brain injury in these cases is uncertain, but diffuse axonal shearing, contusion, and subdural hemorrhage, similar in nature to other modes of closed head injury, may be reasons.[6] Disruption of normal patterns of electrical power of the brain as well as disruptions of intra- and interhemispheric communication may be present and persist from the acute to the chronic stage in many patients.[6]

Also, there is high comorbidity of TBI and emotional disorders, particularly Posttraumatic Stress Disorder (PTSD). Even when PTSD is not formally diagnosable, a number of PTSD features may be present. Matters are further confounded by the overlap in symptoms characteristic of both TBI and PTSD, including concentration and recall problems, sleep disturbances, mood/emotional irregularities, and other difficulties. PTSD itself is associated with a wide range of psychiatric comorbidity, poor quality of life, and social dysfunction. Disentangling the effects of TBI and PTSD or less fully pronounced posttraumatic stress features can be extremely challenging. Also, both distinct and shared mechanisms may interact in producing features associated with TBI and PTSD.[1,2,4,7–9]

In any case, the nature of these injuries poses significant rehabilitation challenges.[4,8] There is a critical need to develop more effective treatments. Recent developments in the bio-energy domain of complementary and alternative medicine have suggested the potential for electroencephalograph (EEG) biofeedback. EEG biofeedback is typically performed within an operant-conditioning framework, wherein subjects acquire the skills to change patterns of EEG activity, which involves considerable time and effort.[10]

Alternative procedures have also developed. One involves offsetting stimulation of brainwave activity by means of an external energy source. The Flexyx Neurotherapy System (FNS) involves conduction of electromagnetic energy (EM) stimulation via the connecting EEG cables.[11] FNS has been further adapted by utilization of two-channel, rather than one-channel only, neurofeedback. This article reports on findings obtained in FNS treatment of a series of highly-symptomatic OEF/OIF veterans.

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## METHODS

Seven OEF/OIF veterans with persistently bothersome symptoms of TBI and PTSD were referred for FNS treatment. All seven participants were not progressing on standard medication or comprehensive rehabilitation treatments. In order to be candidates for FNS treatment, they needed to be free of seizure disorder, sleep apnea, or ongoing substance abuse. Participants completed pre- and post-treatment questionnaires, including the Neurobehavioral Functioning Inventory (NFI)[12] and the PTSD Symptom Scale (PSS).[13] The NFI measures the frequency of neurobehavioral dysfunction, with separate scale scores for Depression, Somatic, Memory/Attention, Communication, Aggression, and Motor problems. The PSS contains 17 items designed to diagnose PTSD according to DSM criteria. The items are also grouped according to three PTSD symptom clusters of re-experiencing, avoidance, and increased arousal. A total severity and three subscale scores can be obtained. Also, at the beginning of each treatment session, participants rated their current levels of symptoms on separate 0–10 scales with appropriate anchors, including cognitive clarity (e.g., 0: fully clear; 10: worst imaginable cognitive problems), overall body pain, quality of sleep, fatigue, anxiety, depression, irritability/anger, and overall activity.

FNS equipment consists of a laptop computer and J&J Enterprises (Poulsbo, WA) I-300 Compact 2 (C-2) Channel EEG module with on-board feedback-generating power; it uses proprietary software to link the digital brainwave recording device (C-2 module) through the computer, which then sets the parameters for the C-2 module to emit pulsed EM stimulation.[11] The system returns a signal to the participant via conduction from the C-2 module, varying as a function of the detectable peak EEG frequency (but offset from it), thereby permitting strategic distortion of the EEG. The amount of EM stimulation was standardized, with the feedback frequency being offset from the dominant EEG frequency at +20 Hz. Pulses of EM energy operated at a duty cycle of 1%; that is, of the maximum permissible on-time for each pulse, they were powered no more than 1% of the time (e.g., the maximum on-time at 1% for 1-Hz pulse was 0.01 sec). Testing revealed a power level of 100 pico watts through the sensor cable (Weber Innovations, Ann Arbor, MI).

Participants attended approximately 2–3 sessions per week. They sat comfortably with eyes closed and engaged in no specific activity. Electrodes were placed in a predetermined order over all areas of the cortex over the course of the sessions (range: 22–25). Each session included a total of 4 sec. of EM stimulation, spaced over 4 min. The stimulation was not immediately discernible, and adverse reactions (e.g., transient increases in typical symptoms after the first few sessions) were minimal. Participants were not asked to discuss past traumas as part of the process.

Data analyses included paired-sample *t*-tests for NFI and PSS scores. Curve-estimation linear-trend regression models were run for each of the current symptom ratings made over the course of 22 sessions. Given the exploratory nature of this study and desire to identify any significant patterns, no correction for multiple comparisons was made. However, effect sizes in terms of percentage of variance explained ($r^2$) were calculated for each of the primary pre- to post-treatment comparisons.

## RESULTS

Five of the seven individuals fully completed the treatment protocol (22–25 sessions) and provided sufficient pre- and post-treatment outcome information. An additional two individuals discontinued treatment after experiencing substantial symptom-reduction to minimal levels in a shorter period of time (13 and 17 sessions). Descriptive information on these five participants: two women, three men; age range: 23–42; highest education level: two who completed high school, two with some college, and one with a B.S. degree; four served in the Army, one in the Marines; number of deployments ranged from 1 to 4; all with a history of at least mTBI, three with multiple episodes of loss of consciousness lasting up to 45 minutes; four involved in blast/explosive injuries (maximum of nine episodes in one case), and motor vehicle accidents; a variety of assorted physical injuries; all with PTSD; and three with history of psychiatric hospitalization for suicidal ideation.

Pre- to post-treatment comparisons for NFI and PSS scores are presented in Table 1. Results reveal significant decreases on four NFI dimensions and strong trends on the other two, and significant decreases in PSS Total scores as well as the re-experiencing and avoidance symptoms clusters and a strong trend in evidence for decrease in arousal symptoms. All pre- to post-treatment comparison effect sizes were large. Also, linear trends were all highly significant in terms of improvement for each of the current symptom ratings made at the beginning of each of the individual treatment sessions (for the five participants completing at least 22 sessions), including cognitive

**TABLE 1. Pre- to Post-Treatment Comparisons, mean (standard deviation)**

| | Pre-Treatment | Post-Treatment | t [df], p | Effect Size |
|---|---|---|---|---|
| **Neurobehavioral Functioning Inventory (NFI)** | | | | |
| Depression | 52.8 (9.0) | 39.2 (5.9) | 3.5 [4], 0.02 | 0.76 |
| Somatic | 54.4 (6.3) | 41.0 (4.1) | 8.9 [4], 0.001 | 0.95 |
| Memory/Attention | 59.2 (13.0) | 41.4 (7.7) | 4.6 [4], 0.01 | 0.84 |
| Communication | 58.2 (14.7) | 42.8 (9.9) | 3.5 [4], 0.02 | 0.76 |
| Aggression | 49.6 (11.5) | 41.2 (3.0) | 1.8 [4], 0.14 | 0.45 |
| Motor | 56.4 (12.5) | 40.8 (6.9) | 2.6 [4], 0.06 | 0.63 |
| **PTSD Symptom Scale (PSS)** | | | | |
| Re-experiencing | 7.0 (4.7) | 2.2 (2.5) | 3.0 [4], 0.04 | 0.69 |
| Avoidance | 14.2 (6.4) | 3.4 (4.0) | 3.7 [4], 0.02 | 0.93 |
| Arousal | 9.4 (3.8) | 6.2 (4.0) | 2.6 [4], 0.06 | 0.62 |
| Total | 30.6 (13.0) | 11.8 (9.5) | 3.8 [4], 0.02 | 0.79 |

| **Medication Status, Participant #** | **Pre-Treatment Medications** | **Post-Treatment Medications** |
|---|---|---|
| 1[a] | acetaminophen/oxycodone, lithium, promethazine, ropinirole, sertraline, tramadol, zolpidem | lithium, sertraline |
| 2[b] | none | none |
| 3[a] | valproic acid | none |
| 4[a] | hydroxyzine, nortriptyline | none |
| 5[c] | citalopram, gabapentin, venlafaxine | citalopram |
| 6[a] | aripiprazole, clonazepam, sertraline, trazodone, zolpidem | sertraline, zolpidem |
| 7[a] | none | none |

[a]Completed full course of (22–25) sessions; pre- and post-treatment measures available for comparison.
[b]Discontinued treatment after 13 sessions after experiencing substantial symptom reduction to minimal levels; pre- but not post-treatment measures available for data analyses.
[c]Discontinued treatment after 17 sessions after experiencing substantial symptom-reduction to minimal levels; pre- but not post-treatment measures available for data analyses.

clouding ($\beta = -0.23$; $R^2$=0.22; F[1, 108]=31.06; $p<0.001$), pain ($\beta = -0.15$; $R^2$=0.12; F[1, 108]=14.76; $p<0.001$); sleep quality ($\beta = -0.29$; $R^2$=0.49; F[1, 108]=103.69; $p<0.001$); fatigue ($\beta = -0.23$; $R^2$=0.27; F[1, 108]=39.24; $p<0.001$); anxiety ($\beta = -0.20$; $R^2$=0.29; F[1, 108]=43.99; $p<0.001$); depression ($\beta = -0.15$; $R^2$=0.25; F[1, 108]=35.60; $p<0.001$); and irritability/anger ($\beta = -0.11$; $R^2$=0.12, F[1, 108]=15.25; $p<0.001$); and for overall activity ($\beta = -0.20$; $R^2$=0.33; F[1, 108]=52.61; $p<0.001$). All participants, if taking medication at the outset, substantially decreased their medication intake by posttreatment.

## DISCUSSION

Findings from this small series of highly-symptomatic OEF/OIF veterans with mixed TBI/PTSD syndromes suggest that FNS treatment, a novel variant of EEG biofeedback, may be worth exploring more rigorously. This is encouraging because it is increasingly apparent that some of the most treatment-refractory cases are among those individuals who, in addition to sustaining TBI, manifest strong PTSD symptoms.[2,8,9] FNS is one variant of minute-pulsed EM stimulation neurofeedback procedures, having evolved from a precursor photic stimulation paradigm known as EEG-Driven Stimulation[14] and having been followed by another related low-intensity technology, the Low Energy Neurofeedback System.[15] However, our experience with TBI/PTSD is only with FNS and the two-channel modification described herein. Future research is warranted to corroborate these preliminary results in a randomized, controlled trial and to explore the mechanisms that may underlie improvement, as we can only speculate at this stage that FNS may exert a positive effect on intra- and/or interhemispheric connectivity.

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