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EEG Neurofeedback as Adjunct to Psychotherapy for Complex Developmental Trauma-Related Disorders: Case Study and Treatment Rationale

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The present clinical case study describes the long term treatment of "Bea", a survivor of repeated and complex developmental trauma, via trauma-focused psychotherapy combined with electroencephalography (EEG) neurofeedback. Bea's case is described alongside a brief introduction to a rationale for including EEG neurofeedback as an intervention for complex developmental trauma-related disorders. Future research directions are discussed.

Keywords: neurofeedback, electroencephalography (EEG), complex posttraumatic stress disorder (PTSD), dissociation

Bea's husband was overwhelmed and fearful for his wife. Bea had been suicidal, self-harming and essentially unable to function at work or at home since the birth of their second child nearly 20 years earlier. She suffered daily flashbacks, often in public places, a reality that served to further humiliate this already deeply shame-stricken woman. Bea experienced dissociative states in the form of trauma-related altered states of consciousness (TRASC; Frewen & Lanius, 2015) daily in response to stressors that threatened to overwhelm her. In early sessions, usually in response to her own narrative, she could be seen to "disappear from behind her eyes;" it seemed clear that she could no longer hear her therapists' speech. She cried often for hours each day and, in her own words, "Anxiety, is like breathing." She slept during the day, fearing to sleep at night due to posttraumatic nightmares. The level of distress and TRASC to which Bea fell to experiencing at any reference to her history was so overwhelming as to require her therapist to conduct her initial clinical assessment with her husband rather than directly with Bea.

Later confirmed by Bea herself, her husband described Bea as the second child of four children, born to a mother with a history of severe psychological trauma including family disruption, being lashed with a bull whip, and being sexually touched on at least one occasion by her father, a military veteran with longstanding alcoholism and probable posttraumatic stress disorder (PTSD). Bea's paternal uncle was also a certified pedophile, a fact suggesting the possibility that there was sexual abuse in the grandparent generation. Bea's mother witnessed her father's sexual molestation of Bea when she walked into the bedroom, screamed at him, and it stopped. However, Bea's mother subsequently either no longer remembered the event or refused to acknowledge it. In addition to this incident, Bea was orally raped repeatedly by her maternal step-grandfather and a neighborhood boy. Bea's siblings believed the incidents concerning her grandfather—another sibling had experienced this as well—but refused to accept the incident with her father. As a result, several years into beginning a previous trial of psychotherapy, Bea ended all contact with her family. As an adolescent, Bea repeatedly witnessed violent interactions between her parents who finally divorced. After her father left, he failed to maintain contact with his children. Bea described her mother as quick to shame her and neither supportive or warm. In addition, she described her brother as filled with rage, an alcoholic from a young age, and in chronic problems with the law.

Bea's presenting problems were PTSD, depression, dissociation, self-abusive behavior, and an inability to work. Upon initial assessment she was taking an antipsychotic, a mood stabilizer (anticonvulsant), a selective serotonin reuptake inhibitor, a selective serotonin and norepinephrine reuptake inhibitor, and a benzodiazepine, as well as medications for irritable bowel syndrome and high blood pressure; over the course of her illness she had tried many other medications. Furthermore, her husband believed that psychotherapy had only made her worse. She had received psychodynamic therapy for 5 years and for the 14 years before this she had been part of a dialectical behavior therapy (DBT; Linehan, 1993) program involving both individual and group therapy focused on developing her emotion regulation skills. Bea's husband was very concerned that the present treatment would require talking about her traumas because, in his words, "whenever she does, she falls apart." In general, he reported that he had seen little change in Bea after 19 years of psychotherapy, except for her to become worse. Bea, however, credited DBT with teaching her strategies that had kept her out of hospital for the 6 years prior to beginning the present treatment (Bea was hospitalized three times in the past for near lethal overdoses of her psychotropic medica-

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tions and self-injury including biting herself, cutting, and gouging her eyes, as well as long periods of mutism). To date, psychotherapy and medications, however, appeared to have accomplished little more than this: they had kept her alive, but had not helped her to feel that it was worthwhile to live. Bea stated, "I come to therapy to stay alive for my children. No other reason."

It seemed clear that further psychotherapy alone would likely be insufficient to fully treat the trauma-related problems with which Bea had long suffered. Clinician SF thus decided that it was necessary to target affect-regulation directly by training the nervous system. At the onset of their work together in 2008, SF introduced Bea to what was then and currently remains a novel intervention: electroencephalography (EEG) neurofeedback. Today, following long-term treatment combining EEG neurofeedback (NFB) and psychotherapy, Bea is free of all symptoms of PTSD and psychiatrically discharged without medications. She could not have anticipated such an outcome or the effect it would have on her sense of self. It is that journey that this article will address.

EEG NFB: Treatment Rationale for Trauma-Related Disorders

Different EEG frequency bands are widely known to be associated with different cognitive-affective and basic physiological functions. In general, alpha (8–11 Hz) oscillations, historically associated with general states of relaxed alertness, are further known to play a role in internal attention. By contrast, delta (0–3 Hz) waves are seen during normal sleep but are also common in the waking brains of those who have suffered traumatic brain injury, as are higher amplitude theta frequencies (4–7 Hz), the latter often observed during the transition between waking and sleeping as well as during deep, hypnogogic states. Finally, beta (12–36 Hz) and gamma (>36 Hz) waves are often increased during focused, goal-oriented cognitive activity.

Studies of persons with trauma-related disorders reveal functional abnormalities in frontal, temporal, and parietal cortices linked to different EEG frequency bands. However, findings across studies are heterogeneous to date, possibly reflecting different phenotypes, for example, differing between high-arousal versus low-arousal (or dissociative) subtypes (e.g., Lanius et al., 2010). For example, elevated high beta (25–36 Hz; Begić, Hotujac, & Jokić-Begić, 2001; Cohen et al., 2013), low alpha (8–11 Hz), and high peak alpha frequency (Wahbeh & Oken, 2013) may be markers of chronic hyperarousal and hypervigilance, whereas theta oscillations may be associated with dissociative states (Giesbrecht, Jongen, Smulders, & Merckelbach, 2006; Krüger, Bartel, & Fletcher, 2013). In fact, in a large nonclinical population, McFarlane and colleagues (2005) showed that participants exposed to early life stress evidenced reduced power across all EEG bands, as well as increased peak alpha frequency. More recent literature is turning to the investigation of functional connectivity (Kim et al., 2012; Lee et al., 2014) and signal complexity (Bob & Svetlak, 2011; Chae et al., 2004; Hopper et al., 2002) as biomarkers of the long-term effects of complex developmental trauma histories.

An important and novel question is whether cutting-edge advances in neurotechnology, specifically brain-computer interfaces such as EEG NFB that analyze EEG data in real time, can be effectively harnessed to guide the brain toward regulation of EEG abnormalities in traumatized persons (Fisher, 2014). NFB or brain wave training is computerized biofeedback to the frequency domain of brain functioning and has a long history (e.g., Hardt & Kamiya, 1978; Kamiya, 1968, 2011; Sterman, 2000; Wyrwicka & Sterman, 1968). As described in numerous nontechnical sources (e.g., Demos, 2005), NFB involves presenting to participants a "picture" of targeted EEG amplitudes or coherence in real time, typically on a computer screen via visual feedback, as well as through auditory feedback (e.g., consistency or volume of a tone). With such feedback participants learn to directly increase or decrease EEG amplitudes of their own volition, including within the context of ongoing mindfulness meditation practice. In fact, whereas research participants are rarely provided specific guidance regarding how to self-regulate their EEG, depending on the targets of training, certain strategies are likely to be more beneficial than others; for example, one study found that engaging in positive imagery was often an effective strategy in up-regulating alpha amplitudes particularly in the left relative to the right hemisphere (Nan et al., 2012).

Referring to the history of NFB as a psychophysiological intervention, it was discovered in the late 1960s that cats could learn to control induced seizures through operant conditioning of their brain waves (Wyrwicka & Sterman, 1968): They were rewarded with food every time they made the stabilizing frequency of 12–15 Hz, now called the *sensory motor rhythm* or SMR. NASA funded this study because astronauts orbiting the earth were having seizures after inhaling the fumes from rocket fuel and their exposure could not be prevented; since they could not be given drugs, the astronauts had to learn to prevent errant brain wave activity and indeed this proved feasible through NFB. In fact, after successful work with cats and then with monkeys, Sterman (2000) was able to take people with intractable seizures off the psychosurgery waitlist with NFB training; not one of his patients returned for psychosurgery. Using green and red lights as feedback, these patients, like the cats and monkeys before them, were able to learn to increase the amplitude of 12–15 Hz EEG oscillations and, in the process, to control their seizures.

There are many different models for understanding the treatment effects of NFB, none of which are sufficient in themselves to fully explain this highly complex phenomenon. For example, Gruzelier (2009) discussed the role of alpha-theta NFB in regulating long distance alpha-theta oscillations and impacting creative performance and working memory. NFB of alpha oscillations has also been shown to improve cognitive performance for functions negatively affected in traumatized persons including for short-term and working memory (Escolano, Aguilar, & Minguez, 2011; Nan et al., 2012), mental rotation (visuospatial ability; Hanslmayr, Sauseng, Doppelmayr, Schabus, & Klimesch, 2005; Zoefel, Huster, & Herrmann, 2011), processing speed and executive function (Angelakis et al., 2007). One of the most useful models for clinical application, called the *arousal/regulation model*, was developed by Susan and Siegfried Othmer (2008) and emphasizes NFB as an intervention for regulating hyperarousal, hypoarousal, and so-called "instability" of the central and autonomic nervous systems. As but one example of a NFB protocol for lowering central arousal, EEG-alpha enhancement NFB is an intervention originally developed on the basis of research showing that longterm mindfulness meditation practitioners frequently exhibited high EEG-alpha amplitude relative to the general population, thus suggesting

its role in relaxation and wellbeing (e.g., review by Cahn & Polich, 2006). However, whereas mindfulness meditation practice modulates EEG-alpha activity only indirectly, through attentional processes, EEG-alpha NFB aims to do so directly.

Despite the fact that the efficacy of alpha NFB for reducing anxiety has been established for decades (Hardt & Kamiya, 1978), and effects for improving cognitive function are also increasingly well documented (e.g., Hanslmayr, Sauseng, Doppelmayr, Schabus, & Klimesch, 2005; Zoefel et al., 2011), surprisingly little research has evaluated NFB as a treatment for PTSD. A promising early line of research by Peniston and Kulkosky demonstrated in both prepost and randomized trials that eyes-closed alpha-theta NFB reduced PTSD symptoms relative to treatment as usual in male inpatients with combat-related PTSD with or without comorbid alcoholism (Peniston & Kulkosky, 1989; Peniston, Marrinan, Deming, & Kulkosky, 1993). These findings were replicated in the Cri-Help Study with alpha-theta brain wave training of 121 volunteers with poly drug abuse in a rehabilitation facility; at 12 month follow-up, 71% of those treated by NFB were abstinent as compared with 44% of the controls (Scott, Kaiser, Othmer, & Sideroff, 2005).

More recently, researchers demonstrated that a single session of EEG alpha-NFB also improved subjective emotional state and modulated functional MRI (fMRI) resting-state connectivity within the fMRI Default Mode Network (DMN) and Salience Network (SN) in 21 individuals (18 women) with PTSD related to childhood-interpersonal trauma. Here, alpha desynchronization NFB was associated with a significant increase in resting-state alpha amplitudes post-NFB that was correlated with increased calmness, greater SN connectivity with the right insula, and enhanced DMN connectivity with bilateral posterior cingulate, right middle frontal gyrus, and left medial prefrontal cortex. These findings may be important insofar as they implicate neural networks shown to be dysfunctional in persons with PTSD in prior research both during a resting state (DMN [Bluhm et al., 2009; Lanius et al., 2010; Qin et al., 2012; Sripada, King, Welsh, et al., 2012b; Zhou et al., 2012] and SN [Rabinak et al., 2011; Sripada, King, Garfinkel, et al., 2012a; Sripada, King, Welsh, et al., 2012b]) and in response to symptom provocation paradigms including exposure to reminders of traumatic memories (Etkin, & Wager, 2007; Hayes, Hayes, & Mikedis, 2012; Patel, Spreng, Shin, & Girard, 2012; Sartory et al., 2013). Although promising, these results are based on only a single session of NFB alone, and the long-term objective neurophysiological (EEG and fMRI) and neurocognitive outcomes of alpha-NFB for PTSD remain to be evaluated.

Beyond alpha-NFB training, what is generally known about the brain regions underlying the symptomatology of complex developmental trauma-related disorders can be used to guide the development of NFB-treatment protocols for these disorders; for example, treatments targeting the function of the medial temporal lobes may partly address aberrant amygdala function, memory problems, and dysregulation of primary process affective consciousness frequently observed within persons with PTSD. Indeed Gapen et al. (2016) found preliminary support for protocols rewarding 12–15 Hz (sensory motor rhythm) interhemispherically at temporal sites (T3-T4) and particularly at right temporoparietal cortex (T4-P4), with accompanying theta (4–7 Hz) and upper beta (22–36 Hz) inhibition. Such findings were observed in 23 individuals with PTSD, 17 of whom completed 40 sessions of treatment, and evidenced medium-to-large effect size decreases in self-reported PTSD symptoms ($d' = .69$, 34% variance) and affect dysregulation ($d' = 1.01$, 25% variance), with the change in PTSD symptoms found to partially mediate the change in affect dysregulation.

However, acknowledging the heterogeneity of EEG findings in group studies of traumatized persons, we would argue that an individualized approach to NFB treatment is likely to be most successful, specifically, as guided by patient self-reports and quantitative EEG data acquired in response to initial treatment. Although requiring further validation through the conduct of systematic studies, clinical experience suggests that there are at least two major EEG abnormalities exhibited by persons with complex developmental trauma-related disorders: (a) excess slow wave activity of the delta (0–3 Hz) and/or theta (4–7 Hz) bands, and (b) excess fast wave or high beta (22–36 Hz) activity; in fact, certain persons reveal both abnormalities. As such, clinical experience suggests the efficacy of protocols rewarding frequencies between 10.5–13.5 Hz, or 9–12 Hz, and inhibiting excess slow wave (0–6 Hz) and fast wave (high beta, 22–36 Hz) frequencies, with the primary intention of lowering an overaroused nervous system (Fisher, 2014). Regarding electrode placement, Table 1, based on Fisher (2014), describes several treatment protocols that may be relevant to the treatment of persons with complex developmental trauma-related disorders; the clinical and neurophysiological effects of such interventions, varying by location and target frequency, require evaluation in randomized controlled trials.

It is important to acknowledge that a specific EEG biomarker for trauma and stressor-related disorders has yet to be discovered and, given the complexity of the human brain and the heterogeneity of response to psychological trauma, is perhaps unlikely to ever be. In other words, we take the position that every individual's brain wave patterns are unique and, while quantitative brain maps (qEEGs) are often obtained as a means of identifying an individual's pattern of dysfunction compared to a database of normally functioning brains, such assessments do not always yield information that is clinically useful, which was unfortunately true in Bea's case. As such, and not dissimilar with psychotherapy in this respect, the practice of NFB often takes the form of a dance between hypothesis generation and evaluation, the latter taking into account both subjective and objective EEG data in response to treatments provided.

EEG NFB Therapy: The Case of Bea

Bea did not have a documented history of seizures but something akin to that kind of violent storm seemed likely to be occurring in her brain. As is often the case for patients like Bea, her long list of medications included mood stabilizers which, it will serve as a reminder, are referred to as anticonvulsants when prescribed by a neurologist. Such medication regimes, at least implicitly, suggest that severely traumatized persons like Bea are being understood by psychiatrists to suffer from something akin to seizure activity.

Perhaps consistent with this conceptualization, during the first 10 months of the clinical intervention, Bea would frequently explode or implode during therapy sessions as evidenced by self-harming and reckless behavior. She frequently asked for ice to calm or ground herself, an intervention that she had learned from

Table 1
Electrode Placements of Potential Efficacy in Neurofeedback Treatment of Persons With Complex Developmental Trauma-Related Disorders

Placement	Hemisphere	Rationale and treatment targets
C4 or C4-P4	RH	General assessment and training of frequencies associated with emotional and psychosomatic function
T4-P4	RH	Subcortical limbic "fear" circuitry directly (e.g., right amygdala)
T4-F8	RH	Emotional and embodied self-awareness (e.g., mediated by right insula)
T6-P4	RH	Embodied self-awareness (e.g., mediated at right temporoparietal junction); may also be relevant to reading of facial expression
FpO2	RH	Prefrontal cortical inhibition of limbic "fear" circuitry (e.g., over right amygdala)
FpO1	LH	Reward processing (e.g., orbitofrontal cortex, striatum)
P5	LH	Intrusive verbal mentation / voice hearing (e.g., mediated by auditory association cortex; Wernicke's area)
Fz	Midline	Conceptual and embodied self-awareness mediated by dorsomedial and ventromedial prefrontal cortex; Prefrontal/ Cingulate inhibition of limbic "fear" circuitry (e.g., amygdala)
Pz	Midline	Conceptual and embodied self-awareness and episodic memory (e.g., mediated by posterior cingulate/precuneus)
T3-T4	Interhemispheric	Bilateral central "instability" possibly underlying emotion dysregulation (e.g., panic attacks) and migraine headaches

Note. Electrode placements typically refer to those of the Standard International 10–20 system excepting FpO1 and FpO2 (above left and right eyelids). First electrode site indicates training site and second reference site; single electrode placements use same side earlobe as reference or right in case of training at midline. It is important to note that all protocols are based largely only on clinical experience and therefore should be considered experimental; randomized controlled trials to evaluate efficacy and ensure nonrisk are needed. RH = right hemisphere; LH = left hemisphere.

DBT. Invariably she left therapy sessions sobbing, often driving directly to a convenience store to binge on what we colloquially refer to as "junk food." Bea also frequently sought comfort from her husband although it became increasingly evident from joint sessions that, although well-meaning and supportive, he rarely knew how to help her.

NFB sessions typically took the form of Bea sitting in a chair and playing a video game, for example, completing a picture divided in the form of a grid, a maze, or racing a spaceship, without use of her hands, but rather entirely with her brain. In other words, success in the game was calibrated to Bea's real-time EEG such that she would gain points when she succeeded in increasing the amplitude of the frequencies that should, in theory, help to calm her neurophysiological state, while decreasing the amplitude of frequencies presumably related to dysfunction (e.g., Demos, 2005), in Bea's case high amplitude slow wave (delta and theta) activity. The treatment was delivered within a independent practice setting, with sessions typically being about one hour and consisting of about 15–30 min NFB, with the remaining time spent in set-up and traditional talk therapy.

Her clinician (SF) followed the symptomatic picture of high and unstable arousal to train her brain, and many NFB protocols were employed during the long course of Bea's treatment. One of the advantages of NFB training is the requirement for ongoing assessment of progress and evaluation of protocols (International Society for Neurofeedback & Research, 2013); Bea or Bea's husband would report on her response to each training session. Generally speaking, the training focused on her right temporal and parietal lobes at frequencies that she reported were calming to her and, although assessed only informly, the goals were to diminish her level of ambient fear and reactivity, the intensity of her anxiety, anger, and shame, episodes of dissociation, and the number and the intensity of flashbacks that she experienced, as well as the amount of time it took for her to recover from flashbacks. Further, changes in objective measures such as sleep onset and maintenance, need for antianxiety medications, frequency and intensity of nightmares, and regularity in bowel function were also tracked informally as treatment indicators.

We would argue that the goal of NFB is for the brain to learn its own capacity for regulation in the frequency domain (Fisher, 2014). By contrast, as Bea's case and so many others suggest, medications fail to offer the brain the same opportunity for learning. When she arrived for treatment, one might conceptualize Bea's brain as practicing its own regulation errors repeatedly, with these errors manifesting as her symptoms. In comparison, NFB can be thought of as guiding her brain back to its unforgotten capacity for regulation and, as it did, her symptoms began to fade. Most importantly, as her brain learned to self-regulate, Bea began to gain self-regulation over fear, shame, and rage. Interestingly, as she quieted these emotional states, she experienced less dissociation and a more robust sense of herself began to emerge. It is possible that by quieting the repetitive firing of the circuitry of emotional processing, NFB can help weaken the experience of a traumatized identity; as arousal levels reorganize and quiet at the neuronal level, affect also organizes and quiets, and so do the narratives that these states give rise to and reinforce. Accordingly, clinical experience suggests that even the most fixed aspects of personality and personality disorder can begin to reorganize.

Within the first 10 sessions of NFB treatment Bea reported significant changes. She noticed that she did better on a business trip than she or her husband had expected. She found it easier "to socialize with strangers." She reported that it took her less time to recover after "emotional eruptions" than it had in the past, and that she even surprised herself by laughing. In psychotherapy, she began to talk about her history spontaneously, and over the course of several months, Bea stopped all self injurious behavior and dissociative episodes virtually ceased. She was, after only seven sessions, able to interrupt a posttraumatic flashback that began when a man inadvertently bumped into her, another telling sign of an apparent treatment effect. Her nightmares became less frequent and less disturbing to her, corresponding with greater ease in initiating sleep. She was able to stay at work longer and nap less during the day. In fact, by Session 10, she reported that she sometimes had a strange feeling of "no anxiety." These periods themselves provoked a new kind of anxiety that, by her description, was more "existential in nature." As much as she welcomed

many of these changes, the lack of familiarity of these experiences also often frightened her. For example, it was not uncommon for her to remark "I don't know who is saying this" during unusual moments of calm. Nevertheless, this was not, on clinical impression, an experience of dissociation, but rather a long-awaited indication of a newfound and emerging sense of self.

With regular coaching and encouragement, with ongoing NFB, and increasingly with traditional trauma-focused psychotherapy, it was apparent that Bea's level of affect dysregulation continued to lessen. Despite the occasional inevitable setback, with continuing treatment conducted in weekly sessions over the course of several months, it seemed evident that Bea's brain had slipped out of its prior habitual firing patterns, and had formed new and more adaptive ones. Today, she no longer qualifies for a psychiatric diagnosis, has been weaned off all medications, and was happy to report that she had discarded her last unfinished bottle of Lorazepam. Further, Bea completed a new course of study, left an unsatisfying place of employment, and began a new career in the helping professions. She reports finding herself at ease with her patients, both men and women. She is now able to receive routine medical and dental care, treatments she had avoided for 20 years prior. Moreover, she has developed a trauma-informed perspective regarding her mother's behavior toward her during childhood, and she has renewed her relationships with her siblings. Currently Bea only rarely talks about her trauma history except to be amazed that she is no longer overtaken by it. Most of all, Bea is no longer her overwhelming feelings. She is Bea.

Conclusion

Because of a history of treatment failure with medications and both cognitive–behavioral and psychodynamic therapies, Bea was considered by many previous mental health providers to be an untreatable patient. However, with the introduction of EEG NFB and the apparent improved regulation of her nervous system, her once crippling trauma-related symptoms slowly remitted. Her case suggests that a dysregulated nervous system may have been at the core of her symptom formation, and that resolution of these symptoms depended on her brain relearning how to regulate itself (Fisher, 2014).

Research since the late 1960s demonstrates that both animals and humans can learn NFB (Kamiya, 1968, 2011; Sterman, 2000; Wyrwicka & Sterman, 1968). To date, however, despite increasing acknowledgment of the effects of early trauma and neglect on the development and function of the human brain, little systematic research has examined the potential efficacy of NFB in the treatment of complex developmental trauma-related disorders in a methodologically strong, double-blinded randomized controlled trial. Cases like Bea's, however, suggest that such research is long overdue.

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