PS₁

Title: Noninvasive Cranial Nerve Stimulation for Human Cognitive Performance Enhancement

Presenter(s): Taylor Hearn, BS Bioengineering; Sarah Wyckoff, PhD Neuroscience; Stephen Helms Tillery, PhD Neuroscience; William Tyler, PhD Behavioral Neuroscience

Abstract: Electrical stimulation of various cranial and spinal nerves is a rapidly growing area of study. Noninvasive approaches especially provide a safer, less expensive alternative to the pharmacological treatment of various psychological conditions. Specifically, vagus nerve stimulation (VNS) has been shown alleviate symptoms of major depressive disorder, trigeminal nerve stimulation (TNS), epilepsy, and cervical spinal nerve stimulation (CNS), stress (Berry et al., 2013; DeGiorgio et al., 2013; Tyler et al, 2015). These stimulation sites are all believed to innervate the locus coeruleus-norepinephrine system (Berry et al., 2013). Taking into account the extensive role norepinephrine plays in various executive functions, these stimulation techniques should be able to affect executive functioning in healthy subjects as well (Sara, 2009). As such, our protocol seeks to elucidate the neuromodulatory effects of noninvasive cranial nerve stimulation on attention.

A passive auditory oddball task was selected to measure attention. Subjects were instructed to listen to a series of 100 ms tones followed by 500 ms of silence. For each 600 ms trial, there was an 82% chance the tone would be at 750 Hz and an 18% chance it would be at 1,500 Hz. Tones were presented until 150 1,500 Hz tones were presented. Subjects were not required to physically or consciously respond to either type of tone. This task was created entirely with custom MATLAB (Natick, MA) software.

CNS, TNS, and VNS were all delivered using a custom, current-controlled stimulator connected to 2.5 cm round Axelgaard PALS® electrodes (Fallbrook, CA). Stimulation trains were delivered for 10 minutes between oddball tasks. Each train was symmetrically biphasic, charge-balanced, and cathodic-first. Subjects were randomly assigned to receive either CNS, TNS, or VNS at 30, 300, or 3,000 Hz with pulse durations of 50, 350, and 50 μs, respectively. After all testing, subjects completed a subjective report stimulation to describe the stimulation experience.

Electroencephalography (EEG), electrocardiography, galvanic skin response, respiratory rate, and hand temperature were all utilized to assess the physiological responses to stimulation.

Data collection is ongoing. EEG data will be averaged across subjects for each stimulation location and parameter set and presented as voltage traces and spectrograms to examine effects in both time and frequency domains. Physiological data will be averaged similarly to EEG data. Subjects' subjective experiences will also be reported.

PS 2

Title: The Frontal Alpha Asymmetry and Neurofeedback in Patients with Major Depressive Disorder

Presenter(s): I-Mei Lin, PhD; Ya-Ting Hung, BS; Sheng-Yu Fan, PhD; Yu Lee, MD; Nien-Mu Chiu, MD; Chi-Fa Hung, MD; Cheng-Fang Yen, MD; Yi-Chun Yeh, MD; Mei-Feng Huang, MD; Tai Ling Liu, MD; Peng-Wei Wang, MD; Huang Chi Lin, MD

Abstract: Background & Description: Previous study indicated that Frontal Alpha Asymmetry (FAA) is a biomarker for patients with Major Depressive Disorder (MDD; Davidson, 1998). Asymmetry scores (A1) were calculated from log-transformed of alpha power (8-12Hz), log (F4) $\hat{a} \in \text{``log (F3)}$. Some studies based on theoretical of FAA and applied alpha asymmetry neurofeedback (ALAY) showed some improvement in the depressive symptoms. However, the changes of electroencephalography (EEG) parameters were inconsistent (Baehr et al., 2001; Cheon et al., 2016; Choi., 2011; Wang et al., 2016). This study hypothesized the differences on EEG patterns between participants with FAA (A1 $\hat{a} \in \text{``log}$) and without FAA (A1+) among the healthy controls and patients with MDD; as well as the effect of ALAY neurofeedback on A1 score between the A1($\hat{a} \in \text{``log}$) group or the A1(+) group among patients with MDD.

Method: Study 1: The participants were composed 127 patients with MDD (72 in the A1+ group and 55 in the A1â€" group) and 129 healthy controls (87 in the A1+ group and 42 in the A1â€" group). Beck Depression Inventory-II was administered, and a 19-channel EEG cap with BrainAvatar (BrainMaster, Bedford, USA) has collected EEG raw signals at F3 and F4 and transformed to absolute alpha power (8-12Hz), and then calculated alpha asymmetry score (A1). Study 2: A total of 48 patients with MDD were assigned to the ALAY neurofeedback groups (A1+, n = 11 and A1â€", n = 13) and the control groups (A1+, n = 10 and A1â€", n = 12) based on their A1 score at pretest. The ALAY neurofeedback groups received 60 min, twice a week for 10 consecutive sessions of neurofeedback that was assisted by BioGraphy Infiniti 6.0 (Thought Technology, Quebec, Canada). The goal of neurofeedback was to increase A1 score with the visual and the auditory feedback. The control group received the normal treatment as usual. All of the participants underwent pretest and posttest measurement which included the performance on psychological questionnaires and EEG, which included the F3 alpha, F4 alpha, and A1 score.

Results: For EEG patterns, there was higher alpha power in the left prefrontal lobe (F3) in the A1(\hat{a} €") group compared to A1(+) group in the healthy controls; as well as lower alpha power in the right prefrontal lobe (F4) in the A1(\hat{a} €") group compared to A1(+) group in patients with MDD. Regarding the effect of ALAY neurofeedback, the ALAY A1(\hat{a} €") group significant increased the A1 score at posttest than the pretest. However, ALAY A1(+) group did not show significant improvements, as well as two MDD controls (A1+ and A1 \hat{a} €"). Both neurofeedback groups (ALAY A1 \hat{a} €" and ALAY A1+) significantly decreased the depression total score and cognitive depression, and ALAY A1(\hat{a} €") also decreased the anxiety score.

Conclusion: The high alpha power in the left prefrontal lobe in the healthy controls, and low alpha power in the right prefrontal lobe in the MDD group, were found in participants who had FAA. The neurofeedback therefore was beneficial for patients with FAA in decreasing depressive symptoms and increasing the A1 score.

PS 3

Title: A Real Time fMRI-neurofeedback for Mild to Severe Depression Compared to Frontal Alphaasymmetry Neurofeedback and Cognitive-Behavioral Therapy

Presenter(s): Mikhail Melnikov, PhD; Mark Shtark, PhD; Dmitriy Bezmaternykh, MPhys; Lyudmila Kozlova, MD; Mikhail Pokrovskiy; Andrey Savelov, PhD; Tato Sokhadze, PhD; Kira Natarova, PhD; Evgeniy Petrovskiy, MPhys; Tatiana Syrtchina, MD; Tatiana Andamova, MPsych

Abstract: Background: For the decades EEG neurofeedback has been the only one method of self-regulation of the brain activity in mental disorders. Current developments in fMRI technology made possible neuroimaging neurofeedback targeted to a well-defined cerebral area. Implementations of the technology aimed to enhance subjects' control of the activity of brain structures involved in emotion regulation were successful both in healthy volunteers (Johnston et al., 2011) and in patients with major depression (Hamilton et al., 2016; Linden et al., 2012; Young et al., 2017).

Objectives: The aim of our study was to examine effects of the real-time fMRI neurofeedback as a treatment arm for mild to severe depression. Alpha-asymmetry neurofeedback and cognitive-behavioral therapy (CBT) served as control treatment arms.

Methods: Thirty subjects (10 males, 20 females, aged 20-50, mean age of 33) were recruited and randomly assigned to experimental or one of two control groups. Participants of experimental group received 8 weekly sessions of fMRI neurofeedback targeted bilaterally to the area within medial prefrontal cortex. During each session blocks of enhancing and suppressing the response of the target area were alternating. Subjects continuously received visual feedback reflecting percent of signal change within the region of interest (ROI).

The source signal was recorded using Philips Ingenia 3T MR scanner with an EPI sequence, TR=1000 ms. Temporal dynamics of the signal from ROI was captured from the IViewBOLD graphs and presented on the screen as a yellow circle, diameter and brightness of which depended on the signal values. Offline fMRI analysis was performed using SPM 12 software. Concurrent EEG was recorded with a 32 channel MR-compatible BrainAmp system, corrected for MR, cardiac, and ocular artifacts and processed in EEGLab software. First control group patients received sixteen 25-minute sessions of frontal alpha-asymmetry neurofeedback. Participants from the second control group were treated with 8 individual and 8 group sessions of CBT. Each subject underwent psychiatric examination (MADRS), psychological assessment (BDI, SDS (Zung Self-Rating Depression Scale), HADS), and EEG-fMRI recording at rest and during performing an emotionally salient task at start, at middle, and at theend of the course.

Results: Patients from all the groups significantly improved from the treatment. A status of some patients according to DSM-5 changed to milder depression or to no depression condition. The fMRI-neurofeedback group showed significant improvements on MADRS, BDI, SDS, and HADS that were statistically comparable with those in alpha asymmetry neurofeedback and CBT. Patients of the fMRI group demonstrated ability to control prefrontal cortex signal both in usual feedback and in transfer (no feedback) sessions and gained positive changes of emotional state during sessions.

Conclusions: FMRI-based neurofeedback holds a promise for a targeted regulation of emotional circuits and can be considered as a potentially clinically efficacious technique of self-regulation in mood

disorders. However, cost-benefit ratio remains a problem for this application. Studies of EEG-fMRI correlates during the real time fMRI-neurofeedback sessions may be instrumental for enhancing EEG neurofeedback treatment protocols.

PS 4

Title: Preliminary Evidence for Stress-Reducing Effects of Bilateral Alternating Stimulation Tactile (BLAST) Following Significant Quantitative Electroencephalography (qEEG) Reduction in Beta Wave Activity

Presenter(s): Amy Serin, PhD; Dominic Di Loreto, MA; Emily Kade, MA

Abstract: Background: Bilateral alternating stimulation in tactile form (BLAST) technology has been found to significantly reduce subjective distress and physiological body sensations in response to thinking about a stressful event (Serin, Hageman & Kade, 2018). TouchPoints are noninvasive devices that deliver BLAST and are believed to reduce sympathetic nervous system arousal associated with anxiety (Busscher et al., 2013) by de-potentiating amygdala activity (Harper et al., 2009) responsible activating the body's stress response (Ehrlich et al., 2009). Beta EEG rhythm has been found to correlate to high situational and personal anxiety (Pavlenko et al., 2009). The purpose of this study was to utilize quantitative electroencephalography (qEEG) recordings to identify significant changes in electrical brain activity upon thinking of a stressful event and subsequently upon the delivery of BLAST. It was hypothesized that upon thinking of a stressful event beta activity would increase and subsequently reduce significantly upon delivery of BLAST. Methods: A total of 21 participants (9 male, 12 female) ages 7-63 (M age=27.8; SD=16.5) participated in the study and were recruited through the Serin Center. The sample consisted of 14 clinical participants with heterogeneous diagnoses of anxiety, major depressive disorder, and attention deficit hyperactivity disorder. The remaining sample consisted of non-clinical participants. QEEG data was collected at the Serin Center's locations in Peoria and Scottsdale Arizona. Data was collected utilizing a NeuroField Q20 amplifier and was stored using NeuroGuide by Applied Neuroscience Inc. Participants underwent a 5-minute baseline recording followed by an instruction to think about a stressful event. Participants were then asked to hold TouchPoints devices. 19-channel qEEG recordings were taken while thinking about the stressful event, during the delivery of BLAST (holding the TouchPoints), and then a baseline was taken again upon removal of the TouchPoints. Paired t-test analysis was conducted before and after BLAST with NeuroGuide's Neurostat software. Results: Preliminary EEG recordings comparing the stress condition to the TouchPoints condition exhibited significantly reduced activity in frontal Theta, specifically in 5Hz at Fp2 and F4 sites and reduced activity in Beta 1 at 12-14Hz in the frontal channel locations (Fp1, Fp2, Fp3, Fz, F4). Significant right frontal decreases are shown in Beta 2 16-18Hz, Beta 3 bands at 19 and 23Hz, and Gamma 1 at 30-35Hz with activity decreasing along the midline also in Beta 3 19Hz and 23Hz and in Gamma 1 at 30Hz. Conclusion: The significant reduction in beta activity provides preliminary evidence that BLAST technology may have a therapeutic effect on reducing subcortical activity associated with anxiety and stress. Our results are consistent with previous studies (Pavlenko et al., 2009) suggesting beta wave activity is correlated with increased levels of anxiety. This preliminary data implicates the potential efficacy of BLAST as a mediator of SNS arousal and stress through beta-activity reduction in both clinical and non-clinical samples. Follow-up research is required utilizing a comparison control group with a larger sample to assess for gEEG differences in brain activity.

PS 5

Title: Event-related potential study of illusory figure processing deficits in children with autism spectrum disorder

Presenter(s): Estate Sokhadze, PhD, BCN; Ayman El-Baz, PhD; Desmond Kelly, MD; Ricardo Jose De Leon, MS; Thomas Feiner, BS; Manuel Casanova, MD

Abstract: Background: Analysis of event-related potentials (ERP) is one of the most effective methods of investigation of information processing stages in the brain. ERP methodology represents a valuable technique to study normative cognitive processes in typically developing (TD) subjects, and at the same time may serve as a sensitive tool to assess differences in individuals with autism spectrum disorder (ASD). It was been shown in visual and auditory modalities in various types of oddball tasks that children with ASD present abnormalities in ERPs (Kemner et al., 1999; Bomba & Pang, 2004). Both the frontal P3a to novel stimuli and the parietal P3b to attended target stimuli were reported to be abnormal in autism (Cui et al., 2017; Townsend et al., 2001).

Objectives: In a series of studies (Baruth et al., 2010; Sokhadze et al., 2009, 2010) using various oddball tasks we showed that group differences between ASD and TD children can be found for both attended and non-attended stimuli not only in late potentials (P3a, P3b) but also in early ERPs (P100, N100) and response-locked ERP (ERN). Among oddball tasks the three-stimulus visual oddball with illusory figures was most informative for this purpose. The goal of the study was investigation of group differences in ERP recorded at midline frontal and parietal sites for determination if these topographies are reflecting atypicality of ERP in the ASD.

Methods: Seventy children with ASD and 30 typical children performed on an oddball task with illusory figures. EEG was collected using a 128-channel EEG system. The task involved the recognition of a specific illusory shape, in this case a square or triangle, created by three or four inducer disks (Kanizsa, 1976). The regions-of-interest (ROI) for ERP analysis were only frontal and parietal midline areas.

Results: Children with ASD did not differ from typical children in reaction time (RT), but they committed more errors (12.9% vs.2.2 %, F=14.9, p<0.001) and did not show normative post-error RT slowing (F=27.6, p<0.001). The error-related negativity was lower in ASD (F=8.5, p=0.004). The early ERPs (P100 and N100) to non-target stimuli were of higher amplitude and delayed in the ASD group (ps<0.05). The late ERPs (P3a and P3b) to non-target stimuli were prolonged in ASD without amplitude differences, though P3a was delayed as well to targets in the ASD (458 vs. 426 ms, F=4.9, p=0.03).

Conclusions: Results are in concordance with our prior studies where children with ASD showed excessive reactivity to task-irrelevant stimuli at the early sensory stage processing of information leading to delayed cognitive ERP to targets resulting in error monitoring and correction deficits. It was important to replicate these findings when ERPs were analyzed only at the midline frontal and parietal areas as it may have practical implications. It creates opportunity for our group to start development of a custom-made experimental control and EEG acquisition system with limited number of channels (e.g., Fz, Pz) for ERP analysis in oddball test with illusory figures that can be used for functional diagnostic and as outcome of neurofeedback or neuromodulation interventions.

PS 6

Title: Effects rTMS-based Neuromodulation Dosage on Event-related Potentials and Evoked and Induced Gamma Oscillations in Children with Autism Spectrum Disorder

Presenter(s): Estate Sokhadze, PhD, BCN; Eva Lamina, PhD; Emily Casanova, PhD; Desmond Kelly, MD; Ayman El-Baz, PhD; Guela Sokhadze, MS; Manuel Casanova, MD

Abstract: Background. Autism is defined as a spectrum of behavioral disorders that have in-common impairments in social interaction and communication skills, language deficits, and a restricted repertoire of interests and stereotyped activities. There are several theoretical models of the neuropathology of autism spectrum disorders (ASD), and one of them suggests the presence of an excessive cortical excitation/inhibition (E/I) ratio (Casanova, 2003; Rubinstein & Merzernich, 2003; Uzunova et al., 2015) that affects functional connectivity. This model explains atypical event-related potential (ERP) and evoked and induced gamma oscillations observed in ASD during task performance. Repetitive transcranial magnetic stimulation (rTMS), especially using low frequency inhibitory stimulation, can be considered as a method of modulating the E/I bias.

Objectives: In our prior exploratory studies (Sokhadze et al., 2009, 2010) we used different schedules of rTMS to investigate outcomes of rTMS in ASD. In this study, 124 high functioning ASD children (IQ>80, <18 years of age) were recruited and assigned to either a waitlist group or one of three different number of weekly rTMS sessions (i.e., 6, 12, 18) to investigate effects of dosage on functional and behavioral outcomes. The project was aimed at selection of more effective length of rTMS course.

Methods: TMS consisted of trains of 1.0 Hz pulses applied over dorsolateral prefrontal cortex. The experimental task was a three-stimulus visual oddball with illusory Kanizsa figures. Behavioral response variables included reaction time and error rate along with EEG indices such as ERP and evoked and induced gamma oscillations. One hundred and twelve patients completed the assigned number of rTMS sessions.

Results: We found significant positive changes from baseline to post-TMS treatment period in motor responses accuracy (lower percentage of committed errors, restored normative post-error slowing), in ERP indices and in evoked and induced gamma responses. Parental reports showed significant reductions in aberrant behavior scores as well as decreased scores of repetitive and stereotypic behaviors. The gains of outcomes increased with the total number of treatment sessions. Results of our clinical research study showed most significant changes from baseline in functional measures of performance in oddball task and in behavioral symptom ratings following 18 sessions of rTMS treatment. Several measures showed a difference from baseline and waitlist in reaction time and ERP/EEG variables after 12 sessions of rTMS, but only a few of them reached statistical significance post-6 session rTMS course.

Conclusions: Our results suggest that rTMS, particularly after 18 sessions, facilitates cognitive control, attention and target stimuli recognition by improving discrimination between task-relevant and task-irrelevant illusory figures in an oddball test. Improvement in executive functions and behavioral symptoms of autism further suggests that TMS has the potential to target core features of ASD. The results of this dosage-response study could serve as important pre-requisites that could inform the

planning of a blinded randomized clinical trial. Among potential implications of the study should be considered potential of combining rTMS with neurofeedback training (Sokhadze et al., 2014) aimed at reinforcement of neuromodulation effects using operant conditioning in similar manner as reported by our group earlier.

PS 7

Title: Lateralized Readiness Potentials in Children with Autism Spectrum Disorder during Posner Cueing Task: An Event-related EEG Study

Presenter(s): Guela Sokhadze, MS; Tato Sokhadze, PhD BCN; Manuel Casanova, MD

Abstract: Background: Autism spectrum disorder (ASD) is a developmental disorder characterized by social communication deficits, and engagement in restricted, stereotyped behaviors. An estimated 80% of individuals with ASD also display dyspraxia (Weimer et al., 2001; Dowell et al., 2009), a condition involving difficulties in motor coordination and sequencing, as well as speech production. However, it is unclear how the processing, preparation, and execution phases of motor movement are affected by dyspraxia in ASD. We examined EEG activity and behavioral indices in children diagnosed with ASD during performance of a visuo-motor spatial attention task.

Methods: Participants included 30 children diagnosed with ASD (15.6 $\text{Å}\pm$ 3.8 years old, 8 girls), and an age-matched control group of 30 typically-developing children (TD) (15.7 $\text{Å}\pm$ 3.9 years old, 7 girls). Subjects performed a modified Posner's attentional cueing task (Posner, 1980). In each trial, subjects were initially presented a visual "cue" stimulus on left or right side of the screen. After a 1 second delay, a "target" stimulus appeared on the same (congruent, 80%) or opposite (incongruent, 20%) side from the cue, and subjects used a left- or right-handed button press to indicate the position of the target. In half of the trials, a more complex diagonal stimulus presentation was used. EEG data was collected for analysis of several event-related potentials (ERPs), including the lateralized readiness potential (LRP), a measurement of asymmetric brain activity that reflects preparation of contralateral limb movement (Eimer, 1998).

Results: Reaction time (RT) was lower for congruent trials compared to incongruent trials for both ASD (401 vs. 481 ms, p < .0001) and TD (339 vs. 374 ms, p < .001). Across all 4 task conditions, ASD group exhibited longer RTs and higher error rates compared to TD (441 vs. 358 ms, p < 0.001; 7.4 vs. 0.8 errors, p < 0.0001). Furthermore, increased task complexity resulted in lengthened RT in ASD group (447 vs. 430 ms, p < 0.05), and in TD group (376 vs. 360 ms, p < 0.01). While the amplitude the early (pre-target) component of LRP was significantly higher in ASD compared to TD (-0.95 vs. -0.23, p < 0.05), the late component (post-target) showed no group differences (-0.53 vs. -0.50, n.s.). Moreover, analysis of ERPs showed several differences between ASD and TD groups, including frontal N100 amplitude, N100 latency, and N200 amplitude.

Discussion and Conclusions: Shorter RTs to congruent trials in ASD and TD suggests that both groups exhibited an attentional bias toward the cued side. However, ASD group had higher RTs and lower accuracy regardless of trial condition, indicating poorer performance compared to TD. EEG analysis demonstrated that ASD group exhibited differences in the early ERPs and LRP, indicating differences at the early cognitive phase of stimulus processing and movement preparation rather that at the late motor execution phase. A more in-depth understanding of abnormalities in LRP and other ERPs during motor task performance could shed light on the underlying neuropathology of dyspraxia in autism, and could potentially serve as a useful biomarker for early diagnosis.

PS 8

Title: The effects of ALAY and high beta down-train neurofeedback for patients who comorbid with major depressive disorder and anxiety symptoms

Presenter(s): San Yu Wang, Bachelor; I-Mei Lin, PhD; Yu-Che Tsai, PhD; Cheng-Fang Yen, PhD; Yi-Chun Yeh, PhD; Mei-Feng Huang, PhD; Tai Ling Liu, MD; Peng-Wei Wang, MD; Huang Chi Lin, MD; Yu Lee, MD; Nien-Mu Chiu, MD; Chi-Fa Hung, MD

Abstract: Background & Description: Previous studies indicated that Frontal Alpha Asymmetry (FAA) and parietal hyperactivity among patients who comorbid with Major Depressive Disorder (MDD; Bruder et al., 1997; Mathersul et al., 2008) and high level anxiety symptoms. The purpose of this study was to examine the effects of alpha asymmetry (ALAY) and high beta down-train (BETA) neurofeedback protocols on emotional symptoms and electroencephalogram (EEG) among patients with MDD and anxiety symptoms.

Methods: Patients with MDD were referred by psychiatrists based on DSM-5 criteria, and with the scores of Beck Depression Inventory II (BDI-II) and Beck Anxiety Inventory (BAI) which were higher than 14 and 8. Eight-seven participants were assigned to ALAY neurofeedback (ALAY group; n = 24), high beta down-train neurofeedback (BETA group), and the control group (n = 23). All participants received BDI-II, BAI, and a five-min resting EEG with eye-closed measurement by using a 19-channel EEG cap with BrainAvatar equipment (BrainMaster Technologies, Inc., Bedford, Ohio) for pretest and posttest. The EEG raw signals were analyzed to alpha power (8-12Hz) and high beta power (20-32Hz), and then calculated to the A1 score (log [F4 alpha] – log [F3 alpha]) and high beta at P3 and P4. Both neurofeedback groups received 60-min treatment, twice a week, for 10 consecutive sessions by using ProComp Infiniti (Thought Technology Ltd., Montreal, Quebec, Canada). The goal of the ALAY group was to increase the A1 score, while the BETA group was to decrease high beta at P3 and P4.

Results: There was a significant decreased the symptoms of depression and anxiety in both ALAY and BETA groups (ALAY group: F(1,23) = 26.07, p < .001; F(1,23) = 13.73, p = .001; BETA group: F(1,22) = 24.27, p < .001; F(1,22) = 33.06, p < .001); as well as lower anxiety level was found in the posttest in ALAY and BETA groups compared to the control group (F(2,67) = 9.48, p < .001). However, lower level of depressive symptoms at posttest was found only in the BETA group compared to the control group (F(2,67) = 4.56, p = .014). There was a significant decrease in P3 high beta in the BETA group at posttest than that at pretest (F(1,22) = 8.64, p = .008) while significant increase in P3 high beta in the control group at posttest, than that at pretest (F(1,22) = 6.28, p = .020). However, there was no significant interaction effect which was found in A1 score, F3 total alpha, or F4 total alpha between the ALAY group and the control group (F(1,44) = 0.91, p = .345; F(1,45) = 0.002, p = .967; F(1,44) = 0.02, p = .882).

Conclusion: This study indicated that both ALAY and BETA neurofeedback protocols significantly decreased the symptoms of depression and anxiety among the patients who comorbid with MDD and high-level anxiety. Moreover, there was a significant decrease in high beta activity at posterior region (P3) which was found in high beta down-train neurofeedback protocol.

PS 9

Title: The Concussion Cure

Presenter(s): Paul Henry Wand, MD

Abstract: This unique and innovative book has two main components:

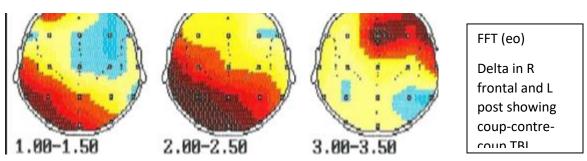
1. How to diagnose a concussion when conventional testing is normal.

2. How to treat concussions by addressing the pathophysiology in the brain after a concussion has occurred.

HOW TO DIAGNOSE A CONCUSSION

There are three principal ways to make the diagnosis.

- 1. Perform a detailed neurological examination (or have a consult done by a competent neurologist)
- 2. Perform a careful and complete EEG with at least 10 minute of eyes open, 10 minutes of eyes closed, and 3 minutes of hyperventilation if not medically contra-indicated. If these results are normal, you may add reading and listening for 10 minutes each. The EEG should be at 26 minutes, or 46 minutes with the latter added on. Perform a careful QEEG with special attention to artifacting. Be sure to analyze suspicious discharges with JFTA, and LORETA looking for focal findings. Look for topographic changes consistent with coup-contrecoup and match with the history of how the injury happened. Perform both the TBI Discriminant Score and Concussion Index by Dr. Thatcher.
- 3. Order or have a physician order a Ceretec SPECT Scan preferably on a dedicated triple headed camera system. If they have the Neurogam statistical package, make sure that data is included in the images as some places do not include it automatically. Request the images be included either in the report or provided for separately. Never send to a center that rarely does SPECT scans, so ask before sending how many do they do a month? Many nuclear scan centers do not do the brain as it is much more difficult to image properly.



HOW TO TREAT A CONCUSSION

1. From the QEEG, formulate a treatment protocol using the symptom checklist and the most significantly abnormal findings on the Q. For the training, always perform the LORETA type of feedback unless there are no findings on the LORETA slices which would be unusual. Always use

Z-score training and perform trend analysis after each session and carefully observe that the Z scores are trending down toward the normal. This will also tell you when to stop treating.

- 2. Order or have a physician prescribe a pharmaceutical called nimodipine, a specific calcium channel blocker to increase circulation in the brain. It is a safe, effective drug which I have used with great success since 1990.
- 3. Order or have a physician prescribe HBOT or hyperbaric oxygen at 1.5-2.0 ATM usually in a series of 40 sessions. Some patients need more, and this treatment can be serially over time.
- 4. Nutraceuticals as discussed in detail in my book which include those to increase oxygen in the brain, those to reduce inflammation and swelling.

FINAL NOTE

It has been my clinical experience since 2007 that when all of the modalities of treatment are performed at the same time, that there is a synergistic effect, and thus the clinical outcome is improved. This principle is true for all categories of patients, ranging from the mTBI to severe.

Top Row Pre-HBOT and Bottom Row Post-HBOT

