Appl Psychophysiol Biofeedback (2016) 41:251–261
DOI 10.1007/s10484-015-9326-5

A Pilot Study of Neurofeedback for Chronic PTSD

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Published online: 19 January 2016

Abstract EEG Biofeedback (also known as neurofeedback) has been in use as a clinical intervention for well over 30 years; however, it has made very little impact on clinical care. One reason for this has been the difficulty in designing research to measure clinical change in the real world. While substantial evidence exists for its efficacy in treating attention deficit/hyperactivity disorder, relatively little evidence exists for its utility in other disorders including posttraumatic stress disorder (PTSD). The current study represents a "proof-of-concept" pilot for the use of neurofeedback with multiply-traumatized individuals with treatment-resistant PTSD. Participants completed 40 sessions of neurofeedback training two times per week with sensors randomly assigned (by the study coordinator, who was not blind to condition) to sensor placements of either T4-P4 or T3-T4. We found that neurofeedback significantly reduced PTSD symptoms (Davidson Trauma Scale scores averaged 69.14 at baseline to 49.26 at termination), and preceded gains in affect regulation (Inventory of Altered Self-Capacities-Affect Dysregulation scores averaged 23.63 at baseline to 17.20 at termination). We discuss a roadmap for future research.

Keywords EEG biofeedback · Neurofeedback · Treatment outcome · Posttraumatic stress disorder · Complex trauma

Introduction

The empirical evidence for the efficacy of psychosocial treatments for PTSD is substantial (Bisson et al. 2007; Ehring et al. 2014); however, meta-analytic reviews show that less than half of patients receiving current psychosocial treatments demonstrate clinically meaningful improvements, and that the majority of patients continue to have substantial residual symptoms (Bradley et al. 2005; Jonas et al. 2013). Pharmacological treatments of PTSD (Baker et al. 1995; Davidson et al. 2001; Stein et al. 2006;

These data have been previously presented at the annual meeting of the International Society for Traumatic Stress Studies, Baltimore, November 2013.

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van der Kolk et al. 1994) have at best shown moderate effect sizes and there is insufficient evidence to estimate loss of diagnosis (for a review see Jonas et al. 2013). For these reasons the currently available scientific evidence for the treatment for PTSD does not reach the level of certainty that would be desired for such a common and serious condition. There is a clear need to explore alternative and adjunctive therapies to improve outcomes.

Cortical plasticity can be changed with computer brain interface (CBI) techniques. For example, lasting changes in cortical plasticity have been detected following transcranial magnetic stimulation (TMS) (Ros et al. 2010a, b). Neurofeedback (NF), a relatively user friendly method to operantly condition brain activity, has been shown to be able to induce a specific increase of functional connectivity within the alertness/salience network (dorsal anterior and mid cingulate), when compared with a sham control group (Ros et al. 2013).

Research utilizing NF has reported positive treatment outcomes or normalizing trends in numerous disorders including children with Autism (Jarusiewicz 2002; Kouijzer et al. 2009) and Attention Deficit Disorder (ADHD) (Monastra 2005; Monastra et al. 2006), and in studies designed to enhance cognitive and musical performance (Gruzelier et al. 2014). However, the evidence to date remains controversial. While one meta-analysis of 15 studies of ADHD found a large effect size for impulsivity and a medium effect size for hyperactivity symptoms in children (Arns et al. 2009), a more recent meta-analysis found general benefits; however, effects become non-significant when only "probably blinded" assessments were utilized (Sonuga-Barke et al. 2013). In sum, the research base generally shows promise for NF in the treatment of ADHD, but more research is needed on how and when to train an individual to achieve optimal benefits (Moriyama et al. 2012). Additionally, the theoretical framework to explain the effects of NF is only beginning to emerge, and will require the integration of multiple techniques to identify network/circuit level models. (Loo et al. 2016; Ros et al. 2014).

Neurofeedback Research in PTSD

Neurofeedback of slow wave (alpha and theta) frequency bands has been successfully employed in the treatment of PTSD (Peniston and Kulkosky 1991). After receiving NF only 3 of the 15 NF patients had a recurrence of PTSD symptoms over a 30 month long monthly follow-up assessment period compared to 100 % of the control group. In addition, the NF group showed significantly more improvement on 13 MMPI scales than the controls (Peniston and Kulkosky 1991). Given the promise of these results, it is surprising that there has been little to no research following up on these results. One potential reason for the lack of further studies has been the criticisms of the reporting of the results, which failed to mention a lack of independent samples and pre-training with skin temperature biofeedback (Graap and Freides 1998). The current study is intended to "re-ignite" treatment outcome research for NF with PTSD, and is a pilot project to examine the feasibility and effectiveness of NF in an outpatient population of individuals with chronic PTSD. This study represents phase 1 of our research program; the primary purpose of which is to demonstrate the feasibility of NF as an intervention technique. We recruited individuals who met criteria for PTSD, and who were receiving treatment-as-usual weekly individual talk therapy. We hypothesized that NF would significantly reduce PTSD symptoms as measured by the Davidson Trauma Scale. In addition, we examined the relationship of individuals' self-reported affect dysregulation symptoms to self-reported PTSD symptoms. This study served as a pilot study to provide the basis for a full-scale rigorously controlled trial of NF in a civilian population with chronic PTSD.

Our choice of treatment protocols for the current study was guided by both research evidence and clinical experience. Since Peniston and Kulkosky (1991) seminal work, brain imaging has provided significant evidence pointing to the right parietal areas of the brain as implicated in the maintenance of PTSD (Rauch et al. 1996; Lanius et al. 2002; Georgopoulos et al. 2010). For this reason NF training was done using a bipolar placement with T4 as the active site, P4 as the reference site, and the left ear (A1) as the ground. Based on clinical experience, we decided to compare this to a bipolar placement using T3 as the active site, T4 as the reference site, and the left ear (A1) as the ground. Finally, given the pilot nature of our research we determined that a waitlist control was inappropriate as we were interested primarily in the feasibility of deploying NF for this population.

Method

Participants

The sample included adults between the ages of 32–64 who were recruited from the greater Boston metropolitan area. Participants were recruited from the community using flyers and e-mails to local therapists. The sample was primarily white (n = 19) and female (n = 15). This was intended to be an ecologically valid effectiveness trial; thus, exclusion criteria were minimal and diagnosing was not rigorous. All inclusion and diagnostic information were collected by a doctoral level clinician during the phone screen. Participants

were eligible for inclusion if they were currently attending individual treatment (for at least 3 months prior to screening), met criteria for PTSD as determined by endorsing at least one cluster B, three cluster C, and two cluster D symptoms on the Davidson Trauma Scale, were medically stable, and were able to maintain medications throughout the course of the study. We sought to include as representative a sample as possible; therefore, exclusion criteria were minimal. Factors that led to exclusion included psychosis, active suicidal ideation, documented history of traumatic brain injury leading to functional impairments, treatment instability, significant unstable medical conditions, and history of seizure disorder. We did not control for modality or frequency of individual treatment or exclude any classes of medication or substance use/abuse. Additionally, we did not differentiate between acute or chronic trauma exposures. However, the entire sample had experienced multiple stressful life events as measured by the Stressful Life Events Screening Questionnaire. Of the 13 types of life stressors, the minimum number endorsed was 3 and the maximum was 9 with a mean of 6.59. Fifteen of the 17 who completed the study identified some form of childhood physical, sexual, and/or emotional abuse as their index trauma. Additionally, 100 % of the sample experienced some form of trauma prior to age 18 with a mean of 27.1 years since the index trauma exposure. Thus, the sample consisted of individuals with early-onset, chronic PTSD symptoms. Thirty-six individuals were screened for possible inclusion in the study. Of those, 11 did not meet criteria for PTSD, and two individuals declined to participate. This left a total of 23 individuals who received treatment, and 17 who completed the 40 sessions (73 % completion rate).

Measures

Demographics

We employed a standard demographics questionnaire that was developed internally. It asked for such information as age, ethnicity, employment, and living situation.

Stressful Life Events Screening Questionnaire [SLESQ, (Goodman et al. 1997; Goodman et al. 1998)]

This SLESQ is a 13-item self-report measure which consists of a detailed yet brief set of behaviorally-specific questions designed to elicit complete information about the type, recency and number of traumas. The measure includes eleven specific and two general categories of events including being in a life threatening accident, physical and sexual abuse, and witnessing another person being killed or assaulted.

Davidson Trauma Scale (DTS, Davidson et al. 1997)

The DTS is a 17-item self-report measure assessing the presence and severity of PTSD symptoms. Each item contains a frequency and severity score rated on a 0–4 scale. The total score ranges from 0 to 136. Individuals with PTSD following combat exposure or natural disasters obtained a mean score of 62 (SD = 38.0).

Inventory of Altered Self-Capacities (IASC, Briere 2002)

The IASC consists of 63 items rated on a five point Likert scale assessing disturbed functioning in relation to self and others. The IASC measures seven domains of functioning: Interpersonal Conflicts, Idealization-Disillusionment, Abandonment Concerns, Identity Impairment, Susceptibility to Influence, Affect Dysregulation, and Tension Reduction Activities. The Cronbach's alpha for the scale and subscales ranges from .78 (Tension Reduction Activities) to .96 (Identity Impairment) with an average Cronbach's alpha of .93. The Affect Dysregulation (AD) subscale is of particular interest in this study and assesses mood swings and problems in affect regulation and control. The AD subscale contains nine items; thus scores can range from 9 to 45. In the clinical standardization sample the mean was 21.45 and Cronbach's alpha was .95.

Changes Observed After Neurofeedback

This checklist was developed for this study and asks participants to rate over- and under-arousal symptoms on a five point Likert scale that ranges from 1 = "not at all" to 5 = "extremely." The measure contains 36 items, and the sole purpose was to guide decisions on whether to make changes to the reward band. Thus, the checklist was developed to capture the "typical" (guided by clinical experience) responses to NF training. Examples of items include: "I have been calm," "I have been anxious," and "I have had difficulty falling asleep."

Minimap EEG Assessment

The minimap is a form of EEG assessment adapted from clinical use for this study. Electrodes were placed sequentially at the following sites: O1, PZ, CZ, F3, F4, and FPZ. Data were collected in 2-minute increments under the following conditions: at O1 with eyes open, eyes closed, and eyes open; at PZ with eyes open, eyes closed, and eyes open; at CZ with eyes open, challenge (silent reading), eyes closed, and eyes open; and at F3, F4 and FPZ with eyes closed. This totaled thirteen discrete periods of data collection. The frequency bands measured were: 0–4, 4–8, 8–12, 8–9, 10–12, 12–18, and 22–36 Hz.

Procedure

The study employed a two-group, active treatment design intended to assess the effectiveness of two alternative placements of sensors on the scalp. Thus, participants were randomized to receive training either at T4-P4 or T3-T4 (sites defined by the International 10–20 system). The study coordinator used a random number generator to assign participants, and neither participants nor interventionists were blinded to condition. The training parameters were identical beyond placement of sensors and followed a flexible, principle-based manual that provided rules to adjust the training protocol based on the clinical response (defined as the number of over- or under-arousal symptoms) of each participant. We employed standard inhibit frequencies (4–7 and 22–36 Hz) and a beginning reward frequency (12–15 Hz) based on previous research (Jokic-begic and Begic 2003) and based on previous clinical experience. Training utilized the EEGer neurofeedback system manufactured by EEG Spectrum, Intl. Participants were trained either with a two-laptop configuration, or a single computer, dual monitor configuration.

No changes were made to the protocol except adjustments to the reward band frequency. Adjustments were made based on rated symptoms of over-arousal (including nightmares; sleep difficulties; hyperactivity; aggressive behavior, anger, anxiety; and self-reports of high arousal including self-harm, suicidal and/or homicidal ideation), and symptoms of under-arousal (including inattention, decreased alertness or mental clarity; nausea; depressive symptoms; and decreased energy/fatigue) captured by the Changes After Neurofeedback checklist as well as clinical judgment. If participants reported significant over-arousal symptoms for at least two training sessions, the reward frequency was lowered by 1 Hz. This procedure was continued until the participant reported no change, positive benefit, or symptoms of under-arousal. If the participant reported symptoms of under-arousal, the reward band was raised by ½ Hz until those symptoms remitted. Finally, if there was no change on any self-report measure, verbal report, or clinician assessment after 16 sessions of training, the participant was switched to the alternative sensor placement. (Only one participant was switched.) Finally, training time was maintained at 12 min until the participant reported positive change, after\which the training time was raised in 3-min increments until a maximum of 21 min was achieved.

Intervention technicians included masters and doctoral-level clinicians who were all trained to administer NF by EEG Spectrum, Intl. and met weekly to discuss ongoing treatment with a board-certified NF practitioner. Two NF systems produced by EEG Spectrum, Intl. utilizing the EEGer software were employed for the NF training. Sessions were reviewed by the study coordinator and supervising psychologist to assure fidelity to the protocol.

Participant eligibility was assessed in two steps: (1) telephone screen; and (2) in-person assessment. If individuals met all criteria, they were scheduled for an in-person assessment and completed the consent process, the SLEQ, DTS, IASC and minimap procedure. Participants were then randomly assigned to either of the protocols and a study clinician. See Fig. 1 for a flow chart of participant screening, randomization, and drop-outs. Efforts were made to maintain continuity of treatment with the ideal of two training sessions per week. This was not always possible, and one participant was dropped from treatment after missing four consecutive weeks of training. Participants completed the changes form during each meeting, and the DTS and IASC after every eight training sessions (giving a total of six assessment points total). After session 40, participants completed the same battery as baseline with the exception of the SLEQ. Participants were compensated $75 for their participation in the study.

Results

Data Analysis

Multilevel growth curve analyses were conducted to examine: (a) change over time in PTSD symptoms (DTS), (b) change overtime in AD (IASC-AD), and (c) the relationship between PTSD symptoms and AD across time (Singer and Willett 2003; Suvak et al. 2009). Growth curve analyses using multilevel regression procedures offer a number of strengths that facilitate sophisticated and powerful examinations of change over time. Strengths of this approach include: (a) capability of handling missing data and unbalanced designs (i.e., the number of assessment points and the timing of assessments can vary across participants), (b) highly efficient and powerful estimation procedures that include all data points available, and (c) modeling flexibility that provides multiple options for how to model time and allows for the inclusion of continuous or categorical, time invariant or time varying, predictors and covariates. This modeling flexibility enables the sophisticated examination of the relationship among multiple variables over time. The use of multilevel regression also allowed us to get the most of our relatively small sample size. The use of multilevel regression techniques was recommended as part of The Institute of Medicine's guidelines to maximize information from small clinical trials to obtain reliable and valid results (Institute of Medicine 2001).

Hierarchical Linear and Non-Linear Modeling software (HLM6; Raudenbush, Bryk and Congdon 2005) with restricted maximum likelihood estimation was used to

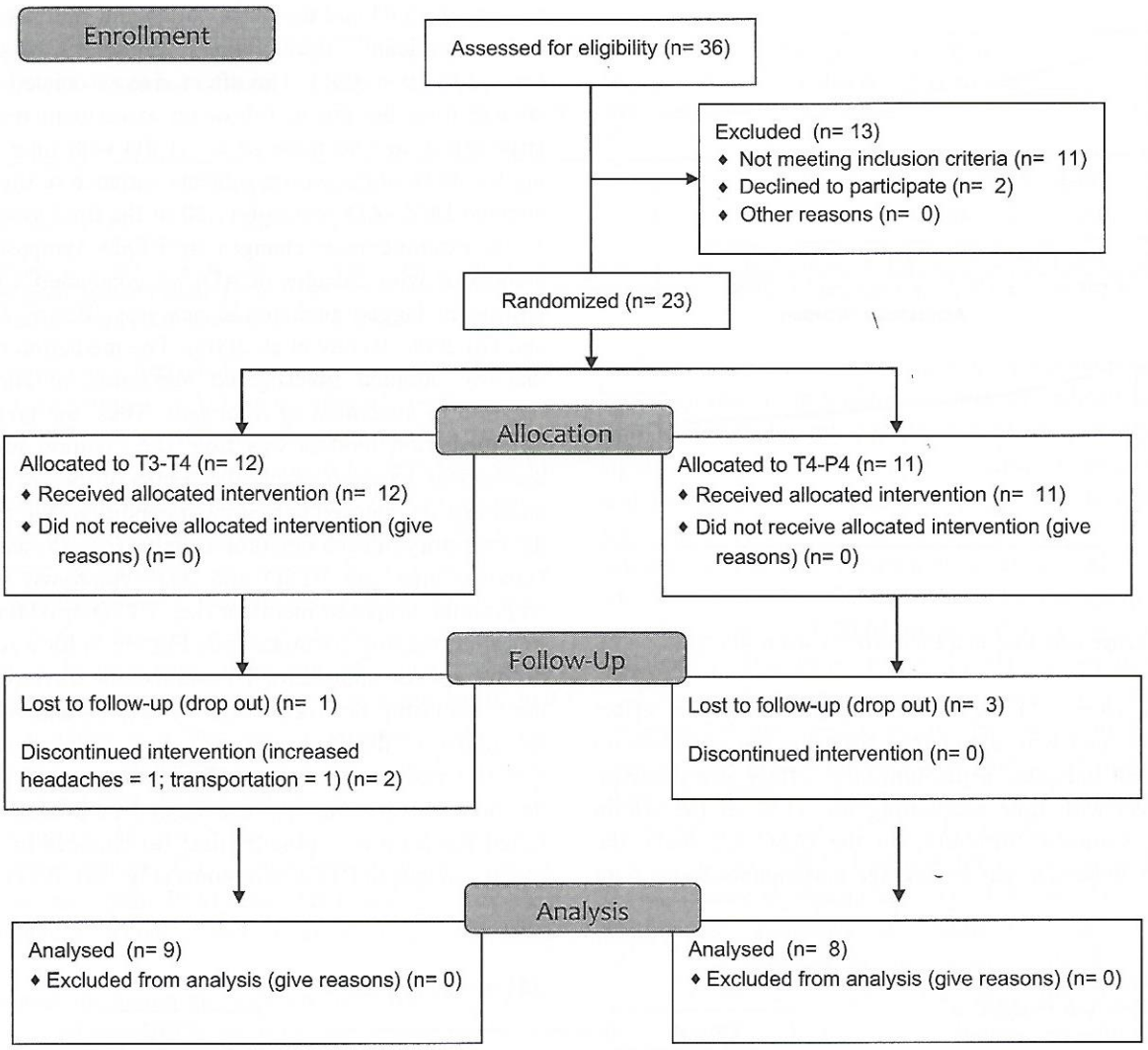

Fig. 1 Flowchart of participants

conduct primary analyses. To evaluate the significance of Level-1 associations, we evaluated the regression coefficients produced by HLM. Effect size (*d*) for change in the outcomes from the pre to follow-up assessment was computed by the procedures described by Feingold (2009), which produces effect size estimates from growth curve analyses that are comparable to those derived from more traditional repeated measures designs (e.g., repeated measures ANOVA) with .20, .50, and .80 representing small, medium, and large effects, respectively.

Results

The first set of analyses consisted of standard growth curve analysis to examine change in PTSD symptoms and AD during the course of the study. Our time variable began at zero (pretreatment assessment) and increased by one for subsequent assessments (with the last assessment coded as 5). We examined three models for each outcome: (a) a linear change model with the time variable coded as specified above, (b) a non-linear power-polynomial quadratic change model, which added a time squared variable to the linear model, and (c) a non-linear change model that modeled time using the natural-log of the linear time variable.[1] for indicated that the natural-log non-linear model (i.e., model c) fit the data best for each outcome. Figure 2 depicts change over time in each of the outcomes. Both outcomes exhibited a decelerating pattern of change with larger initial decreases that gradually diminished across the study. The regression intercept indicated that on average participants began with DTS score of 69.14 and the slope coefficient indicated a significant decelerating

[1] Because the natural-log of zero is undefined, the transformed variable was calculated by takin the natural log of our time variable plus one. The natural-log of 1 is zero; therefore, the intercept for the natural-log model represented outcome levels at the first assessment.

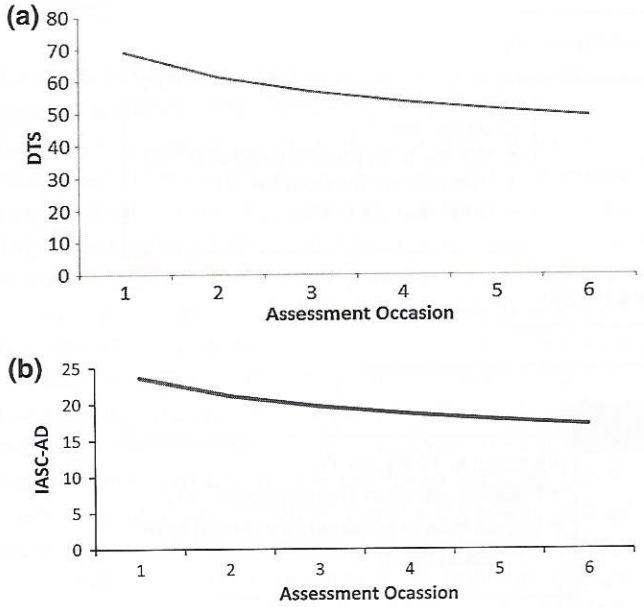

Fig. 2 Change over time in **a** PTSD (DTS) and **b** AD (IASC)

decrease ($b = -11.11$, $t = -3.83$, $p = .00$). The effect size associated with the change from the pre to follow-up assessment indicated a medium-large effect size decrease ($d = -.69$) with time accounting for 34 % of the within subjects variance. Similarly, for the IASC-AD scale, the intercept indicated that on average participants began with a score of 23.63 and the slope coefficient indicated a large and significant decelerating decrease ($b = -3.60$, $t = -5.66$, $p < .001$). The effect size associated with the change from the pre to follow-up assessment indicated a large effect size decrease ($d = -1.01$) with time accounting for 25 % of the within subjects variance resulting in an average IASC-AD score of 17.20 at the final assessment.

To examine how changes in PTSD symptoms were associated with changes in AD, we conducted a series of multilevel lagged-mediational analyses (Bauer, Preacher, and Gil 2006; Kenny et al. 2003). The mediation approach that we adopted investigated *mediation of change*, as opposed to *mediation of treatment*. Thus, the predictor in our mediation models was time (i.e., natural log of the assessment occasion, as per the results of the growth curve analyses), as opposed to treatment condition. We examined the trajectory of each outcome variable (i.e., the association between time and PTSD and AD symptoms) with and without the proposed mediator (i.e., PTSD or AD) included as a time-varying covariate (See Fig. 3a, b for a schematic of this analytic approach). To evaluate the directionality of the relationship between PTSD symptoms and AD across the course of the study the mediator variable was lagged (i.e., the mediator variable at Time $_{T-1}$ was used to predict the outcome at Time $_{T}$). The lagged mediation analyses tested the following possibilities: (a) changes in AD preceded changes in PTSD symptoms (Fig. 3a), (b) changes in

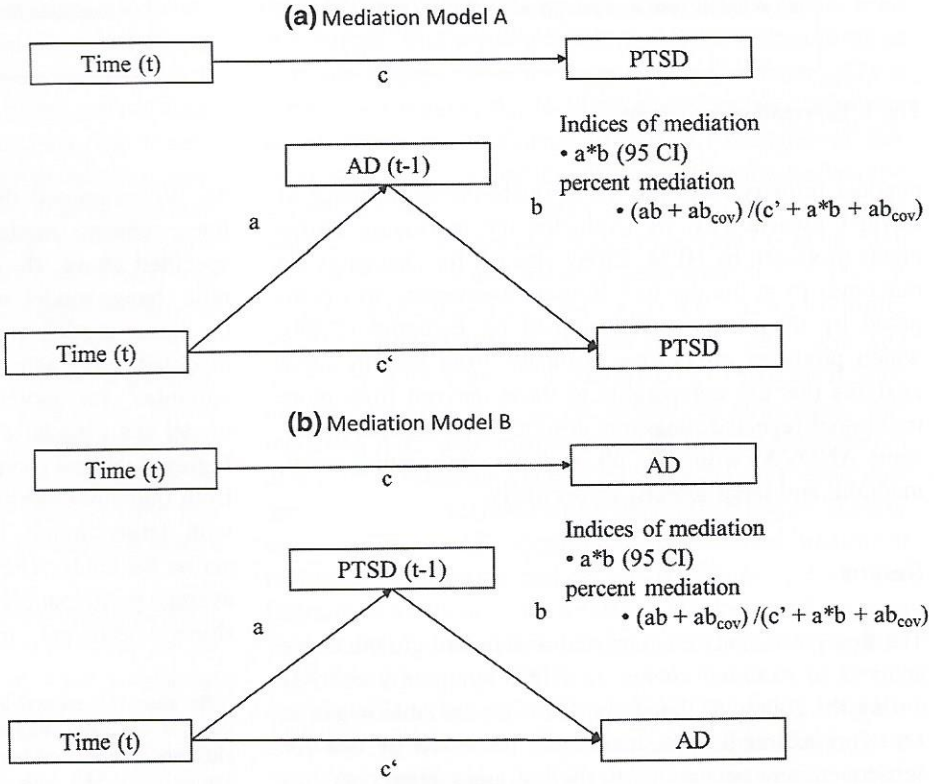

Fig. 3 Schematic of mediation models. Time was modeled as natural log of session number with "t" indicating the level of the variable at a particular assessment occasion and "t − 1" indicating the level of the mediator at the previous (i.e., lagged) assessment occasion

Table 1 Summary of mediation analyses

Model	M Mediator	Y Outcome	c Change in Y	a Change in M	b M predicting Y controlling for M	c' Change in Y controlling for M	Cor ab[x] correlation of a and b	ab (95 % CI) Indirect effect	Percent mediation
A	AD	PTSD	**−12.18**	**−5.23**	−0.05	**−12.69**	**0.55**	0.28 (−2.72, 4.90)	**−0.13**
B	PTSD	AD	**−5.23**	**−12.18**	**0.15**	**−3.51**	**0.82**	**−1.65** (−2.34, −0.48)	**0.24**

Bold indicates $p < .05$; c, a, b, and c' represent the paths of the mediation model as depicted in Fig. 2; ab = the indirect effect; Cor ab = correlation between paths a and b; 95 % CI = 95 percent confidence interval; PTSD = symptoms of PTSD as assessed by the DTS; AD = Affect Dysregulation assessed by the IASC-A

PTSD symptoms preceded changes in AD (Fig. 3b) (c) PTSD symptoms and AD are reciprocally related across time with changes in PTSD symptoms leading to later changes in AD, and vice versa (support for both Fig. 3a, b), or (d) the changes that occurred between the two variables occurred at the same time with no longitudinal influence of one variable on the other (no support for Fig. 3a, b). The following findings would indicate a significant longitudinal influence of the mediator on the outcome: (1) a substantial reduction in the regression coefficient indicating the degree to which the outcome changes over time when the lagged mediator is added as a time varying covariate (which can be evaluated by comparing paths c and c' of Fig. 3a, b), and (2) a significant indirect path from time (which is the predictor variable in growth curve analyses) to the outcome through the mediator. This approach is increasingly being applied to data from PTSD treatment studies to identify mechanisms of change (Aderka et al. 2011; Liverant et al. 2012).

Multilevel mediation analyses are very similar to traditional cross-sectional mediation analyses. However, a few adjustments are needed to address the nested-data structure. For instance, because of the multilevel design, paths a (predictor to mediator) and b (mediator to outcome when controlling for the predictor) vary across Level-1 units (i.e., participants) and can potentially covary. Estimates of the confidence interval for the indirect effect (a*b) and estimates of percent mediation need to take into account this potential covariation between paths a and b (Bauer et al. 2006). We used the multivariate approach described by Bauer et al. (2006) that simultaneously estimates paths a and b in the same model and produces estimates of the covariation of these two paths. To produce confidence intervals and significance tests for indirect effects (i.e., a*b) we used the asymmetric distribution of products tests, as described by MacKinnon et al. (2002, 2007) which can take into account the covariation between paths a and b. To produce an estimate of the strength of the indirect effect or mediation, we computed the percentage of the total relationship between the predictor and the outcome (total effect = c' + ab + covariance of ab) that was accounted for by the indirect effect (ab + covariance of ab).

Table 1 summarizes the results of the mediation analyses. The most important finding was that the indirect path from time to PTSD through AD (Model A) was not statistically significant (see the indirect path a*b and associated 95 % CI) and did not lead to a reduction in path c. On the other hand, the indirect path from time to AD through PTSD was significant (see the indirect path a*b and associated 95 % CI for Model B) and accounted for 24 % in the change in AD, and path c was reduced. This suggests that changes in PTSD account for subsequent changes in AD, but not vice versa.

To evaluate the impact of sensor placements (T4-T3) we included a dummy-coded sensor placement variable as predictors of the growth curve change parameters (intercept and slope). For PTSD symptoms, the sensor placement × Time interaction was not statistically significant ($b = -3.60$, $t = -5.66$, $p = .222$); however, the effect size estimate for the difference in change over time between the two sensor placements indicated a medium-to-large difference ($d = -.70$) between the placements. The T4 placement ($b = -13.93$, $t = -3.57$, $p = .002$, pre to follow-up decrease = 24.29, $d = -1.15$) tended to exhibit greater decreases than the T3 placement ($b = -7.26$, $t = -2.02$, $p = .056$, pre to follow-up decrease = 12.99, $d = -.45$). For AD, the sensor placement x Time interaction again was not statistically significant ($b = .20$, $t = .89$, $p = .893$) and the estimate of the difference in change between the two placements indicated a negligible effect size difference (d = −.06) with both groups showing comparable decreases ($d = -1.05$ and $-.99$ for the T3 and T4 placements, respectively). In sum, this pattern of findings suggest that our study was under powered to detect medium-large effect size differences between sensor placements, but the pattern of effect sizes estimates show some support for a larger decrease in PTSD symptoms for the T4 placement relative to the T3 placement.

Discussion

The results of the current study support the notion that NF may be a promising addition to existing treatments for PTSD. Overall, we found that individuals' PTSD

symptoms reduced significantly (from a score of 69.14 to 49.26), and that reductions in PTSD symptoms were related to decreases in an individuals' affect dysregulation (which decreased from a score of 23.63 to 17.20). We were unable to detect statistically significant differences in our comparison of sensor placements, and both appeared to provide benefit; however, there was a trend toward the T4-P4 placement showing a larger decrease in symptoms. Overall, we believe these findings provide support to justify further investigations including a rigorous control group.

While forty sessions of NF provided significant decreases in PTSD symptoms, it by no means provided complete recovery (merely a 20-point reduction). Our sample consisted of a chronic treatment resistant population, who had received a mean of more than 10 years of therapy. All had significant trauma exposure that resulted in developing PTSD prior to age 18. The mean time since their primary trauma exposure was more than 27 years. While we expected the most significant results with hyperarousal symptoms all three clusters of PTSD symptoms reduced significantly.

In addition, the reductions in PTSD symptoms preceded reductions in affect dysregulation symptoms. This contrasts with the findings of Linehan's group (Harned et al. 2010) who used DBT as a means of increasing affect regulation prior to exposure therapy. However, we can conjecture that affect regulation appears to be related to executive functioning (Zelazo and Cunningham 2007), which is likely a frontal lobe function. Our NF sensor placement was over the temporo-parietal regions of the brain, and therefore likely not directly affecting the neural underpinnings of the executive functioning system. Emerging data suggest that NF training targets brain areas in close spatial proximity to sensor placement. Colleagues of ours (Ros et al. 2013) recently studied a group of 21 individuals with PTSD related to childhood abuse in which they investigated whether one 30-min session of voluntary reduction of alpha rhythm (8–12 Hz) would be related to differences in EEG, network functional connectivity, and subjective measures of mental state. Alpha rhythm desynchronizing neurofeedback was associated with decreased alpha amplitude during training, followed by a significant increase (or "rebound") in resting alpha synchronization. This rebound was linked to increased calmness, greater salience network connectivity with the right insula, and enhanced default mode network connectivity with bilateral posterior cingulate, right middle frontal gyrus, and left medial prefrontal cortex. This suggests that changes in the brain corresponded spatially to the placement of NF sensors (Ros et al. 2013). Therefore, we may speculate that our NF protocol targeted limbic structures associated with the maintenance of PTSD. As recent models of the mechanisms of PTSD implicate a hyper-responsive limbic system with a hypo-responsive medial prefrontal cortex (McNally 2006; Rauch et al. 2006; Shin et al. 2004) we can speculate that there may have been a cascade effect. In summary, as the limbic structures became more regulated, this may have allowed the prefrontal cortex to better regulate affect.

When taken in the context of previous research on NF with PTSD, this study begins to justify and provide a roadmap for future studies. This is the first study to examine the effectiveness of NF in a non-veteran PTSD population. These results indicate that we may expect similar effects for NF in individuals with chronic PTSD. Finally, we speculate that NF has clear potential to increase the tolerability of treatment because clients are not asked to expose themselves to emotionally difficult traumatic material.

Of course, there are many more questions raised by these results than there are answers provided. For example, PTSD is not necessarily a homogeneous construct in that different individuals show markedly different symptom profiles. It will be important to further examine whether different sensor placements correlate with different symptom profiles. While our study was unable to find statistically significant differences between our sensor placements (both provided benefit), analyses appeared to indicate a trend for greater improvements with the T4-P4 protocol. Given the complexity of the brain, it is unreasonable to expect that a "one-size-fits-all" approach can be found with NF. (To wit, this same critique should be applied to any treatment approach.) The next step in advancing our research agenda is to replicate these results within a rigorously controlled trial. We have recently completed testing this intervention in comparison to a wait-list control. We determined that a sham placebo control was unethical given the time and resources that would be demanded of participants to travel to the clinic two times per week for up to 12 weeks. Additionally, given limited funding available, we would not have been able to reimburse participants adequately. However, we would like to begin to unpack the active components of the treatment including whether and what kind of talk therapy might contribute to positive outcomes, how many sessions are required for benefit, and whether medication can help or hinder response. Additionally, we were able to assess participants using pre- and post-intervention quantitative EEG's. This will allow us to examine whether EEG changes correlate with symptom changes.

Limitations

The current study can only be considered a pilot that provides the foundational framework for future research. We have shown that NF is well-tolerated and appears to help individuals with chronic PTSD. It appears that NF can help

civilians in a similar manner as veterans. However, this conclusion is greatly limited by the lack of a control group, lack of blinding to condition, and lack of the ability to systematically assess EEG changes associated with the NF training. Thus, it is unclear whether the benefit of NF came from the intervention itself, or from meeting with a NF technician twice a week. Thus, research employing both a wait-list control and an active control is warranted. Additionally, it will be important to compare NF to other active interventions, and to test out combinations of NF and talk therapy interventions.

The study is limited in power because of the small sample size. Additionally, the study lacks generalizability because the sample was heavily skewed toward being white and female. The small sample size means we may have lacked the statistical power to differentiate the efficacy of our alternate protocols. Thus, replications with larger and more diverse samples of participants are warranted.

Finally, we required all participants to be attending weekly therapy. However, we had no way to control for modalities or qualifications of therapists. Thus, it is unclear what therapy entailed for each participant. We also did not screen participants out for any medications that they were prescribed, and we had several participants who were actively taking benzodiazepines. Given clinical observation that this decreases treatment gains, it is possible that our results were negatively impacted.

Conclusion

Despite the aforementioned limitations, we found significant decreases in PTSD symptoms and affect dysregulation. Importantly, we have evidence that as PTSD symptoms decrease, it may cascade to other neural systems underlying self-regulation including affect regulation. While the limitations negatively affect our ability to identify the specificity of NF gains, they increase the ecological validity of the study. This was an effectiveness study in which the intervention was employed in a community setting with complex participants. In short, we enrolled many of the participants who would be excluded from more "rigorous" trials because of substance use, comorbidity, and other factors. In total we only screened one person out of the study. Once participating, we found that participants "no-show" rates were low, and we had a very low drop-out rate. Of 23 individuals screened for participation, 17 completed the 40-session protocol (73 % completion rate). Of those who were non-completers, one person completed the 40 sessions but did not return for the final assessment (78 % rate of completing the intervention). This compares favorably to extant forms of treatment, which report anywhere between 0 and 50 % dropout (for a review see Schottenbauer et al. 2008). Historically, when individuals drop out of a treatment outcome study, we ask the question, "what is wrong with the participants that they cannot tolerate the treatment?" We prefer to ask the question, "what is wrong with the treatment that the participants cannot tolerate it?" NF represents a paradigm shift that has the potential to address the latter question and reduce the stigmatization of chronically traumatized individuals who lack the capacities necessary to participate in extant forms of treatment.

Acknowledgments Richard Jacobson, Ilya Yacevich, Regina Musicaro, Marla Zucker Ph.D., Hilary Hogdon Ph.D., Janice Stubblefield. This study was supported by a Grant from the ANS Foundation.

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