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A QEEG Activation Methodology That Obtained 100% Accuracy in the Discrimination of Traumatic Brain Injured from Normal and Does the Learning Disabled Show the Brain Injury Pattern?

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Abstract

Previous research has focused on determining whether the quantitative EEG (QEEG) can discriminate a traumatic brain injury (TBI) participant from a normal individual. The research has differed with respect to the critical variables involved in the discrimination task. All the research has limited its approach to the collection of eyes-closed data and most confine themselves to less than 32 Hz. The present research employs four cognitive activation tasks, an eyes-closed task, 19 locations, Spectral Correlation Coefficient (SCC) and phase algorithms in the beta2 frequency range (32–64 Hz), and the relative power of beta2 in six frontal locations to obtain 100% correct identification in original discriminant analysis. In addition, 50 random misclassifications—involving different participants—across the five tasks in a group of 196 subjects were correctly identified as misclassifications. To determine if a learning disability would show a similar pattern to a TBI pattern, a preliminary analysis of a group of 94 normal and learning disability (LD) participants were examined for their QEEG differences. The pattern evident in the analysis for the LD group (decreased coherence and phase alpha) was not the pattern evident in the TBI group, while the TBI pattern of decreased coherence and phase beta2 was not dominant in the LD group.

Keywords: quantitative EEG; traumatic brain injury; discriminant analysis; TBI discriminant; cognitive activation QEEG

Background

Previous research which has addressed the issue of statistically discriminating traumatic brain injury participants from normal individuals include publications by Thatcher, Walker, Gerson, and Geisler (1989); Thatcher, Biver, McAlaster, and Salazar (1998); Hughes and John (1989); Tabano, Cameroni, and Gallozzi (1988); Trudeau et al., (1998); Barr, Prichep,

Chabot, Powell, and McCrea (2012); Thornton (1997, 1999, 2000, 2003, 2014); and Thornton and Carmody (2009).

Tabano et al. (1988) investigated posterior activity of subjects (*N* = 18) at 3 and 10 days following a mild traumatic brain injury (MTBI) and found an increase in the mean power of the lower alpha range (8–10 Hz), a reduction in fast alpha (10.5–13.5 Hz) with an accompanying shift of the mean power of the lower alpha range (8–10 Hz), and reduction in fast alpha (10.5–13.5 Hz) with an accompanying shift of the mean alpha frequency to lower values. They also reported a reduction in fast beta (20.5–36 Hz) activity. They did not conduct a discriminant analysis of traumatic brain injuries (TBI) vs. normals.

Thatcher et al. (1989) were the first to attempt to conduct a discriminant function analysis. They used the eyes-closed QEEG data to differentiate between 608 MTBI adult patients and 108 age-matched controls and obtained a discriminant accuracy rate of 90%. Moderate to severe cases were not included in the analysis, nor was the high frequency gamma band (32–64 Hz) or cognitive activation conditions. The useful QEEG measures included increased frontal theta coherence (Fp1–F3), decreased frontal beta (13–22 Hz) phase (Fp2–F4, F3–F4), increased coherence beta (T3–T5, C3–P3), and reduced posterior relative power alpha (P3, P4, T5, T6, O1, O2, T4). Three independent cross validations (reported within the original research) resulted in accuracy rates of 84%, 93%, and 90%.

Thatcher et al. (1998) were able to demonstrate a relationship between increased theta amplitudes and increased white matter T2 Magnetic Resonance Imaging (MRI) relaxation times (indicator of dysfunction) in a sample of mild TBI subjects. Decreased alpha and beta amplitudes were associated with lengthened gray matter T2 MRI relaxation times. The subjects were 10 days to 11 years post-injury. This study integrated MRI, QEEG with eyes closed, and neuropsychological measures in a sample of MTBI subjects. Thatcher et al. (2001) employed this method to develop a severity of brain injury value.

One review of the research in the TBI area indicated that numerous eyes-closed EEG and QEEG studies of severe head injury (Glascow Coma Scale [GCS] score of 4–8) and moderate injury (GCS score of 9–12) have agreed that increased theta and decreased alpha power (microvolts) and/or decreased coherence and symmetry deviations from normal groups often characterize such patients (Hughes & John, 1999). The authors asserted that changes in these measures provide the best predictors of long-term outcome. The Thatcher discriminant function (Thatcher et al., 1989) correctly identified 88% of the soldiers with a blast injury history and 75% with no blast injury history (Trudeau et al., 1998).

Other studies have reported that similar QEEG abnormalities are correlated with the numbers of bouts or knockouts in boxers (Ross, Cole, Thompson, & Kim, 1983) and with professional soccer players who frequently used their heads to affect the soccer ball's trajectory ("headers"; Tysvaer, Storli, & Bachen, 1989). Neither of these research reports attempted to develop a discriminant function analysis.

Barr et al. (2012) took EEG recordings from five frontal locations (F7, Fp1, Fp2, F8, and a location below Fz) immediately post-concussion and 8 and 45 days after. They examined the frequency range up to 45 Hz on measures of absolute power, relative power, mean frequency, coherence, symmetry, and a fractal measure. Using a brain injury algorithm, abnormal features of brain electrical activity were detected in athletes with concussion at the time of injury, which persisted beyond the point of recovery on clinical measures. Features that contributed most to the discriminant applied in this study included

- relative power increase in slow waves (delta and theta frequency bands) in frontal;
- relative power decreases in alpha 1 and alpha 2 in frontal regions;
- power asymmetries in theta and total power between lateral and midline frontal regions;
- incoherence in slow waves between fronto-polar regions;
- decrease in mean frequency of the total spectrum composited across frontal regions; and
- abnormalities in other measures of connectivity, including mutual information and entropy.

A resulting discriminant score was employed to distinguish between the TBI and normal group. If the discriminant score was above 65, there was a 95% probability that the individual had experienced a TBI. The average discriminant score changed from the immediate post-concussion score of 75 to a score of 55, 45 days later; thus rendering its ability to discriminate 45 days after the original concussion not as useful as would be desired. The TBI's cognitive status, as assessed with neuropsychological measures, had returned to the "normal" range at day 45, although brain abnormalities were still present (TBI n = 59). The researchers did not internally attempt to replicate the findings within the sample that they had obtained.

Leon-Carrion, Martin-Rodriguez, Damas-Lopez, Martin, and Dominguez-Morales (2008) documented the discriminant ability of the QEEG to accurately classify brain injury in 100% of the "training set sample" (n = 48) and obtained a 75% correct classification in "an external cross-validation sample" of 33. The average time between the QEEG evaluation and incident (TBI, CVA) was 22 months. The authors noted that "coherence measures were the most numerous variables in the function," employing the frequency range of 1–30 Hz.

Previous research by Thornton (1997, 1999, 2000) focused on the damage to the Spectral Correlation Coefficients (SCC; based upon the Lexicor algorithms) and phase values in the beta2 (gamma; 32–64 Hertz) range when comparing the traumatic brain injured subject to the normal group during eyes-closed and different cognitive activation tasks. The TBI sample size ranged from 22 to 32 with 52 normal participants in the 1999 and 2000 studies. Lexicor Medical Technology (Boulder, CO) company developed their own algorithms for coherence and phase. The coherence measure algorithms were not the same as employed in the Barr et al. (2012) study.

The Thornton results (1997, 1999, 2000) did not indicate any deficits in the amplitudes or relative power of delta, theta, or alpha. In the Thornton (2003) article addressing auditory memory, the alpha level was set to .02 due to high number of significant findings in the beta2 SCC and phase values predominantly in the values involving the frontal lobe. The TBI group showed lower beta2 coherence (SCC) values. The article studied the relations between the QEEG variables and memory performance in 85 TBI patients and 56 normal subjects.

Thornton and Carmody (2009) and Thornton (2014) investigated the use of frontal beta and delta activity as well as coherence (SCC) and phase relations within the frontal locations to distinguish between TBI and normal participants. The Thornton (2014) chapter obtained a 97.5% to 100% accuracy rate in the discrimination analysis.

Methods

The participants underwent a cognitive QEEG evaluation, which consisted of an eyes-closed condition (300 seconds), auditory attention task (200 seconds), visual attention task (200 seconds), four auditory memory tasks (200 seconds), one reading task (100 seconds) in addition to a problem-solving (Ravens matrices) task. The auditory attention task consisted of the participant listening to the sound of a pen tapping on a table while their eyes were closed, and raising their right index finger when they heard the sound. The visual attention task required the participant to look at a page of upside down Spanish text. The participant was asked to raise their right index finger when a laser light was flashed on the text. The auditory memory tasks required the participant to listen to four individually administered stories with their eyes closed, quietly recall the story, and then repeat the story back to the examiner. The reading task required the subject to read a story presented on a laminated sheet for 100 seconds, quietly recall the story while their eyes are closed, and then recall the story to the examiner. During all of these tasks QEEG data was collected. The data for the eyes-closed condition and four cognitive activation tasks (auditory and visual attention, listening, reading) were employed for the discriminant analysis.

Participants

A different number of participants were available for the different tasks. There was a range of 162–197 subjects involved in the different conditions. The listening task had the largest number of participants. The average age of the total sample (listening task data) was 37 with a range between 9.4 years to 72.42 years. There were 95 males and 102 females in the listening task group (*N* = 197). There were 88 participants classified as TBI and 109 participants classified as normal. The time between the date of the head injury and evaluation ranged from 12 days to 30 years. The child group consisted of 49 normal children (average age = 10.6) and 45 children (average age = 10.6) who could be classified as having a learning disability (LD). There were 63 males and 31 females in the child sample. Thirty-seven of the 45 LD group were males, not an untypical finding. The subjects were protected and the data was collected in accordance with the Declaration of Helsinki.

Quantitative EEG (QEEG) Measures

Activation / Arousal Measures

RP: Relative Magnitude/Microvolt or Relative Power: the relative magnitude of a band defined as the absolute microvolt of the particular band divided by the total microvolt generated at a particular location across all bands

Connectivity Measures

C: Coherence or Spectral Correlation Coefficients (SCC): the average similarity between the waveforms of a particular band in two locations over the epoch (one second). The SCC variable is conceptualized as the strength or number of connections between two locations and is a correlation of the magnitudes.

P: Phase: the time lag between two locations of a particular band as defined by how soon after the beginning of an epoch a particular waveform at location #1 is matched in location #2.

Results

Figure 1 presents the relations that were significantly below the normative reference group ($p < .05$) for the SCC and phase values. The blackened circle is the indication that the location is the origin of a metaphorical flashlight, which is sending out a beam to three other locations. The flashlight locations were chosen according to the number of significant relations emanating from that location. In deciding if whether the source of a connection between two locations (A and B) is A or B, the location with the higher number of other significant relations was determined to be the source. It is of interest to note that the deficit patterns were distributed throughout the 19 locations and were not primarily focused on frontal locations, contrary to a commonly held belief that a head injury's primary location of injury is the frontal lobes. The most affected locations evident in Figure 1 appear to be the T4 location and posterior locations (T5, O1, O2, T6) for the SCC relations. There is also a pattern, mostly emanating from frontal locations, of the effect occurring across the hemispheres for more distant locations, while posterior flashlights involved mostly shorter connections. Figure 2 presents summary figures of all the significant relations (coherence and phase) as well as the frontal RPB2 locations. As the figures indicate, the effect is broad and diffusely located.

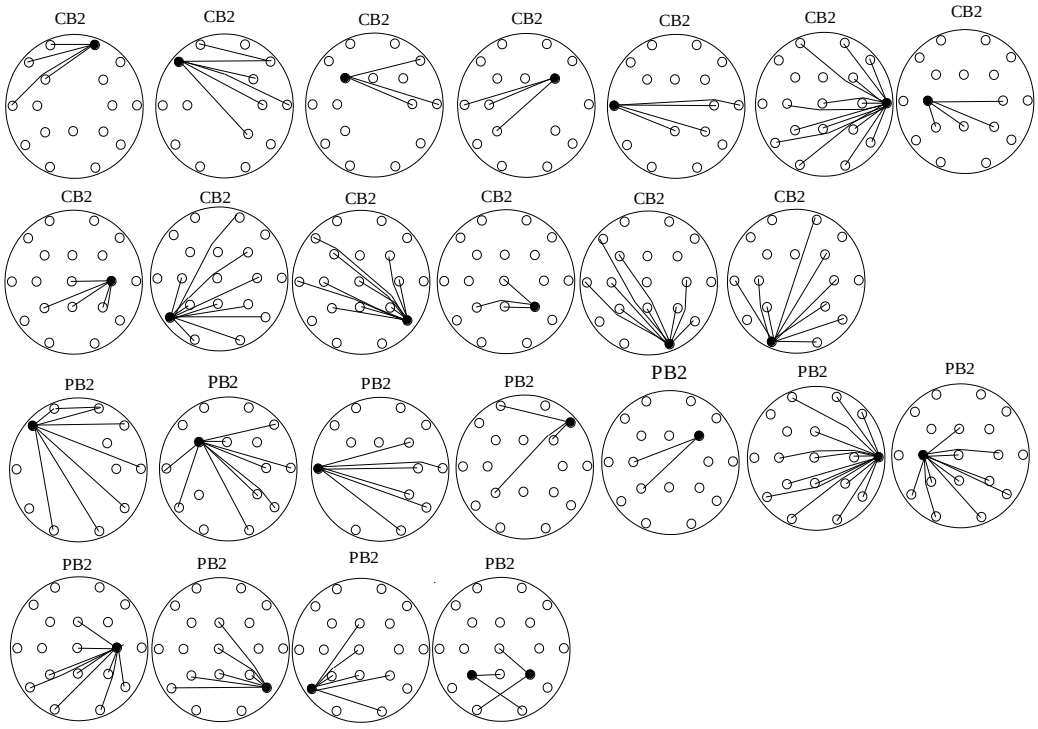

Figure 1. Significant SCC and phase deficits in the TBI participant. CB2 = Coherence (SCC) Beta2; PB2 = Phase Beta2.

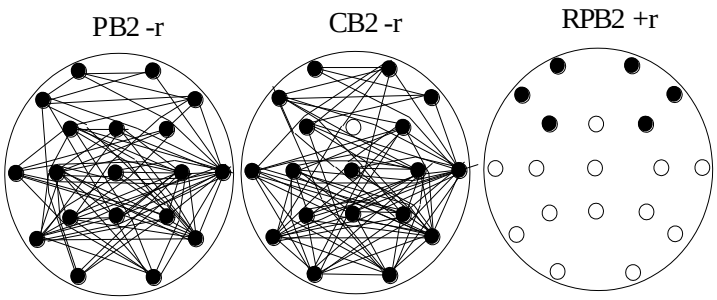

Figure 2. Summary head figures. CB2 = Coherence (SCC) Beta2; PB2 = Phase Beta2; RPB2 = Relative Power Beta2.

The average standard deviation (*SD*) difference between the normal and TBI group for the SCC variable was .47, and .44 for the phase variables for all the variables, which is significant at the .05 alpha level. The frontal relative power values of beta2 indicated a similar average *SD* value difference of .47 between the TBI and normal group.

Tables 1–5 present the resulting discriminant analysis for the five tasks. As the tables indicate, the discriminant analysis was 100% effective in distinguishing between the TBI and normal participants. The variables employed were the SCC and phase values in the 32–64 Hz range and the RPB2 values for the six frontal locations indicated in Figure 2.

Table 1
Classification Matrix (EC) – Eyes Closed

	EC	**TBI**	**Normal**
	Correct	*p* = .56	*p* = .44
TBI	100	102	0
Normal	100	0	81
Total	100	102	81

Table 2
Classification Matrix (AA) – Auditory Attention

	AA	**TBI**	**Normal**
	Correct	*p* = .51	*p* = .49
TBI	100	90	0
Normal	100	0	86
Total	100	90	86

Table 3
Classification Matrix (VA) – Visual Attention

	EC	TBI	Normal
	Correct	*p* = .52	*p* = .48
TBI	100	87	0
Normal	100	0	81
Total	100	87	81

Table 4
Classification Matrix (Listen) – Auditory Memory

	EC	TBI	Normal
	Correct	*p* = .45	*p* = .55
TBI	100	88	0
Normal	100	0	109
Total	100	88	109

Table 5
Classification Matrix (RS) – Reading

	EC	TBI	Normal
	Correct	*p* = .46	*p* = .54
TBI	100	75	0
Normal	100	0	87
Total	100	75	87

To determine if the discriminant algorithm could accurately indicate a misclassification, five TBI subjects and five normal subjects were misclassified for each task as to their status and the discriminant analysis was recalculated to determine if the inaccurate classification was identified. Ten different subjects were selected for each task for a total of 50 misclassifications. The discriminant reanalysis was 100% correct in the identification of all of the misclassifications.

Could a Learning Disabled Participant Be Identified as Brain Injured?

A potential diagnostic problem would be the presence of a pre-existing learning problem, which could show a similar pattern to a TBI. A preliminary investigation of this problem was undertaken by the author with the clinical data available. Two problems were initially evident. One is a lack of sufficient adult learning disabled (LD) to compare to adult TBI group. The second problem is the presence of a strong developmental pattern showing increases in

almost all coherence and phase relations. This developmental pattern would negate the use of comparing the values of children to normal adults or TBI adults, as the child's numbers would be lower strictly due to development patterns. Thus, the only viable method to assess for the diagnostic problem would be to compare children with LD to the normal control group and determine if the LD child deviant patterns from the normative reference group would be similar to the adult TBI patterns from a corresponding adult normative reference group. The reasonable assumption is that a child's TBI pattern would be the same as an adult's TBI pattern. Figure 3 presents the results of this analysis for a group of 49 normal children and 45 children who pursued EEG biofeedback treatment for cognitive/learning problems. To allow the reader to see the more dominant patterns, the circles were blackened for locations that contained three significant relations.

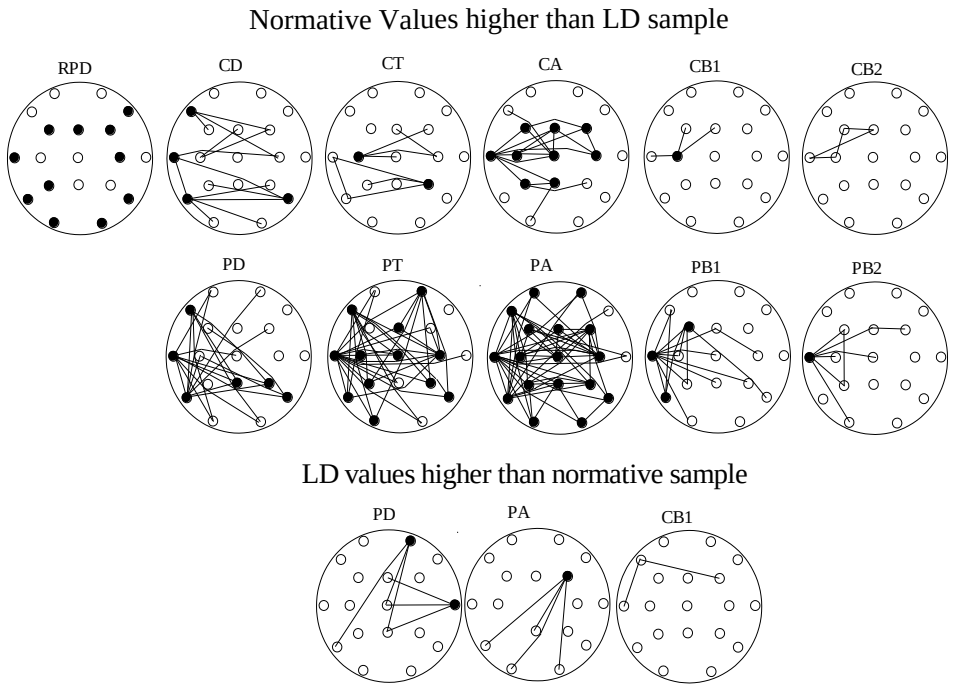

Figure 3. LD vs normal patterns during reading task. CD = Coherence Delta; CT = Coherence Theta; CA = Coherence Alpha; CB1 = Coherence Beta1; CB2 = Coherence Beta2; PD = Phase Delta; PT = Phase Theta; PA = Phase Alpha; PB1 = Phase Beta1; PB2 = Phase Beta2; RPD = Relative Power Delta.

As Figure 3 indicates, the LD pattern shows deficit patterns predominantly in the phase theta and alpha variables from the F7, T3, and T5 locations. The coherence and phase beta2 variables do not appear strongly involved in the deficit patterns. While preliminary, it is encouraging that this diagnostic potential problem may not present a real problem. Other cognitive tasks underwent a similar analysis. While the patterns differed, there was no strong evidence of an overwhelming deficit in the coherence or phase beta2 values in the LD group, which was evident in the TBI group. Correspondingly, in the TBI group the pattern of overwhelming decreased values in the lower frequencies was not evident. However, there are several qualifications with this data. The diagnostic issues with the LD group were not validated with standardized psycho-educational or neuropsychological batteries. In addition,

there were uncertain issues with respect to a history of head injury in the LD sample, which confuses the diagnostic issue. What, however, is also evident in the data is that the LD sample does not show the standard pattern of ADD or ADHD as there were no elevations in theta or alpha relative power.

Discussion

A method that can obtain 100% accuracy is a valuable aide in the diagnosis of a traumatic brain injury and is a valuable asset to the medical personnel in charge of rendering the diagnosis. It is important, however, that the method and results be further replicated for confirmation. Nevertheless, in the case of a pre-existing concussion the software would not be accurate in the determination of a present concussion. In the sports arena, this problem could be addressed by a baseline evaluation prior to the athletic season. The concern of a pre-existing learning problem appearing as a TBI does not appear to be an issue, according to some preliminary data analysis the author has available. However, the data analysis involved children and the conclusion extrapolated to adults. A sounder basis for the conclusion would be obtained with adolescent and adult learning disabled and an adolescent and adult TBI group.

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Treating Trauma Survivors with Neurofeedback: A Grounded Theory Study

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Abstract

Neuroscience, the mental health field, and the concept of trauma as an underlying factor in mental and physical disorders have been inextricably linked since the inception of the mental health professions. Numerous quantitative studies have indicated that neurofeedback may be effective in ameliorating trauma symptoms; however, there is a paucity of research exploring the factors that produce those positive outcomes. The purpose of this qualitative grounded theory study was to explore the factors and processes that influence treatment outcomes when neurofeedback is used with trauma survivors. Thirty interviews were completed with 10 experienced mental health and neurofeedback professionals identified through a nomination process with a snowball sampling method. For this study a wide definition of trauma was used that included traumatic brain injury, the DSM-IV-TR (American Psychiatric Association, 2000) criteria for posttraumatic stress disorder, and the seven symptoms associated with complex trauma (Courtois, 2008; Courtois & Ford, 2009; Herman, 1992, 1997). Research results indicate that the neurofeedback practitioner is central to the treatment process, that practitioner therapeutic skills are crucial to positive neurofeedback outcomes, and that counseling and neurofeedback may effectively complement each other in trauma treatment.

Keywords: trauma; neurofeedback; grounded theory

Background

Neuroscience, the mental health field, and the concept of trauma as an underlying factor in mental and physical disorders have been inextricably linked since the inception of the mental health professions in the late 1800s, when Sigmund Freud and other physicians from the new professions of neurology and psychiatry studied hysteria. During World War I combat neurosis or shell shock was identified. In 1980, after the Vietnam War, the impact of trauma was officially recognized when posttraumatic stress disorder (PTSD) was included in the Diagnostic and Statistical Manual of Mental Disorders, III (DSM-III, American Psychiatric

Association, 1980). Since the mid-1970s, the feminist movement has encouraged recognition of the adverse effects of interpersonal trauma (Herman, 1997), described in the construct of complex trauma, or CPTSD (Courtois, 2008; Herman, 1997).

On July 1, 2009, the Council for Accreditation of Counseling and Related Educational Programs (CACREP) officially recognized the importance of trauma when it mandated that disaster response, crisis intervention, and trauma knowledge be integrated into nearly every aspect of the counseling curriculum (CACREP, 2009). With the emphasis that CACREP has placed on trauma in the curriculum, it seems reasonable to investigate a wide variety of trauma treatments. Neurofeedback, based on recent neuroscience research, is one of those treatments.

Through neuroimaging techniques, neuroscience research has indicated that trauma affects the manner in which the brain receives and processes information (van der Kolk, 2006). Traumatic experience during the early developmental years may have an especially deleterious impact on all domains of functioning (Perry, 2002; Rothschild, 2000). Poor emotional regulation due to overarousal of the limbic system often results in limited problem-solving skills, inadequate relational abilities, and somatic symptoms (Courtois, 2008; van der Kolk, 2003).

With foundations in neuroscience, neurofeedback is based on the brain's plasticity that allows it to modify brain wave patterns in response to environmental changes (Charney, 2004). Neurofeedback is biofeedback applied to the brain and the central nervous system (Hammond, 2006). Numerous quantitative studies have indicated that neurofeedback may be effective in ameliorating symptoms often rooted in traumatic experience such as depression and anxiety (Hammond, 2005), substance abuse (Kelley, 1997; Peniston & Kulkosky, 1999; Scott, Kaiser, Othmer & Sideroff, 2005), posttraumatic stress disorder (Peniston & Kulkosky, 1991; Smith, 2008), and somatic symptoms such as migraine headaches (Stokes & Lappin, 2010) and fibromyalgia (Mueller, Donaldson, Nelson, & Layman, 2001). However, there is a paucity of research exploring the factors that produce those positive outcomes. The purpose of this grounded theory study was to explore the factors and processes that influence outcomes when neurofeedback practitioners treat trauma survivors.

Method

Since the purpose of qualitative research is to discover rather than to test variables (Corbin & Strauss, 2008), qualitative studies are appropriate when factors for quantitative research have not been fully identified. Grounded theory is a qualitative approach in which the researcher develops theory from fieldwork by exploring interacting concepts in a complex phenomenon. To be cohesive, one concept may be more prominent than the others as concepts interact in repeating action/interaction/emotional response patterns (Corbin & Strauss, 2008).

Participants

Neurofeedback practitioners considered to be effective in treating trauma survivors were identified through a nomination process with a snowball sampling method. Participants consisted of seven women and three men whose ages ranged from the mid-50s to late 70s. All were Caucasian. All were actively engaged in private practice in six different states representing four different regions of the United States, including large and medium

metropolitan areas, a small college town, and rural towns of 3,000 to 4,000 people. The study included 3 participants who operate an integrated practice in which they divide their time more or less equally between neurofeedback and counseling services and 7 participants who offer primarily neurofeedback in their practices. Participants included licensed counselors, social workers, psychologists, and one practitioner certified in neurofeedback by the Biofeedback Certification International Alliance (BCIA).

Participants' years of experience in psychotherapy practice ranged from 14 to 40 years, with 25 years being the median. Experience in neurofeedback practice ranged from 4 to 21 years, with 15 years being the median. The number of clients seen per week ranged from 8 to 10, to more than 100, with 25 or more being the median. Between 25% and 95% of their clients have some form of trauma as an underlying issue, with 65% being the median.

Data collection took place in three rounds over a 9-month time frame. The first round was a face-to-face semi-structured interview, with one exception due to geographical limitations. The second round was a semi-structured telephone interview, and the third round was an email follow-up. To preserve confidentiality and anonymity, participants were given a pseudonym.

Data Analysis

The research team consisted of six students or recent graduates of the counseling doctoral program at Old Dominion University. All had completed training in qualitative research. Using a constant comparative method (Creswell, 2007), the research team analyzed the data and met on a regular basis to reach consensus on themes. The lead researcher transcribed the majority of the interviews. The initial open-coding process entailed analyzing individual participant's words or phrases, followed by axial coding in which themes were categorized across participants. Finally, using a selective coding process, the practitioner was identified as the prominent theme around which the other themes were organized (Corbin & Strauss, 2008). The four other themes included: (a) the therapeutic process, (b) the neurofeedback process, (c) client factors, and (d) external factors. In order to triangulate the data (Patton, 2002), participants were given the opportunity to review their transcripts, read the analysis results, and provide feedback.

Although Creswell (2007) recommends bracketing assumptions, others hold that bracketing is impossible since assumptions and biases often have deep cultural roots difficult to identify (Corbin & Strauss, 2008). Rather than bracket assumptions, I sought to use my experience to enhance the data gathering and analytic process by acknowledging my assumptions in an initial concept map (Strauss & Corbin, 2008) that I shared with my research team before data collection began. I utilized a reflexive journal and regularly met with my research team and research partner to debrief, to reach consensus, and to modify the concept map. The Final Concept Map that was developed from the interviews is depicted in Figure 1.

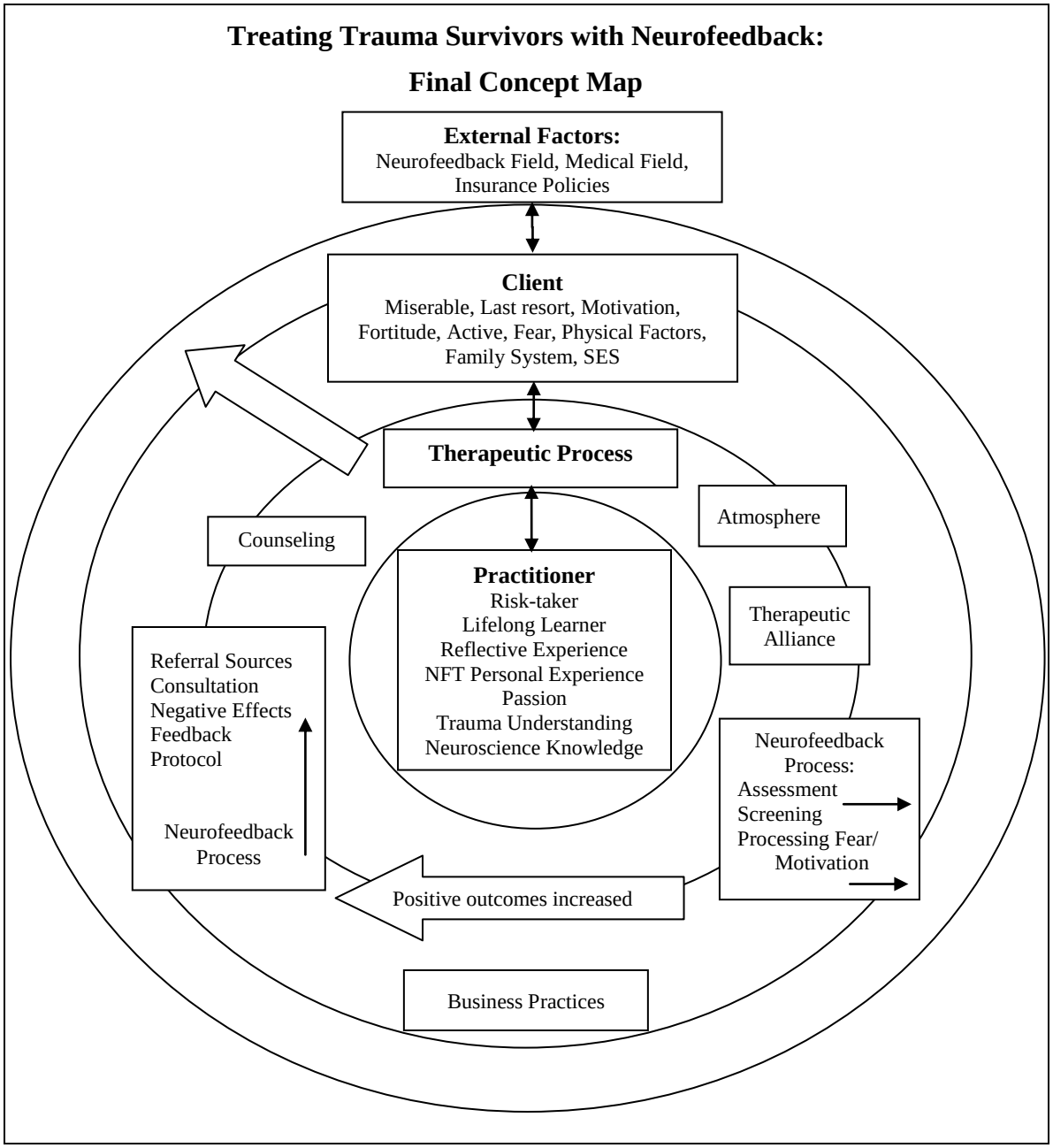

Figure 1. Final Concept Map. This was developed from the prominent themes that emerged from the three rounds of neurofeedback practitioner interviews. Research results indicate that the practitioner is central to the treatment process. Other themes include the therapeutic process, the neurofeedback process, client factors, and external factors.

Trustworthiness

Important criteria for determining the rigor of a qualitative study include the amount of researcher time spent in the field as well as the amount of data from which the researcher draws conclusions (Creswell, 2007). The data for this study consisted of 31 hours of

recorded interviews in addition to a written email follow-up. I spent approximately 40 additional hours in the field with participants in informal ways. In addition, I attended a 4-day neurofeedback training before data collection began and a 1-day conference during the study. The external auditor for this study examined the audit trail, which consisted of 1,290 pages of materials.

Results

The central research question for this study was, "What are the factors and processes that influence treatment outcomes when neurofeedback is used to treat individuals with trauma symptoms?" Five major themes, each with subthemes, emerged from the data. The central theme is the practitioner.

Theme 1: The Practitioner: "The Magic is Not in the Box"

The practitioners in this study described themselves as being "open" to new ideas and experiences, and as frequently thinking "out of the box." They entered the mental health field because they are people-oriented and relational, but then made the choice to tackle technological challenges that they describe as being "daunting," "a huge leap," "a steep/huge/enormous learning curve," and a "step of faith." They described "sweating blood" and being "scared to death" when they incorporated neurofeedback into their practice. Gaylen mortgaged her house to purchase her first neurofeedback set. All were willing to take the "leap," to undergo a "huge paradigm shift" because what they saw before them was so "dramatic" and "compelling" that they could not "walk away." They are risk-takers who embrace new challenges.

Subtheme 1.1: Risk-takers. The risk-taking continues throughout the neurofeedback process. Practitioners stated that neurofeedback could be "scary" for both practitioner and client, as negative effects may happen unexpectedly. Trauma survivors may feel more "vulnerable" as they lose their "protective armor" or as flashbacks of traumatic events suddenly occur. Barbara described the strong inner fears of the therapist in the face of uncertain treatment results:

> I have to always stay calm with clients … and if there is a problem to always reassure your client that everything's going to be fine because there's that placebo effect that takes place, even if you're scared to death inside and wondering, "Oh no, what did I do and how am I going to fix this?" (Barbara)

Marian routinely expects minor side effects that indicate that neurofeedback results are not merely from a placebo effect.

> A lot of people say this tiny signal can't be having an effect so it has to be a placebo effect, but I'm convinced it's not because the most common reaction … you will have a headache or feel really tired or re-experience the pain at the time of trauma with the first treatment or two … so negative things happen … if they have ever had a concussion, they are likely to have a headache … because there is a recall of the somatic experience.

Marian emphasized that clinicians must be "unflappable" in working with trauma survivors.

Subtheme 1.2: Lifelong learners: "Always looking." These expert practitioners seem to possess an inner drive to surpass mere competence in order to be the best that they can be in their unique settings.

> I think experts are always looking … the people that I think of as the best [emphasis] … are always looking for new approaches … they are talking to colleagues … when they get stuck, they are talking … they are finding other people who are at their level of experience or that they trust. (Gaylen)

As a result of "always looking," these practitioners have accumulated a "big bag of tools" (Sarah) and a "whole array in my armament to help people" (Julia) that may include play therapy, Eye Movement Desensitization and Reprocessing (EMDR), Dialectical Behavior Therapy (DBT), psychodrama, guided imagery, or hypnosis. Some utilize biofeedback along with neurofeedback to access the autonomic nervous system as well as the central nervous system. They often utilize multiple types of equipment. No matter what skills they already possess and what equipment they currently use, they are continually striving to improve.

> What I really want to do is keep learning and stay on the cutting edge … I don't want to coast … I want to always be one of the best … If I'm offering something to my clients, I want to be able to offer them the best that's out there … I'm always a little behind because there's always a learning curve … but I … keep working and keep learning. (Barbara)

Staying on the "cutting edge" in order to offer clients "the best that's out there" requires dedication to continuing education. Most of the participants cited ongoing training as one of the most important factors for positive client outcomes. Julia stopped counting her continuing education units after she surpassed 1,000 post-doctorate hours. Participants attend national conferences and local trainings, participate in email list serves, and consult regularly with colleagues. Their conversations are interspersed with references to seminars, books, and professional journal articles. Most have had at least one strong mentor. In addition, most participants have published in professional journals. Some have researched their own practices in order to assess their effectiveness. All these activities result in consistent professional growth:

> I'm a better clinician than I was a year ago—and I'm certainly a heck of a lot better than I was 10 years ago—but every year… I am better than the year before. (Barbara)

In addition to investing in themselves, the participants invest in others and in the field as a whole. They serve on the boards of their professional associations, organize local trainings for other professionals, and present at conferences. They mentor newer colleagues both formally and informally by taking on interns in their practices and through informal consultation.

Subtheme 1.3: Reflective experience. Participants counted their years of experience as being vastly important in client outcomes. When asked how much experience matters, most simply answered "a lot." Experience gives "more information to draw from … it just pops right out when you need it" (Shirley). Experience helps practitioners to "recognize phenotypes" (Julia) and to handle abreactions. It encourages practitioners to keep searching for new protocols as they realize the limits of what they currently know. In addition, experience is not measured only in years, but in reflection on their varied experiences.

Practitioners discussed their disappointments and mistakes as well as their successes, and what they learned from both.

Subtheme 1.4: Neurofeedback personal experience. Six practitioners in this study experienced dramatic physical and emotional changes from neurofeedback training. Others were convinced through seeing rapid changes in clients or family members.

Gaylen describes herself as a "poster child for everything neurofeedback can address." She had PTSD and a traumatic brain injury (TBI) as a result of childhood trauma until neurofeedback training eliminated her seizures, migraine headaches, and all PTSD symptoms, including her life-long startle response. Julia had spent "tens of thousands of dollars" seeking a cure from chronic fatigue syndrome and envisioned founding a "healing center" if she could ever recover. After neurofeedback she founded and manages one of the largest clinics in the country. Marian had struggled her entire life with memory and learning problems stemming from a childhood TBI. After neurofeedback training, "it all changed … it changed my life ... I couldn't be doing this [the research] if I didn't have that treatment." She says, "Nothing else does this." After Helen witnessed "profound" mood changes in her son due to neurofeedback training, she also began treatment and was cured of chronic fatigue syndrome. She currently manages a clinic and travels nationally and internationally to train others. David was cured of "30 years of back pain" in a neurofeedback session. Barbara "made more progress in 3 months of neurofeedback than in 20 years of counseling" for anxiety and panic disorder.

Subtheme 1.5: Passion. Risk-taking, lifelong learning with motivation to better serve clients, combined with their own personal experiences of neurofeedback, produces a passion for the field and a vision for its future.

> Just like everybody who gets into this field now, it's like, "Oh my God, have you thought of all the people you could be helping?" Let's get busy; let's make this happen in the world. (Helen)

And these practitioners are making neurofeedback "happen in the world" in creative ways. Sarah has a plan to attract interns to her underserved area. Gaylen has trained mental health professionals to use neurofeedback with abused children in a children's clinic. Charles carries portable neurofeedback equipment to clients' homes if they are housebound. Marian recruits veterans for TBI and PTSD studies. Helen's practice sponsored a research study at a residential treatment facility for drug and alcohol addicted individuals. David treats military veterans free of charge, saying, "Money doesn't stand in the way around here." Several mentioned at times working either *pro bono* or on a sliding scale. Helen stated, "It's not work; it's a passion."

Subtheme 1.6: Trauma understanding. Participants agreed that trauma is a common experience that takes many forms, including PTSD, complex trauma, and TBI. However, clients may not realize that the underlying source of their symptoms is trauma. David noted that "half or less" of his clients come to therapy for the actual underlying problem, "So we have to figure it out." Practitioners emphasized going slowly with trauma survivors due to potential abreactions and that trauma survivors need counseling in addition to neurofeedback.

Complex trauma, often rooted in early childhood abuse or neglect, causes disruptions in "every system of cognitive, learning, behavioral, emotional, and physiological [domains]."

With trauma survivors Gaylen focuses on “always to be quieting fear.” Julia described complex trauma as having been “marinated in fear,” resulting in a “body clench” as if a person has been “holding onto something” for a long time. If a client has many somatic complaints, including migraine headaches or chronic upsets such as irritable bowel syndrome, or emotionally seems “paralyzed in life,” then Julia suspects trauma as an underlying factor. Helen described complex trauma survivors as having “no place of safety” within themselves, such that they feel overwhelmed and constantly threatened. Addictions and other self-destructive behaviors may result from the feeling of low self-worth that often accompanies trauma. Sarah stated that underlying trauma issues in children are often misdiagnosed as AD/HD or some other disorder. Childhood trauma causes a “coping deficit” because the prefrontal cortex cannot develop when a child is continually in a “survival mode.” Complex trauma can take many forms.

Marian’s definition of trauma includes all degrees of TBI, including Cesarean sections and other “traumas that aren’t usually thought of as traumas, but they’re a trauma for the brain.” She assesses carefully for any type of head injury because even mild head injuries can dramatically alter emotional responses and thinking processes.

Subtheme 1.7: Neuroscience knowledge. Participants discussed neurofeedback treatment protocols in terms of neuroscience knowledge**.** Sarah discussed the inability of the prefrontal cortex to develop normally in a child exposed to ongoing trauma. Gaylen described how “the brain becomes mind,” meaning that personality is formed through the underlying firing of brain neurons. She does not utilize cathartic therapies due to the maxim “what fires together wires together.” Discussing traumatic events before the brain is calmed down only serves to strengthen the neuronal pathways in an anxious, fear-based neurological system. Marian described the “neurochemistry of trauma” as the brain being “stuck,” being “frozen in a protective state” from “extreme fear.”

Fear may cause the brain to be stuck, but neurofeedback can help it get unstuck by modifying brain-firing patterns. Marian emphasized that neuroplasticity, defined as “the brain’s ability to change,” is a practitioner’s “biggest ally” in neurofeedback treatment:

> One thing I learned when I was working with hospice and cancer patients who were dying … is that there is something in every cell that keeps trying to fix itself, and the brain is just sitting there waiting for us to find the right key … neuroplasticity is the thing.

Important factors in successful treatment involve the neuroplasticity of the client’s brain coupled with the practitioner’s ability to find “the right key” based on neuroscience knowledge.

Theme 2: Therapeutic Process

The therapeutic process begins with the office atmosphere, continues with the formation of the therapeutic alliance, and builds as the therapist responds to client feedback during every session. Neurofeedback is a major piece, but actually only one piece of this holistic process.

Subtheme 2.1: Atmosphere. The therapeutic process begins the moment a client enters the door. Barbara deliberately sets up a “friendly kind of atmosphere.” David spoke of the importance of a “loving atmosphere,” a “sense of love and God’s presence” that “sets up the possibility for healing.” Sarah’s office, located in a renovated church with stained glass

windows, sets up a “safe, welcoming” environment that is a “huge factor” in therapeutic outcomes. The walls of Julia’s waiting room are decorated with enlarged newspaper articles of neurofeedback success stories. A notebook filled with research articles and more success stories sits prominently on a table in the center of the room. I found myself wanting to be a client as I read these materials, and I wondered what degree of hope and confidence might be instilled in clients even before the actual neurofeedback process begins.

Subtheme 2.2: Therapeutic alliance. The office atmosphere provides the prelude to the formation of the therapeutic alliance, which grows over time and is particularly fragile with trauma survivors (Gaylen). In his 40 years of counseling experience, David’s most important lesson has been the centrality of empathy, an ability to “hear” clients, and to “feel a sense of what they feel.” The practitioner may be like a “coach” or a “support person” (Helen). “Trust” within a “nurturing relationship” is important (Barbara). A caring relationship is crucial because “anecdotally we learned that people are not used to being treated with care” (Sarah). Having military clients, Marian sat through the movie *Hurt Locker* even when she wanted to walk out, thinking, “These guys live [emphasis] this so surely I can watch a movie.” It is important to “be there” as a strong, stable, and calm support for trauma survivors (Barbara). Even in the midst of complicated technological equipment, David summed up the importance of the relationship in the neurofeedback process by saying, “I am not a technician … I am still a therapist.”

Subtheme 2.3: Business practices. Business practices constitute the foundation on which practitioners build the therapeutic process with clients. Marian observed that one prominent organization, now out of business due to poor decisions, no longer exists to train people. Practitioners discussed careful scheduling to “stay afloat” and equipment prices and sales. Julia’s marketing expertise shows in her ability to attract attention from the press. Helen markets her practice by offering a free first appointment in which she familiarizes people with neurofeedback. Without a savvy business sense, practitioners could not be helping anyone.

Theme 3: Neurofeedback Process: **“We’re Not Fixing Brain Waves”**

Neurofeedback is more than “fixing brain waves” (Helen). It sets in motion a personality “transformational process” as the practitioner works at the intersection of the brain and the mind.

> The most important lesson that I’ve gotten has been that the core of all of our psychological problems rests in the firing of the brain in some way, … that all mental processes sit on top of this and that we have access to it…. In neurofeedback you see an evolution of … the mind as the brain regulates itself. (Gaylen)

Subtheme 3.1: Thorough assessment: “Detective agency,” “Outside the box.” This “transformational process” starts with a thorough assessment and screening. Marian likened the assessment to being a “detective agency” in which practitioners look “outside the box” for the root problem. Marian screens for head injuries, Lyme disease and other parasitic infections, sometimes the root of psychological and emotional problems. She assesses for trauma by asking, “What’s the worst thing that has ever happened to you?” Clients may be sent for nutritional counsel or allergy testing, and may be told to stop ingesting aspartame, a neurological toxin.

Subtheme 3.2: Screening: “Don’t waste your money with me!” Either in conjunction with the assessment or separately, many practitioners utilize some type of screening process.

Julia is very straightforward with clients in saying, “Don’t waste your money with me!” Clients are screened for chronic infections, some addictions, and possibly benzodiazepine medications that “take over the brain.” Practitioners may also assess client motivation. Through “good, careful informed consent,” Julia and Shirley stress to clients that neurofeedback is “not an instant fix.” David asks clients, “What do you have to give up?” in order to get better. Gaylen asks, “You can go through a wide range of changes. Are you ready for that?” Both Sarah and Gaylen highlight the importance of processing fear since “health is scary sometimes.”

Subtheme 3.3: Protocol: “Cast a wide net,” “Find the right key,” “Stabilize.” With trauma survivors, practitioners move cautiously to “calm down” and “stabilize.” Julia talked of “casting a wide net” and having “a whole array in my armament to help people.” Participants often utilize multiple techniques because “good therapists have always been very attuned to what people bring, and so they wouldn’t necessarily be wedded to one approach” (Marian). Practitioners “find the right key” in order to talk *to* the brain and not *at* the brain (Marian).

Subtheme 3.4: Constant feedback: “The brain and the mind.” Practitioners choose protocols as they follow client symptoms: “Start with the symptom or the behavior that is most obvious; that’s going to be the brain’s way of talking to you, the outsider” (Marian). Protocols are altered in response to specific client feedback every session. Helen describes this process as “a team effort” in which she tells clients, “You and I are going to have to work together here … I kind of know about neurofeedback, but do you know about you? So you’ve got to talk to me about what’s going on.” When receiving client feedback, the practitioner is having “two different conversations” simultaneously with both “the brain and the mind.” For example, if a client reports that he is calmer (the mind), but is having nightmares of killing people (the brain), the practitioner follows what the over-aroused brain is saying and institutes more calming protocols (Gaylen). Lastly, participants agreed that effective neurofeedback treatment involves art or tuition as well as science. “Intuitive sensing” (Charles) and a “sense of timing” (Sarah) help the practitioner to “apply the model in a sensitive way for the individual” (Helen).

Subtheme 3.5: Negative effects. Although potentially “scary” for both the client and the practitioner, negative reactions may indicate that neurofeedback results are not merely “placebo effect,” as some detractors claim. Negative effects can actually increase client hope in the process.

> Even when we move people in the wrong direction … even when we mess up their sleep or give them a headache or something … that gets people’s attention … because the biggest fear is really that this is a scam … you’re wasting my time. And when we do something strong, even if it’s the wrong thing, it’s like, “Ok, this actually works” because after all, what is people’s experience with medicines? They just as often mess you up as help you … this is strong stuff and if I get the right strong stuff this may help me. (Helen)

Subtheme 3.6: Consultation and referral sources: “I don’t know it all!” All participants emphasized the importance of consulting with colleagues. The statement, “I don’t know it all!” spoken by a practitioner with over 31 years of mental health experience and over 15 years of neurofeedback experience, exemplifies the importance placed on consultation.

Subtheme 3.7: Counseling: “Who am I now?” Counseling and neurofeedback complement each other. Some likened counseling trauma survivors without neurofeedback

to “doing surgery without anesthesia.” Gaylen will not counsel trauma survivors without neurofeedback as a calming component. Particularly with complex trauma survivors, the counseling process can be hindered by transference reactions of fear, rage, and shame, accompanied by therapist countertransference. Neurofeedback provides the “transference cure” by “quieting fear” in the limbic system so that the client can discuss trauma without disregulating the neurological system. Neurofeedback helps to protect the therapist from vicarious trauma.

Neurofeedback calms the trauma survivor’s anxious neurological system, and counseling assists in processing the changes that neurofeedback may effect in every domain. Since “whole identities may be built around affect,” as affect symptoms drop away, the question of identity comes to the forefront. Clients may begin to ask themselves, “Who am I? If I am not this terrified, raging, shame-based person, who am I?” (Gaylen). In neuroscience terms, neurofeedback calms the brain and makes it more flexible. Talking within the counseling relationship helps to move the traumatic experience from the more emotional right brain to the more linear, rational left brain, so that the client can be “done with” the trauma. The client goes back and forth between the emotion regulation that neurofeedback provides and the talking in the counseling relationship that assists the client in identity transformation as affect symptoms drop away.

Theme 4: Client Factors

Neurofeedback clients are generally “miserable,” have “tried everything else,” and then try neurofeedback as a “last resort.” In addition to nutrition, allergies, toxins, and parasitic infections, participants identified the following client factors as potentially affecting treatment outcomes.

Subtheme 4.1: Motivation. Client motivation, described by Sarah as, “You gotta [sic] have a want to,” is foundational. Even if motivated at the beginning of treatment, clients need “fortitude” to stay with the process sometimes over significant periods of time. Moreover, clients must be willing to process and overcome fear of health.

Subtheme 4.2: Family system. A psychologically toxic environment will likely interfere with treatment outcomes. If a client is a “designated patient” that is “holding the family together,” if the family does not want to “lose their scapegoat,” or if a client has an unstable or abusive home life, neurofeedback effects will likely be diminished.

Subtheme 4.3: Multicultural factors. All 10 participants maintained that, since neurofeedback is not as “culturally loaded” as traditional talk therapy, it tends to cross-racial and ethnic boundaries well. Participants specifically noted that “age is not a factor,” as they have successfully treated clients ranging from 3 months to 96 years. Although more women than men come for neurofeedback, both respond well. The one multicultural factor that does affect the process is socioeconomic status. A client must have sufficient financial resources to enter neurofeedback treatment initially and to stay engaged over time.

Theme 5: External Factors

External factors are defined as factors outside the therapist, the client, and the therapeutic process that practitioners identified as influencing neurofeedback treatment outcomes.

Subtheme 5.1: The neurofeedback field. Participants described the neurofeedback field as being "cutting edge," "growing exponentially," "up and coming," and having "endless possibilities." They were enthusiastic about research published in professional journals as well as recent publicity on radio broadcasts, the *New York Times*, and other public forums. Acceptance of neurofeedback as a viable treatment option may be reaching a "critical mass," particularly as people become concerned with medication side effects, especially in children.

All publicity is not positive, however. Since neurofeedback is a relatively new field whose professional identity is unclear, a credentialing process has not yet been standardized. As a result there are "a lot of quacks out there" who may bring bad publicity to the field at a time that it is struggling to earn its place in the mainstream. Both good and bad publicity potentially affect client confidence as well as neurofeedback credibility with medical professionals.

Subtheme 5.2: The medical field. The medical field was also viewed as exerting both a positive and negative influence on neurofeedback outcomes. On the positive side, psychiatrists and other medical professionals are beginning to attend trainings, refer clients, and coordinate treatment with neurofeedback providers. Increased acceptance by medical professionals moves neurofeedback out of the "fringe" and "experimental" realm to a more respected place in the mainstream. This acceptance may encourage clients to initiate and to stay with the process.

On the other hand, the medical field was also viewed as perpetuating the overall "culture of medication" that encourages people to "take a pill and be fixed." Clients with this mentality are less likely to invest the time and finances necessary for successful neurofeedback treatment. Those who do seek neurofeedback are sometimes discouraged by physicians who tell them that they are "wasting their money" on a "ridiculous" treatment that "won't do anything." Some clients resist physician negativity, some change physicians, but others drop out of treatment.

Subtheme 5.3: Insurance policies. Closely related to the medical field are insurance policies that sometimes prohibit reimbursement for neurofeedback services. Clients lacking the financial means to initiate or continue the process are excluded from its positive effects.

Discussion

Findings of this study indicate that the practitioner is central to neurofeedback treatment outcomes with trauma survivors. Other themes included the therapeutic process, the neurofeedback process, client factors, and external factors. A comparison of these findings with existent literature follows.

The therapist has been the focus of numerous studies, as research has indicated that therapist competence is crucial for successful results (Ronnestad & Skovholt, 2003). Jennings and Skovholt (1999) described cognitive, emotional, and relational (CER) characteristics of master therapists that constitute a three-legged stool of expertise. In the cognitive domain master therapists are voracious learners, use experience as a major resource, and value "cognitive complexity and the ambiguity of the human condition" (p. 6). In the emotional domain they are "self-aware, reflective, nondefensive, and open to feedback" as well as "mentally healthy and mature individuals" (p. 7) who take care of themselves. In the relational domain they value the importance of the therapeutic alliance and are comfortable with clients' intense emotions.

Consistent with the findings of Jennings and Skovholt (1999), in the cognitive domain participants in this study were lifelong learners "always looking" for better treatment options. They valued reflexive experience and embraced complexity in looking "outside the box" for root causes. In the emotional domain they were receptive both to client feedback and to colleague consultation. Many maintained their own mental health by engaging in neurofeedback training and other self-care activities. In the relational domain they valued the therapeutic alliance as an essential overarching piece of the neurofeedback process. They emphasized listening, caring for clients, processing fears and being "unflappable" in the face of client emotions. It would seem that effective neurofeedback practitioners, despite the addition of technological equipment to the therapeutic process, are master therapists as well as neurofeedback specialists.

Wounded healers are individuals whose personal experience of suffering is transformed into a constructive healing force for fellow sufferers. The theme of the wounded healer is found in literature across time and cultures, and in both the medical and mental health literature (Jackson, 2001). It surfaces in this study as 7 of the 10 participants alluded to their own personal journeys through various forms of emotional trauma or physical illness or both. Perhaps one participant's comment best describes the wounded healer: "We try it on ourselves first, and then what works, we pass along."

Asay and Lambert (1999) estimated that 15% of treatment outcome is due to expectancy or placebo effects, sometimes called the hope factor. Participants engender client hope through their office atmosphere, through staying calm in unexpected client reactions, and in generally "being there" for clients in a strong therapeutic relationship. Even negative effects may actually increase hope that the neurofeedback is working. David unabashedly stated, "Placebo works!"

Results of this study indicated that the therapeutic alliance is an essential ingredient to positive neurofeedback outcomes as it is crucial to successful psychotherapy (Horvath & Symonds, 1991; Sexton & Whiston, 1994). According to the common factors model, 30% of client improvement depends on the therapeutic alliance (Asay & Lambert, 1999). Although this study did not seek to quantify the amount of influence the therapeutic alliance exerts on treatment outcomes, participants were clear that the therapeutic relationship is important.

Findings in this study differed from the common factors model (Asay & Lambert, 1999) in several ways. According to Asay and Lambert (1999), only 15% of psychotherapy outcomes are derived from actual techniques, which are more or less interchangeable. In contrast to these findings, participants in this study emphasized the necessity of having a "big bag of tools" and "a whole array in my armament to help people." For the most part, participants viewed the ability to change neurofeedback treatment protocols as an essential ingredient in successful outcomes. In addition, findings of this study emphasize the therapist as being central to the process, in contrast to the Asay and Lambert (1999) model that de-emphasizes the therapist role. It may be that since neurofeedback therapy requires more technological knowledge, the therapist is more central to the neurofeedback process than to the psychotherapy process.

Research participants in this study concurred that client factors as well as therapist factors contribute to treatment outcomes. Asay and Lambert (1999) assert that as much as 40% of client improvement derives from extra-therapeutic factors related to the client. These client factors may include the nature of the problem, motivation, ability to relate, ego strength, and

family and social support. Participants in this study identified motivation, the family system, and the nature of the problem as potential client factors that contribute to treatment outcomes.

The research participants' perception that multicultural factors exert little or no influence on neurofeedback treatment outcomes, with the exception of socioeconomic status, is congruent with the neurofeedback literature. Although few specifically multicultural studies exist, one study has indicated that neurofeedback can be adjusted in a culturally sensitive manner with positive treatment outcomes in a Native American population (Kelley, 1997). Studies have also indicated successful neurofeedback results with groups composed of both males and females and those representing a range of ages (Scott et al., 2005). Drawing from the responses of the study participants as well as from neurofeedback research, it seems that neurofeedback may be a treatment option in a wide range of cultural situations. The challenge is creating a conduit for neurofeedback services to be brought to these diverse populations.

Limitations

Several limitations to the findings of this study should be noted. Since snowball sampling seeks information-rich informants rather than a representative participant sample, the findings of this study cannot be generalized beyond this group, and the voices of some highly regarded practitioners may have been excluded. Furthermore, the lack of diversity in the participant sample may have skewed themes in ways unknown at this time. Lastly, this article is a condensed version of a larger study. Due to its condensed form, participant profiles and other information that would have provided a fuller context for the themes could not be included.

Implications

The results of this research study have several implications for counselors and counselor educators. First, although none of the neurofeedback practitioners in this study advertise themselves as specializing in trauma, between 25% and 95% of their clients have some form of trauma as an underlying issue, with 65% being the median. With such a significant portion of clients being trauma survivors, it would seem that CACREP has made a strategic decision to improve counselor competence by incorporating trauma education into the counseling curriculum (CACREP, 2009).

Secondly, several practitioners highlighted the fact that clients may engage in psychotherapy for years with minimal improvement because the root cause of the difficulty is actually an undiagnosed head trauma or an underlying infection. Teaching on the impact of physical root causes of mental disorders, instruction on how to conduct a holistic assessment as well as on the importance of appropriate referral sources could be a beneficial component to mental health education.

Lastly, since findings from this study indicate that neurofeedback may be an effective complement to counseling with trauma survivors, it would be beneficial for counselors to be familiar with this treatment option, whether or not they decide to incorporate neurofeedback into their practices. Being knowledgeable about the manner in which a counselor and a neurofeedback practitioner can work together may hold significant potential for increasing positive client outcomes. Some counselor education programs have begun offering a track

that leads to biofeedback and neurofeedback certification by the Biofeedback Certification International Alliance (BCIA). More universities could consider offering this track.

Future Research

The findings of this study indicate that neurofeedback is an effective treatment modality for trauma symptoms and that the neurofeedback practitioner plays a central role in treatment outcomes. Future research may expand on the themes highlighted in this study.

Since the themes in this study were drawn exclusively from the practitioner's point of view, the central role of the practitioner may be overemphasized and the importance of client factors may be underemphasized. A future qualitative study from the trauma survivor's perspective might provide a more balanced view of the process.

One study participant suggested that more research could be completed in the neurofeedback field to specifically answer the question, "When neurofeedback doesn't work, why doesn't it work?" In addition, studies comparing treatment protocols for all the various types of trauma would add to the body of research already in existence.

One salient characteristic of the effective therapists in this study was the value that they placed on continuing education and "always looking" for better treatment options. Further research may provide more understanding of the development of master therapists and effective neurofeedback practitioners. Is there a way to more effectively inspire this motivation for excellence within our mental health training programs and beyond?

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APPENDIX

Demographics Inventory
Neurofeedback Provider Demographic Information

This form will be kept in a secure file by the researcher. Any information derived from it will be identified by a participant identification code only, in order to preserve your confidentiality.

I. General Information:

A. Name: _____
B. Name of practice: _____
C. Practice Address: _____
D. Phone number: _____ Email address: _____

II. Educational Background:

A. In what field is your degree? □ Counseling □ Psychology □ Social Work □ Psychiatry □ Other (please specify): _____
B. Highest degree completed: □ Masters □ Doctorate □ Other (please specify): _____
□ Degree in progress (please specify):_____
C. Please list any licenses, certifications, or other special training that you have, and when you received/completed them:
_____
_____

III. Professional Experience:

A. Number of years in neurofeedback practice: _____
B. With which organization were you trained? _____
C. In addition to neurofeedback, do you also provide counseling/therapy services in your practice? □ Yes □ No
D. If so, how many years have you been doing counseling/therapy? _____

IV. Client information:

A. Approximately how many clients have you seen **per week**, **during this past year**?
_____
B. If possible, could you please give an estimate of the percentage of your clients with the following presenting problems:

Depression _____% Anxiety_____% Autism spectrum_____%
Attention Deficit/Hyperactivity Disorder _____% Bipolar disorder_____%
Chronic fatigue/fibromyalgia_____% Sleep problems_____%
Headaches/migraines_____% Traumatic Brain Injury_____%
Other common presenting problems: _____

C. What is the estimated percentage of your clients in which some form of trauma is a central issue: _____%

[Including, but not limited to: Childhood abuse (physical, verbal, sexual), rape/sexual assault, domestic violence, combat experience, attachment traumas, physical injuries (accidents, etc.) to self or a loved one, etc.]

Please include any additional comments here, or on the back of this form, if needed:

Thank you very much for your participation in this research project!

Interview Protocol

The initial interview question will be broad and general, in order to allow potentially unanticipated themes to emerge. Sub-questions will be more specific. In addition, the interview protocol may be altered, if themes emerge that need further exploration, or if the participant discusses sub-questions in the initial question.

I. Initial procedures:
   A. Restate purpose of the study: You have been nominated by your peers as someone who is effective in providing neurofeedback services. I would like to explore your experiences and perceptions as a neurofeedback provider concerning the factors and processes that contribute to positive treatment outcomes when neurofeedback is used on individuals with trauma symptoms. Some form of traumatic experience is frequently an underlying factor in mental health issues such as depression, anxiety, substance abuse, self-destructive behaviors, relationship problems, and somatic symptoms. For this study, a wide definition of trauma will be used, which includes both the DSM-IV-TR criteria for posttraumatic stress disorder and the 7 symptoms associated with complex trauma (Courtois, 2008; Herman, 1992).
   B. Make sure forms are signed:
      - Consent to Record interview
      - Informed Consent
   C. Ask for (if not obtained already):
      - Completed Demographic Information Form
      - Resume/curriculum vitae (if available)
      - Brochures or materials which they typically give clients or use to describe their services; professional disclosure statement; articles, etc.

Interview Protocol

Follow up on the Demographic Information Form:

Is there any information that was not included on the Provider Demographic information form, that you would like to add?

Opening questions:

1. How did you first hear about neurofeedback?
   a. What were your first impressions of neurofeedback?
2. What made you decide to set up a practice using neurofeedback, or to incorporate neurofeedback into your existing practice?
   a. Is there anyone or anything in particular that influenced you or helped you?
3. What was it like when you first started using neurofeedback in your practice?
   a. How is your practice different now from when you started?
4. Tell me what a typical day looks like for you in your practice.
   a. Describe the typical clients who come to you for neurofeedback.

Central Interview Questions

You have been nominated by your peers as being effective in using neurofeedback to treat clients.

5. In your opinion, what **factors** have contributed to positive treatment outcomes when using neurofeedback *in general*? (factors: for example, personal, therapeutic, professional, therapeutic alliance, etc.)
   a. In your opinion, what **factors** have contributed to positive treatment outcomes in using neurofeedback on clients *with trauma symptoms*?
   b. Are there any factors that have been obstacles to successful treatment?

c. How might multicultural factors contribute to positive or negative treatment outcomes?
(for example, gender, race, ethnicity, age, sexual orientation)
d. In your opinion, what factors distinguish an expert neurofeedback provider from a good provider, especially in treating individuals with trauma symptoms?

6. In your opinion, what part does years of experience play in a provider's expertise?
7. Now I would like to know more about the **process** of providing neurofeedback to clients who have experienced some type of trauma.
   a. What types of symptoms have you noticed in clients who have experienced trauma?
   b. Now that you've talked about _____, are there any additional symptoms? (Show list of PTSD symptoms and complex trauma symptoms)
   c. Tell me about the process of treating those symptoms.
   d. I'm wondering what differences you notice in treating clients who have experienced trauma, and those who haven't?
   e. Are there any differences in your responses or feelings in treating these clients?
8. Now I'd like to ask you more about actual treatment. In general, how do you go about treating trauma symptoms?
   a. I would like to hear about your experience treating a client with trauma symptoms, when the client seemed to be helped significantly.
   b. How did you know the client was helped?
   c. Now I would like to hear about an experience treating a client with trauma symptoms, when the client was helped very little, or not at all.
   d. How did you know that the client was not helped?
   e. In your opinion, what factors or processes contributed to the difference in the outcomes between the two clients?

Follow-up questions (for telephone interview):

9. What have been the most important lessons that you have learned through your Years of practice?
   a. I would like to know about any critical incidents or circumstances that you have experienced, incidents that changed your views, or challenged you to do things a bit differently.
10. You've told me some factors, processes, and treatments that contribute to positive treatment outcomes. I'm wondering if you can narrow it down a bit. If you had to choose one thing that makes you successful in treating clients with trauma symptoms, what would it be?
11. In your opinion, how much of neurofeedback training is art versus science?
12. What have you noticed while treating clients with trauma symptoms, when neurofeedback is combined with counseling or some other treatment modality?
13. Tell me about your experiences using the alpha-theta protocol. (If not already discussed)

Personal reflection questions:

14. I'm wondering how you balance your professional practice with your personal life.
   a. Do you ever take your clients home with you? (not physically but mentally and emotionally)
15. After having these experiences, what advice would you give to someone who wants to begin using neurofeedback as a treatment modality, particularly with those who have experienced trauma?
16. I'd like to know what you think about the future of neurofeedback, both personally and professionally.
   a. Where do you see yourself in two years? Five years? Ten years?

b. How will you be different then, both personally and professionally, from now?

17. Talk about the future of neurofeedback.
    a. What are your thoughts about the future of neurofeedback?
    b. What are your hopes for the future of neurofeedback?

Email questions:

18. Is there anything else that you would like to tell me about your experiences with neurofeedback?
19. If you could do it all over again (in terms of your mental health/neurofeedback career) what would you do the same?
    a. What would you do differently?

NeuroRegulation

Neural Networks and Neurofeedback in Parkinson's Disease

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Abstract

Aberrant neural network synchrony in basal ganglia thalamocortical circuits has been implicated in the pathophysiology of Parkinson's disease. Manipulating these abnormal activation patterns may therefore offer a novel avenue for treating this disabling condition. Evidence suggests that network activity can be normalized with both dopaminergic drug treatment and deep brain stimulation (DBS), and protocols that directly target specific oscillatory patterns ("closed-loop DBS") are under development. Another potential avenue for the modulation of specific neural activation patterns is neurofeedback. This noninvasive technique entails providing a continuous update of one's neural activity so that volitional control of selected brain regions, networks, or rhythms can be learned. This could be accompanied by specific therapeutic changes in behavior and clinical symptomatology in disease, according to the neural circuits that are modulated. Most neurofeedback research has used electroencephalography (EEG) but recently neurovascular signals measured with functional magnetic resonance imaging (fMRI) have been targeted as well. In this paper, we discuss the evidence implicating certain rhythms, particularly the beta (10–35 Hz) oscillation, in Parkinson's disease. We also perform a systematic review evaluating the therapeutic efficacy of neurofeedback in Parkinson's disease and make suggestions for future research.

Keywords: Parkinson's disease; neurofeedback; electroencephalography; real-time functional magnetic resonance imaging; deep-brain stimulation

Introduction

The precise mechanisms in which the brain encodes, stores, and computes information has long been the subject of intense debate in modern neuroscience. A popular theorem, which has received great interest, invokes the role of neuronal network oscillations in information processing. Local field potential (LFP) oscillations and, on a more macroscopic scale, electroencephalography (EEG) and magnetoencephalography (MEG) recordings are the product of synchronous firing of populations of neurons. Such oscillations represent rhythmic fluctuations in membrane potential and extracellular currents, and reflect the complex

interplay between cellular and synaptic mechanisms (Buzsáki, 2006; Logothetis, 2003). Rather than representing mere electrophysiological epiphenomena, network oscillations actively sculpt and coordinate neuronal activity patterns, facilitating transmission, communication and consolidation of information streams (Buzsáki, 2002, 2010; Buzsáki & Draguhn, 2004; Buzsáki & Wang, 2012; Fries, 2005; Wang, 2010).

The level of oscillatory synchronization on a spatial and temporal scale determines the precise frequency and amplitude of these oscillations (Von Stein & Sarnthein, 2000), with different frequency bandwidths correlating with specific behavioral, cognitive, perceptual, and sensorimotor functions (Brown, 2007; Buzsáki, 2006; Crunelli & Hughes, 2010; Laurent, 2002; Steriade, 2003; Varela, Lachaux, Rodriguez, & Martinerie, 2001). Although aberrant network activity spanning most frequency bandwidths has been intertwined with a variety of disease states (Basar, Basar-Eroglu, Guntekin, & Yener, 2013; Bragin, Engel, & Staba, 2010; Traub 2003; Uhlhaas & Singer, 2010), in the context of motor functioning and disorders much research has placed particular focus on the beta (10–35 Hz) rhythm (Brown, 2007; Davis, Tomlinson, & Morgan, 2012; Engel & Fries, 2010; Stein & Bar-Gad, 2013).

In this article, we first discuss the canonical model of the basal ganglia circuitry and its dysfunction in Parkinson's disease (PD), while addressing its limitations. We then discuss the impact of oscillatory network dynamics and its pathological nature in PD with a particular emphasis on the role of beta (10–35 Hz) oscillations. The electrophysiological impact of rhythms within other frequency ranges will also briefly be discussed. Finally, we perform a systematic review highlighting empirical data that has investigated the role of neurofeedback, a novel method for modulating brain activity, in PD, with corresponding suggestions for future research.

Putative Mechanisms Underpinning PD

PD is a chronic neurodegenerative disorder characterized by progressive loss of dopaminergic neurons in the substantia nigra pars compacta (SNpc) of the midbrain. This manifests clinically as a combination of bradykinesia or akinesia, muscle rigidity, postural instability, and a resting pill-rolling tremor (4–6Hz; Jankovic, 2008). The precise neural mechanisms underpinning this clinical phenotype remain unclear, but certain theories have been proposed.

According to the canonical model (Albin, Young, & Penney, 1989; DeLong, 1990), an imbalance in activity between the direct and indirect neural pathways resulting from striatal dopaminergic denervation is responsible for generating bradykinesia and rigidity. SNpc neurons activate the direct, and inhibit the indirect, pathway via excitatory D1 and inhibitory D2-receptors, respectively, on segregated classes of medium spiny neurons (MSN), which form the sole output of the striatum (Surmeier, Ding, Day, Wang, & Shen, 2007). MSNs send inhibitory projections to the globus pallidus pars interna (GPi) and globus pallidus pars externa (GPe). GPi, in combination with substantia nigra pars reticulata (SNpr), which are both functionally equivalent, represent the only output nuclei of the basal ganglia and send tonic inhibitory projections to the thalamus. The thalamus sends excitatory signals to the neocortex, which completes the basal ganglia thalamocortical loop via dense glutamatergic afferent innervation of the striatum.

Activation of the direct pathway results in inhibition of GPi/SNpr by D1-MSN striatal neurons, thereby disinhibiting excitatory thalamic output to the motor areas of the neocortex, which facilitates locomotor behavior. Activation of the indirect pathway, however, leads to D2-MSN

striatal neuron-driven inhibition of GPe, which causes disinhibition of subthalamic nucleus (STN; as GPe sends GABAergic inhibitory output to STN). The disinhibited STN, which also receives corticofugal excitatory afferents via a so-called “hyper-direct pathway,” is subsequently free to excite GPi/SNpr-mediated inhibition of the thalamus, and consequently reduce activity in the neocortex—this putatively leads to a reduction in locomotion. Ultimately, as a consequence of SNpc degeneration in PD, there is reduced activation of the direct pathway and reduced inhibition of the indirect pathway, which collaboratively provides a mechanistic explanation for the difficulty in initiation of novel movements in PD (see Figure 1).

a.

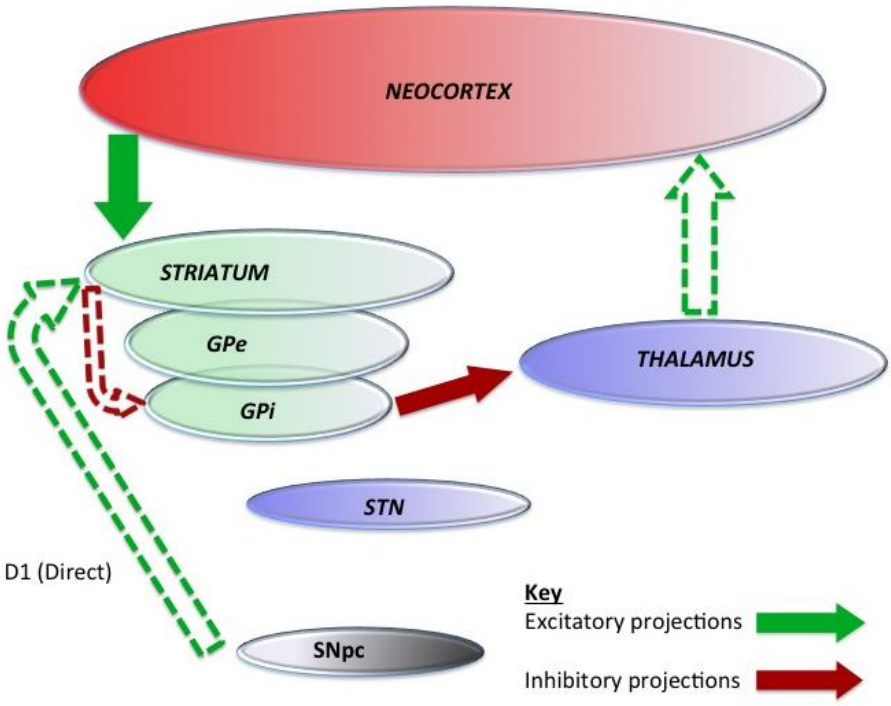

Figure 1. Schematic representations of the canonical basal ganglia circuits in PD. (a) Reduced activation of the striatum in the direct pathway following degeneration of SNpc putatively results in reduced thalamic-driven cortical excitation and subsequent compromised locomotor output.

b.

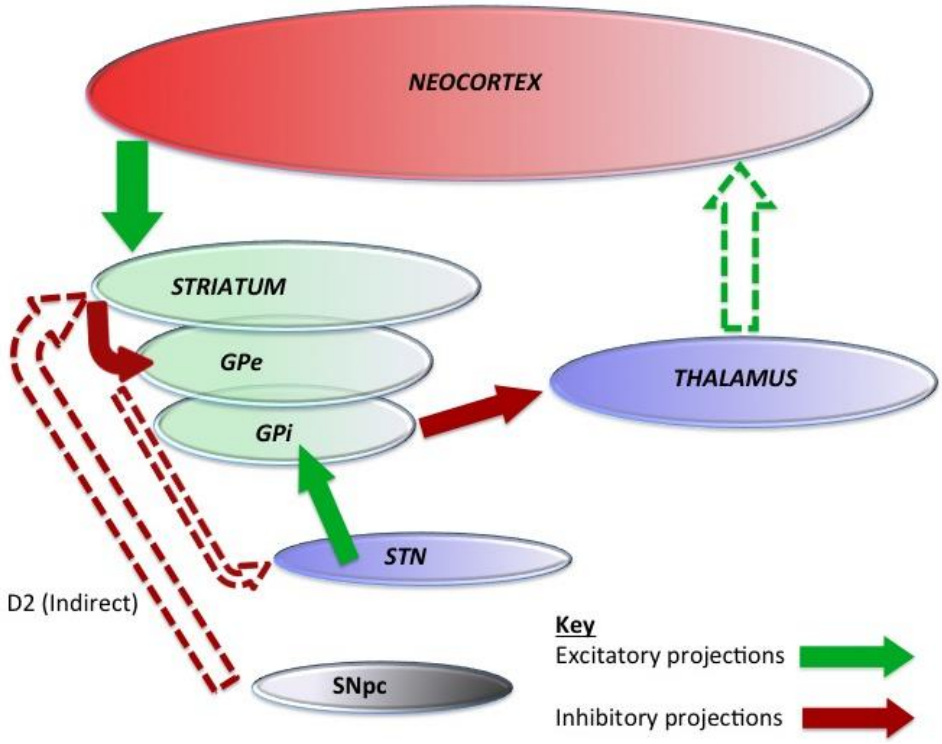

Figure 1. Schematic representations of the canonical basal ganglia circuits in PD. (b) Reduced inhibition of the striatum in the indirect pathway ultimately leads to the same outcome as that of reduced excitation of the direct pathway. Dotted arrows represent reduced activity in the corresponding projections.

The antagonistic functional effects of the direct and indirect pathways have been elegantly illustrated in optogenetic experiments, which have aimed to directly modulate basal ganglia circuits *in vivo* (Gradinaru, Mogri, Thompson, Henderson, & Deisseroth, 2009; Kravitz et al., 2010; Tye & Deisseroth 2012). For example, increasing the activation of the direct pathway in transgenic mouse lines, made to selectively express light-activated ion channels in D1-MSN neurons, has been shown to rescue Parkinsonian symptoms in 6-hydroxydopamine-lesioned mouse models of PD (Kravitz et al., 2010).

However, the canonical model of PD entails an oversimplification. Although it explains the origin of bradykinesia and rigidity in PD, it fails to explain the remaining clinical manifestations, such as resting tremor. It also makes a significant, and possibly unjustified, assumption that firing rate of neurons is the predominant means by which the brain encodes and transfers information from one region to another (Buzsáki, 2006, 2010). Although there has been some empirical support for the firing rate model of PD (Remple et al., 2011; Steigerwald et al., 2008), conflicting findings have emerged from the literature. For example, single unit recordings have revealed that the average firing rate of GPi neurons, in primates rendered Parkinsonian using 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP), remain largely unchanged, although the canonical model predicts an increase (Tachibana, Iwamuro, Kita, Takada, & Nambu, 2011). During high frequency stimulation of the STN, which is known to improve Parkinsonism (Benazzouz, Gross, Feger, Boraud, & Bioulac, 1993), GPi neurons fail to show the expected decrease in firing rate (Hashimoto, Elder, Okun, Patrick, & Vitek, 2003; Moran, Stein, Tischler, Belelovsky, & Bar-Gad, 2011). Furthermore, functional

neuroimaging and noninvasive brain stimulation studies in PD patients have collectively shown that the activation or level of excitability of the motor cortex is not necessarily reduced in PD (Haslinger et al., 2001; Kleine, Praamstra, Stegeman, & Zwarts 2001; Ridding, Inzelberg, & Rothwell, 1995; Sabatini et al., 2000), which again contradicts predictions from the canonical model of PD.

A Physiological Role for Beta Oscillations

Beta oscillations are believed to play a physiological role in coordinating motor function. They are pronounced during steady state, tonic contractions, diminish immediately prior to and during the execution of movements, and rebound after movement (Alegre et al., 2005; Courtemanche, Fujii, & Graybiel, 2003; Hutchison et al., 2004; Kuhn et al., 2004; Williams et al., 2005). Although originally labeled as an “idling rhythm,” increasing evidence has implicated beta in playing a more active role in maintaining the motor set and resisting the enactment and expression of volitional movements (Engel & Fries, 2010). This has been supported by studies, which have shown that stopping or omitting unwanted behaviors was associated with greater synchronization and coherence of oscillations in the beta frequency band (Gilbertson et al., 2005; Swann et al., 2009; Van Wijk, Daffertshofer, Roach, & Praamstra, 2009). Noninvasive brain stimulation techniques have provided further insight into the active role of the beta rhythms in motor control. The use of transcranial alternating current stimulation (tACS) of the motor cortex to entrain beta oscillatory activity has been shown to reduce motor output and the magnitude of force contraction (Joundi, Jenkinson, Brittain, Aziz, & Brown, 2012; Pogosyan, Gaynor, Eusebio, & Brown, 2009). Furthermore, theta burst stimulation (TBS), a particular type of repetitive transcranial magnetic stimulation (rTMS) protocol designed to inhibit neuronal activity, directed onto primary motor cortex of healthy volunteers, has been associated with significantly increased spontaneous beta oscillations. Importantly, this electrophysiological response only occurred in individuals who experienced reduced corticospinal excitability in the relevant M1 region (McAllister et al., 2013).

Insight has also been provided from studies in PD patients undergoing functional neurosurgery for deep brain stimulation (DBS). Within the STN, beta oscillations are largely detected in the dorsolateral region, which is known to topographically represent the motor area (Monakow, Akert, & Kunzle, 1978; Nambu, Takada, Inase,, & Tokuno, 1996; Romanelli, Esposito, Schaal, & Heit, 2005). High frequency stimulation of the dorsolateral STN provides the optimal therapeutic outcome in PD patients, providing indirect evidence for the role of beta in motor processing (Herzog et al., 2004; Maks, Butson, Walter, Vitek, & McIntyre, 2009). DBS of STN in PD patients, which drove increased beta power in the frontal cortex, was associated with improved stopping performance in a behavioral stop signal task (Swann et al., 2011). DBS of STN at beta frequencies can also lead to worsening of bradykinesia in PD patients (Eusebio et al., 2008; Fogelson et al., 2005).

Thus, beta band synchronization is believed to represent an immutability-promoting rhythm, which locks neural circuitry into a low entropy state that maintains the status quo (Brittain & Brown, 2014; Engel & Fries, 2010). Higher oscillatory synchronization in the beta bandwidth engages large neuronal networks, which may span distant brain regions, into stereotyped, predictable spatiotemporal firing patterns. This is hypothesized to lower neural processing capacity, since such ensembles are prevented from operating in relatively more segregated information processing streams resonating at higher frequencies (i.e., gamma), which would otherwise signal more complex, novel behavioral and higher cognitive functions (Bergman et al., 1998; Brittain, Sharott, & Brown, 2014; Nini, Feingold, Slovin, & Bergman, 1995).

Although beta oscillations may display a physiological role in motor processing, and in particular facilitate stopping behaviors (Engel & Fries, 2010; Jenkinson & Brown, 2011), excessive neural network synchrony within this bandwidth has been associated with, and even causally related to, the pathology of PD. Notably, excessive beta synchronization is thought partly to account for symptoms of bradykinesia and rigidity (Zaidel, Arkadir, Israel, & Bergman, 2009), while aberrant oscillations in the theta and gamma range, which we will discuss later, may explain tremor and treatment-related dyskinesia, respectively, although the evidence for the functional role of the latter two rhythms is less robust (Deuschl et al., 2000; Richter, Halje, & Petersson, 2013).

The Putative Pathological Role of Beta Activity in PD

An emerging theory in the PD literature is that striatal dopaminergic denervation leads to excessive beta oscillatory synchronization within the basal ganglia thalamocortical circuits (Hammond, Bergman, & Brown, 2007). Whether this oscillation plays a causal role in driving the pathophysiological manifestations of the disease process, or whether it is merely an epiphenomenon has been the subject of much debate, but increasing evidence is beginning to favor the former scenario. Evidence for the role of beta oscillations in PD have largely stemmed from animal models and from electrophysiological recordings via electrodes in PD patients undergoing functional neurosurgery for DBS.

Nonhuman primates can be rendered Parkinsonian following injection of MPTP as this leads to preferential degeneration of dopaminergic neurons, including the nigro-striatal projections (Pifl, Schingnitz, & Hornykiewicz, 1991; Porras, Li, & Bezard, 2012). Recordings of single-unit activity in STN, GPe, GPi, striatum and motor cortex have revealed significantly increased rhythmic bursting activity following MPTP administration (Boraud, Bezard, Guehl, Bioulac, & Gross, 1998; Goldberg, Rokni, Boraud, Vaadia, & Bergman, 2004; Stein & Bar-Gad 2013). This is accompanied by an increase in correlated, spatiotemporally synchronized neuronal firing in distributed ensembles, which can manifest as an increase in amplitude of beta LFP oscillations within the cortex and basal ganglia (Bergman, Wichmann, Karmon, & DeLong, 1994; Goldberg et al., 2004; Heimer, Bar-Gad, Goldberg, & Bergman 2002; Jaidar et al., 2010; Moran, Stein, Tischler, & Bar-Gad, 2012; Stein & Bar-Gad 2013; Tachibana et al., 2011). Furthermore, recognized treatments for PD, such as STN stimulation and dopamine replacement, have been shown to reduce beta oscillations in the MPTP primate model (Gilmour et al., 2011; Moran et al., 2012).

Rat models of PD, involving selective lesioning of the nigrostriatal pathway following injection of 6-hydroxydopamine (6-OHDA), have provided further support for an association between pathologically elevated beta synchrony and PD. Unilateral hemispheric lesions of the nigrostriatal pathway resulted in increased beta oscillatory activity in SNpr, when compared with the control hemisphere, in combination with contralateral Parkinsonian symptoms (Avila et al., 2010; Delaville et al., 2014). Excessive beta oscillatory synchronization has also been shown to occur in STN-GP networks in 6-ODHA lesioned rat models (Magill, Bolam, & Bevan, 2001; Mallet et al., 2008a, 2008b).

A Dopamine transporter (DAT) knockout mouse has provided a valuable model for studying the neural mechanisms of PD. Costa et al. (2006) simultaneously recorded single-unit activity and LFP oscillations in primary motor cortex and dorsolateral striatum in DAT knockout mice. DAT knockout causes a 5-fold increase in extracellular dopamine levels and results in a hyperkinetic state in novel environments. Administration of the tyrosine hydroxylase inhibitor, alpha-methyl-p-tyrosine (AMPT), which led to rapid depletion of

dopamine levels (within 20 minutes) to < 0.2% of wild-type controls, resulted in significantly increased beta oscillation amplitude in motor cortex and striatum. In the dopamine-depleted state, neurons in these areas were also significantly more likely to be phase-locked to particular phases of the LFP oscillations, resulting in increased coordination of the corticostriatal neuronal ensemble. Importantly, administration of carbidopa/levodopa, which quickly restored dopamine levels back to baseline, reversed the electrophysiological changes and reduced beta oscillatory synchronization of the corticostriatal network. Interestingly, the overall firing rates of the motor cortical and striatal neurons remained unchanged following manipulation of dopamine levels, despite changes in observed behavior from hyperkinetic to bradykinetic states, which again provides evidence against the canonical model of rate-encoding in the basal ganglia circuitry.

Further evidence for the pathogenic role of beta oscillations in PD has emerged from the study of PD patients undergoing functional neurosurgery for DBS. LFP recordings in the STN tend to show prominent oscillatory activity within the beta band (Brown et al., 2001; Levy et al., 2002; Marsden, Limousin-Dowsey, Ashby, Pollak, & Brown, 2001), during which neuronal discharges in the basal ganglia are phase-locked (Kuhn et al., 2005; Weinberger et al., 2006). Importantly, treatment with levodopa, dopamine receptor agonists, or high frequency STN stimulation, attenuates such beta oscillations (Brown et al., 2001; Eusebio et al., 2011; Kuhn et al., 2008; Levy et al., 2002; Priori et al., 2004; Williams et al., 2002). Furthermore, the spatial extent of beta oscillatory activity in the STN has predicted treatment response (Zaidel, Spivak, Grieb, Bergman, & Israel, 2010). The strength of beta oscillatory activity also correlates with the clinical response to levodopa (Weinberger et al., 2006), and reductions in beta LFP activity in STN have been correlated with clinical improvement (Kuhn, Kupsch, Schneider, & Brown, 2006; Kuhn et al., 2008). The level of bradykinesia and rigidity in PD patients has also been positively correlated with the strength of spatially extended beta synchronization within different depths of the STN (Pogosyan et al., 2010), while the proportion of STN units oscillating at beta frequencies has been correlated with levels of limb rigidity (Sharott et al., 2014). Thus, beta oscillations may represent a valuable electrophysiological biomarker for disease activity in PD, particularly bradykinesia and rigidity (Zaidel et al., 2009).

PD is not just associated with increased temporal synchronization of neural firing within basal ganglia circuits, which is facilitated by the increased rhythmicity with which the neurons fire action potentials. There is also an associated enhancement in synchronization in the spatial domain, with neural populations within and between basal ganglia nuclei, and throughout the basal ganglia-thalamocortical loop, firing more coherently as exemplified by animal models of PD (Mallet et al., 2008a, 2008b; Nini et al., 1995; Raz, Vaadia, & Bergman 2000; Raz et al., 2001) and in PD patients undergoing DBS (Alegre et al., 2005; Brown et al., 2001; Marsden et al., 2001; Williams et al., 2002). Raz et al. (2001) simultaneously recorded spiking activity and LFPs in tonically active neurons (TANs) of the striatum and in GPe and GPi in MPTP-treated monkeys. They showed that following MPTP administration, significantly increased coherent oscillatory activity encompassing beta emerged in TAN-GPe and TAN-GPi neuronal pairs, when compared with the normal state prior to MPTP, implying increased striatal-pallidal interregional synchronization in the Parkinsonian state. Another study with MPTP-treated monkeys, demonstrated coherent oscillatory activity in the 10–15 Hz range between STN and GPi neuronal pairs (Moran et al., 2012). High frequency stimulation of the STN, a well-established form of treatment in PD, resulted in a functional decoupling in beta oscillatory synchronization between STN and GPi in these monkeys. Although this implies that decorrelating neural activity patterns in subthalamic-pallidal circuits may be a mechanism explaining the therapeutic efficacy of DBS, the authors did not corroborate this

through an assessment of the corresponding behavioral responses in the MPTP-primates in their study (Moran et al., 2012).

In patients undergoing surgery for DBS in PD, LFPs simultaneously recorded in GPi and STN show significant coherence at approximately 20 Hz and a smaller peak at 6 Hz (Brown et al., 2001). Significant coherence in the beta range has also been demonstrated between STN LFPs in sensorimotor and noninvasive scalp EEG activity over the supplementary motor area (SMA) in PD patients (Alegre et al., 2005; Fogelson et al., 2006; Marsden et al., 2001; Williams et al., 2002). A major limitation in invasive studies involving PD patients is the fact that—for obvious reasons—direct comparisons cannot be made with healthy human controls, but patients have to serve as their own controls. Identifying changes in the levels of coherence and oscillatory synchronization between different foci of the basal ganglia-thalamo-cortical loop in response to PD treatments has indeed provided valuable insights into the functional significance of such interregional neural synchrony. For example, high frequency stimulation of the STN adjacent to the recorded area that demonstrates the most coherence in the 15–30 Hz range with SMA produced the most effective relief of Parkinsonian symptoms (Marsden et al., 2001). Furthermore, levodopa treatment has been shown to substantially reduce coherence, at rest and during motor tasks, between GPi and STN, and between STN and SMA, in the beta range (20 Hz), but interestingly increase coherence at 70 Hz (gamma; Brown et al., 2001; Cassidy et al., 2002). The coherence within the beta range between these structures tends to decrease during movement, while gamma coherence tends to increase (Brown et al., 2001; Cassidy et al., 2002). As will be discussed later, oscillations in gamma range may have a prokinetic role.

Recent evidence has supported significant interhemispheric phase-locking within the beta frequency band in PD patients, at the cortical (Silberstein et al., 2005) and subcortical level (Little et al., 2013b). For example, the strength of cortico-cortical coupling in the beta band has been correlated with severity of Parkinsonism and reductions in such coupling strength have been correlated with clinical improvement following L-DOPA and STN stimulation (George et al., 2013; Silberstein et al., 2005). Significant phase-locking at beta frequencies between bilateral subthalamic nuclei in individual PD patients has been demonstrated, and this is selectively attenuated by levodopa (Little et al., 2013b). Thus, excessive beta synchronization on the interhemispheric scale could be another defining electrophysiological characteristic of PD.

However, some contradictory findings have emerged from the literature as well. The role of interregional spatial synchronization, for example, has recently been disputed by Devergnas, Pittard, Bliwise, & Wichmann (2014). The authors made simultaneous recordings of LFPs in GPi and STN, and EEG in M1 cortex using epidural electrodes, and measured signal coherence between each of these structures, in monkeys rendered progressively Parkinsonian with repeated injections of MPTP. The severity of Parkinsonism was correlated with significant increases in spectral power at frequencies below 15.5 Hz (overlapping with the low beta range) and reductions in spectral power at frequencies above 15.6 Hz in M1 and GPi, but with little change in STN. In agreement with the aforementioned studies, these intraregional changes in spectral power were reversed by levodopa treatment. Furthermore, the increasing strength of low-beta coherence between M1 EEG and GPi and STN LFPs were positively correlated with the severity of Parkinsonism. Although this latter finding suggests that increases in spatial synchronization between different nodes of the basal ganglia-thalamocortical loop may underlie Parkinsonian severity, levodopa treatment did not reverse these changes in interregional signal coherence (at < 15.5 Hz). These results may suggest that it is the increased oscillatory synchronization (at least at frequencies below 15.5

Hz in MPTP-treated primates) within the separate nodes of the basal ganglia-thalamocortical loop that plays a more important role in the manifestation of PD symptoms, as opposed to interregional coherence on a more global scale. Alternatively, the failure of dopamine replacement therapy to fully treat Parkinsonism, may suggest a disparate mechanism for its therapeutic efficacy.

In another paper, Leblois et al. (2007) also rendered monkeys progressively Parkinsonian with cumulative MPTP injections. Electrophysiological recordings made in GPi revealed that, despite the emergence of bradykinesia relatively early in the dopamine depletion process, increased oscillations encompassing beta manifested significantly later in the course of Parkinsonism. Although this undermines a causal role for beta oscillatory activity in the pathogenesis of PD, it is important to interpret such data with caution. Recordings were only made in GPi, and therefore the time course for the development of beta oscillations in this brain structure may not necessarily reflect the temporal dynamics in the rest of the basal ganglia-thalamocortical circuit. In addition, the rapid induction of Parkinsonism in the MPTP-primate model does not truly reflect the course of disease progression in PD patients, and so the genesis and mechanisms of the underlying oscillatory dynamics may differ between human disease and corresponding animal models.

Closed-loop DBS Paradigms

Recent studies employing closed-loop DBS paradigms have provided another means of investigating the pathophysiological significance of beta oscillatory dynamics in PD. Rosin et al. (2011) implanted microelectrodes in GPi and M1 of MPTP-treated monkeys in order to enable the recording of spike and oscillatory activity in both regions, while electrically stimulating GPi. The authors examined the effects of several adaptive closed-loop paradigms, which involved the automatic stimulation of GPi neurons, as a single or train of pulses, in response to action potential spikes detected in GPi or M1. They showed that this closed-loop setup was significantly more effective and efficient in ameliorating akinesia of the MPTP-treated monkeys, when compared with the conventional open-loop DBS paradigm that encompassed GPi stimulation regardless of any ongoing neuronal activity. An unexpected but fortuitous finding of this study was that stimulation of GPi (using a train of pulses) triggered by detection of GPi spikes, as part of a closed-loop paradigm, was associated with worsening of the monkeys' akinesia. Importantly, this was accompanied by a reduction in GPi neuronal firing rates—and not the increase that would be predicted by the canonical model—despite a significantly increased oscillatory bursting activity within the low beta range (12.5 Hz). The authors showed that there were no correlations between pallidal firing rate and oscillatory activity, thereby supporting independent mechanisms. Thus, this study is "proof of concept" that beta oscillatory activity, and not firing rate, is responsible for dictating the level of akinesia in PD models.

If beta oscillations causally drive the pathophysiological manifestations of PD, then manipulating it should lead to corresponding changes in behavior. Indeed, Little et al. (2013a) has provided a major line of evidence for this. The authors used a closed-loop adaptive DBS design in PD patients (n = 8) undergoing neurosurgery for DBS of the STN. LFP oscillations within the beta frequency bandwidth were detected using microelectrodes in STN. The adaptive DBS device was designed such that once the beta oscillatory amplitude exceeded a prespecified threshold, implanted electrodes would automatically send a high frequency train of impulses to the STN, until the beta oscillations dropped back down to below threshold. The authors revealed that this paradigm led to significantly greater improvements in Parkinsonian symptoms (rigidity, bradykinesia and tremor) when compared

with continuous and random intermittent DBS. Clinical parameters were recorded by observers both blinded and unblinded to experimental condition. Closed-loop adaptive DBS led to significantly greater improvements in motor symptom scores on the Unified Parkinson's Disease Rating Scale (UPDRS) compared with continuous DBS (50% vs. 27% improvement). The adaptive design was also more energy-efficient as it achieved greater improvements in symptoms using a 56% reduction in stimulation time. This study thus provides convincing evidence for the causal role of beta in the pathogenesis of PD. These results will need to be replicated by future studies employing larger sample sizes as part of robust randomized-controlled clinical trials.

Gamma Oscillations

High frequency oscillations within the gamma range (35–90 Hz) are believed to be pivotal in neural computational processing (Fries, 2009). It is assumed that they are primarily generated as a consequence of rhythmic inhibitory postsynaptic potentials (IPSPs) reverberating within interneuronal networks, and between inhibitory interneuron and excitatory pyramidal populations, via recurrent feedforward and feedback loops (Buzsáki, 2006; Buzsáki & Draguhn, 2004; Buzsáki & Wang, 2012). The behavioral correlates of gamma oscillations have been extensively investigated and especially linked with higher cognitive functions spanning attention, memory and perceptual binding (Buzsáki & Wang, 2012; Dehaene & Changeux, 2011; Fries, 2009; van der Helm, 2012). In the context of motor programming, less is known about the role of gamma oscillations compared with beta, but the two are thought to play reciprocal roles (Schoffelen, Oostenveld, & Fries 2005). Whereas beta has been inextricably associated with sustaining the motor set in a tonic state, maintaining the status quo and stopping unwanted behaviors, gamma oscillations have been positively correlated with voluntary movements and limb kinematics (Jenkinson, Kuhn, & Brown, 2013).

Gamma represents a broad range of frequencies, and data from human studies have especially implicated the 60–90 Hz frequency range in motor processing (Jenkinson et al., 2013). EEG studies in healthy volunteers, and data from invasive electrocorticographic (ECoG) recordings in patients with epilepsy, have collectively shown that during voluntary arm-reaching movements, there are focal increases in power and synchrony within the gamma band in corresponding regions of sensorimotor cortex and supplementary motor area (Ball et al., 2008; Miller et al., 2007; Ohara et al., 2000; Pfurtscheller, Graimann, Huggins, Levine, & Schuh, 2003). Direct LFP recordings in STN in PD patients provide convergent evidence. For example, performance of a voluntary movement during a stop signal paradigm was associated with an increase in gamma power in the subthalamic nucleus and cortico-subthalamic coherence in the gamma range, whereas successful inhibition of the response had the opposite effect (Alegre et al., 2013).

Treating PD patients with levodopa increases gamma power within the STN and enhances gamma synchrony between STN and GPi (Brown et al., 2001; Cassidy et al., 2002; Kempf et al., 2009), providing support for its beneficial, prokinetic functional role. However, gamma oscillations have also been associated with dyskinesia (Halje et al., 2012; Richter et al., 2013). Indeed, one of the major side effects of levodopa is dyskinesia, and since this medication has been associated with increases in gamma oscillations, such a deleterious clinical correlate of gamma should come as no surprise (Richter et al., 2013). In the hemiparkinsonian, 6-hydroxydopamine lesioned rat model, Halje et al. (2012) showed that periods of dyskinesia following levodopa therapy were strictly associated with gamma oscillations in the cortex and to a lesser extent, the ipsilateral striatum, of the affected

hemisphere, centered at approximately 80 Hz. Furthermore, localized cortical application of a D1-receptor antagonist significantly attenuated the dyskinesia and the corresponding 80 Hz oscillation. Evidence for the pathological role of gamma oscillations in dyskinesia, however, is still in its infancy, and further support for this theory remains to be evaluated in human studies (but see Alonso-Frech et al., 2006).

Although causal evidence for gamma oscillatory synchrony in driving novel motor behavioral patterns is still lacking, the evidence thus far described is supportive of the following theory: Motor processing and the expression of voluntary behavioral movements is determined by the relative balance between oscillatory synchrony in the beta and gamma range, throughout the basal ganglia thalamocortical loops. Neural networks within, and between, the cortex, basal ganglia, and thalamus communicate via oscillatory synchrony in parallel topographic spatial channels, somatotopically corresponding to different body regions. Furthermore, the frequency channels in which these neural loci interact dictate their behavioral representations, thereby creating another dimension to neuronal interplay. Pathological levels of synchrony at different frequency bands lead to corresponding pathological behavioral and clinical patterns. Excessive intra- and interregional beta synchronization, in particular, leads to a loss of segregation of these parallel-processing loops, ultimately creating the akinetic-rigidity phenotype of PD (see Figure 2). Although requiring more empirical support, excessive gamma synchronization may potentially underpin the chaotic kinaesthetic activity that characterizes dyskinesia syndromes.

The role of oscillations outside the beta and gamma range has been less explored. Theta (4–7 Hz) oscillations may also contribute to the development of dyskinesia (Alonso-Frech et al., 2006) and Parkinsonian tremor. Indeed, excessive oscillatory synchronization throughout a loop encompassing the neocortex, basal ganglia, thalamus, and cerebellum in the theta bandwidth, which coincides with the frequency of the Parkinsonian resting tremor, may represent a putative mechanism for such tremor (Schnitzler & Gross, 2005; Timmermann et al., 2003). However, as with beta oscillations, it remains to be determined precisely which component of this loop is responsible for generating the tremor rhythm (Deuschl et al., 2000; Dovzhenok & Rubchinsky, 2012).

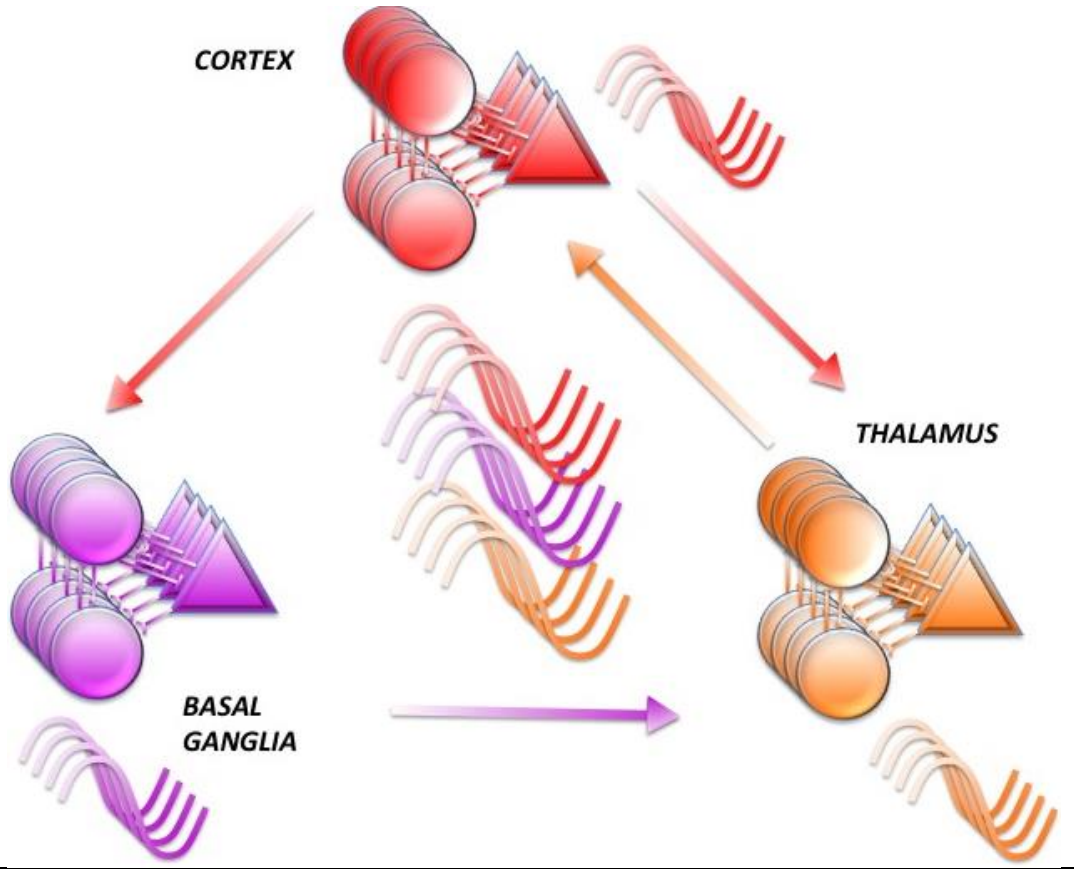

Figure 2. Synchronous interactions between neuronal groups occur at two distinct spatial scales. Oscillatory activity arises at the local neural circuit level within the cortex, basal ganglia, and thalamus. Oscillatory synchronization can also arise on a larger scale, which involves coherent activity across entire basal ganglia-thalamo-cortical networks. Although oscillatory synchronization plays a key physiological role in computational processing, excess synchronous oscillatory activity within local networks, and excess coherence across global networks, putatively contributes to the pathophysiology of PD.

Neurofeedback

Neurofeedback is a technique of learned self-regulation of neural activity. Participants receive online information about a parameter of neural activity and have to regulate it in a particular direction. Volitional regulation of brain rhythms or circumscribed brain regions (or networks) can be achieved through EEG or real-time functional magnetic resonance imaging (real-time fMRI) (see deCharms, 2008; Esmail & Linden, 2011; Ruiz, Buyukturkoglu, Rana, Birbaumer, & Sitaram, 2014; Sulzer et al., 2013). Such regulation of regional neural activity has been shown to be associated with predictable cognitive and behavioral effects (Ruiz et al., 2014) and has been extended to the treatment of psychopathologies, such as depression (Esmail & Linden, 2011; Linden et al., 2012; Linden, 2014). Having discussed the neural networks and oscillatory dynamics that putatively play a pathological role in PD, we will now perform a systematic review of the literature and discuss relevant studies that have explored the application of neurofeedback to PD.

Methods

We searched the following databases, during April and May 2014 (cut off date 27 May 2014), for clinical trials that investigated the therapeutic efficacy of neurofeedback training in Parkinson's disease: EMBASE 1947 to 2014, Ovid MEDLINE 1946 to 2014, PubMed 1990 to 2014, and SCOPUS 1960 to 2014. Our search terms included Parkinson's disease in combination with the terms: neurofeedback, EEG feedback, and real-time fMRI. Inclusion criteria included articles published in the English language and in peer-reviewed journals.

Results and Discussion of Neurofeedback Trials

We identified a total of six relevant papers investigating neurofeedback training in patients with Parkinson's disease: one case study and five controlled trials (two of which were randomized). An overview of these studies is summarized in Table 1.

Thompson and Thompson (2002) published the first case study, which analyzed the effects of EEG neurofeedback in a patient with Parkinson's disease and dystonia. In the ON medication state, the patient was trained to increase the 12–15 Hz rhythm of sensorimotor cortex, while inhibiting 6–10 Hz and 25–32 Hz cortical activity. Neurofeedback training was combined with biofeedback of respiratory and heart rate, in combination with diaphragmatic breathing exercises. The patient showed subjective improvements in her dystonic symptoms, the ability to overcome "freezing" of movements as part of PD, and improved quality of life.

Erickson-Davis, Anderson, Wielinski, Richter, and Parashos (2012) employed the first randomized, sham-controlled neurofeedback study design in PD. Patients in the ON medication state, and reporting levodopa-induced dyskinesia for at least 20% of the waking day, were randomly assigned to complete 24 thirty-minute sessions of active EEG-feedback training (n = 5) or sham feedback (n = 4). The active treatment group was trained to enhance 8–15 Hz (alpha and low beta) cortical activity at C3–C4 EEG electrodes, while inhibiting 4–8 Hz (theta) and 23–34 Hz (high beta) activity. Amplitude and coherence measures of the selected frequency bands were translated to audio feedback for participants. Initial efforts at down-regulating 4–8 Hz oscillations were abandoned as patients reported immediate subjective worsening of PD symptoms and reductions in their sense of wellbeing. Patients in the active treatment group were otherwise able to successfully manipulate EEG activity via operant conditioning, in the remaining target EEG frequency bands. Specifically, the active neurofeedback group significantly decreased 23–34 Hz, and increased 8–15 Hz power, in the right and left frontal and posterior regions. Within the coherence parameter, the only significant changes were increases in the 8–15 Hz range, and this was observed in the left frontal-right posterior linked regions. Despite significant changes in EEG measures, patients in the active treatment group showed no significant improvements in primary outcome measures of levodopa-induced dyskinesia, nor in secondary outcome measures of clinical features of PD represented by UPDRS scores, as with sham controls (whom did not receive veridical feedback).

Table 1
Overview of Studies

Study	Design	Training Paradigm	No. of Sessions	Medication State	Outcome
Thompson and Thompson (2002)	Case Study	Increasing 12–15Hz, while inhibiting 6–10 Hz and 25–32 Hz cortical rhythm; combined with biofeedback and diaphragmatic breathing exercise	30; over 6 months	ON	Overall improved quality of life. No standardized outcome measure was used.
Subramanian et al. (2011)	Non-randomized controlled trial; NF (*n* = 5 PD) vs. MI (*n* = 5 PD)	Up-regulation of SMA BOLD activity	2; over 2–6 months	ON	Only NF group successfully up-regulated SMA and achieved significant improvements in UPDRS and finger tapping scores.
Erickson-Davis et al. (2012)	Randomized-controlled trial; NF (*n* = 5 PD) vs. sham (*n* = 4 PD)	Increasing 8–15 Hz, while inhibiting 4–8Hz and 23–34Hz cortical activity	24; over 12–15 weeks	ON	Neither group showed significant improvements in LID (primary outcome measure) or UPDRS scores (secondary outcome measure).
Buyukturkoglu et al. (2013)	Small controlled study; NF (*n* = 1 PD) vs. NF (*n* = 3 healthy controls)	Up-regulation of SMA BOLD activity	Variable, ranged from 2 to 6	Not specified	Up-regulation of SMA slowed movements on a motor sequence task in all participants
Fumuro et al. (2013)	Non-randomized controlled trial; NF (*n* = 10 PD) vs. NF (*n* = 11 healthy controls)	Enhancing negative SCP shifts (negativation)	2–4 one-day sessions; at 1–6 day intervals	ON	Good NF performance resulted in increased early BP. Poor NF performance had the opposite effect. Behavioral measures not assessed.
Azarpaikan et al. (2014)	Randomized-controlled trial; NF (*n* = 8 PD) vs. sham (*n* = 8 PD)	Increasing 12–15 Hz, while decreasing 4–7Hz cortical activity	8; spanning 2.5 weeks	ON	Significant improvements occurred in static and dynamic balance in the NF group only.

Note: **LID** = Levodopa-induced dyskinesia; **MI** = motor imagery; **NF** = Neurofeedback; **PD** = Parkinson's disease; **SCP** = slow cortical potential; **SMA** = supplementary motor area; **UPDRS** = Unified Parkinson's Disease Rating Scale.

Although the clinical results have been disappointing, several issues must be borne in mind:

1. The study utilized a small sample size (*n* = 9).
2. Patients in the active neurofeedback group were not matched with those in the sham group (the treatment group were significantly older and had lower baseline dyskinesia).
3. The failure to significantly decrease coherence in the high beta range (23–34 Hz) over sensorimotor cortex may have underestimated the potential therapeutic efficacy of EEG feedback training.
4. Most importantly, the target frequency ranges for neurofeedback training may not have been appropriately selected.

Regarding the last point, we must review the frequency ranges that have been implicated in the pathophysiology of PD. The issue is somewhat complicated by the varying frequency ranges in which excessive oscillatory activity is seen amongst different animal models of PD and in PD patients. In rat models of PD, excessive synchrony is predominantly seen within the 30–35 Hz range (Delaville et al., 2014), while in MPTP-treated primates, excessive oscillations tend to be localized to a lower 8–15 Hz range overlapping alpha (Goldberg et al., 2004; Stein & Bar-Gad, 2013; Tachibana et al., 2011). In PD patients, the frequency range representing the corresponding antikinetic beta bandwidth has been attributed to 8–30 Hz, although this varies according to brain region (Levy et al., 2002; Stein & Bar-Gad, 2013). Indeed, with such a broad range, beta has been divided into low beta (up to 20 Hz) and high beta (20–35 Hz). The functional significance of this dissociation remains unclear, but studies have demonstrated that they may be differentially expressed in certain brain regions and respond to treatment differently. For example, in the STN of PD patients, there is often a higher power of spontaneous low beta, which is more significantly suppressed by dopaminergic treatment, when compared with high beta (Brittain & Brown, 2014; Litvak et al., 2011; Lopez-Azcarate et al., 2010). Thus, in the study by Erickson-Davis et al. (2012), training patients to up-regulate oscillatory activity in the 8–15 Hz range (which falls within the low beta range) and down-regulate activity in the 23–34 Hz (high beta), may have produced conflicting behavioral effects and could therefore account for their null findings. Furthermore, the authors did not target the potentially important frequency that has recently been implicated in levodopa-induced dyskinesia (80 Hz) (Halje et al., 2012), which was their primary outcome measure.

Future studies, recruiting larger patient numbers, should aim to identify the clinical effects of purely down-regulating beta oscillatory activity in sensorimotor cortical regions in PD patients. This could be corroborated by attempting to manipulate, via EEG neurofeedback, spectral power within a set of different, narrow, sub-frequency ranges encompassed by beta in a set of parallel experiments. Indeed, this may lend the opportunity to identify the optimal frequency range (within the broad possibilities of beta) that is most closely related to bradykinesia in PD, which may therefore produce the greatest clinical benefits following down-regulation. Although EEG neurofeedback offers little in the way of direct modulation of beta oscillations in the basal ganglia, modulating cortical beta could have an indirect impact on rhythmic subcortical activity. Studies in PD patients have shown excessive coherence of beta oscillatory activity between neocortex and basal ganglia (Fogelson et al., 2006; Marsden et al., 2001; Williams et al., 2002). Assuming that the pathophysiological basis of PD is underpinned by excessive beta synchronization in large-scale cortical-subcortical networks, the maximal possible efficacy of EEG-based neurofeedback would be limited by the top-down influence of cortical beta and its ability to entrain subcortical networks. The benefits of EEG neurofeedback would therefore be governed by direct down-regulation of

cortical beta, and subsequent indirect entrainment of subcortical beta, so that global oscillatory synchronization can be reset to a physiological level.

Cortico-cortical coherence in pairwise EEG comparisons in the beta range is prominent in the OFF state in PD patients, positively correlates with clinical severity and is reduced following dopaminergic medication and high frequency STN stimulation (George et al., 2013; Silberstein et al., 2005). Future neurofeedback experiments could therefore specifically focus on down-regulating cortico-cortical beta coherence, and then see if this is associated with clinical improvements. Not only would this identify another therapeutic avenue for PD, but it would also causally implicate the role of excessive cortico-cortical beta coherence in driving a major clinical impairment, namely bradykinesia, in PD.

In a more recent randomized-controlled trial (RCT), Azarpaikan, Torbati, and Sohrabi (2014) investigated the effects of EEG neurofeedback training in a group of patients, with mild PD in the ON state, on static and dynamic balance. Patients completed eight sessions of active neurofeedback (n = 8) or control sham feedback (n = 8) over a period of 2.5 weeks. In the active neurofeedback groups, participants were trained to enhance 12–15 Hz (low beta) oscillatory activity, while suppressing 4–7Hz (theta), using EEG signals recorded from occipital brain regions (O1–O2). This was achieved by immersing the participants in a video game, which was stopped if oscillatory power increased or decreased, outside a prespecified range, in the wrong frequency band. Sham controls were presented with random feedback, using the same video games, which was not contingent on brainwave activity patterns. Only the experimental group receiving active neurofeedback training successfully enhanced low beta, and suppressed theta, to a level that reached statistical significance. This was accompanied by significant improvements in both static and dynamic balance in the active neurofeedback group only. Unfortunately, the authors did not assess the behavioral effects of this neurofeedback paradigm on other Parkinsonian measures, namely bradykinesia, tremor or global symptom scores (e.g., UPDRS). This therefore limits our interpretation of the full range of behavioral effects that can be derived following manipulation of low beta and theta oscillations in the context of neurofeedback. Indeed, down-regulating theta activity could conceivably improve symptoms of tremor (Deuschl et al., 2000; Dovzhenok & Rubchinsky, 2012). Although this initially produced subjective deleterious effects in a small study (Erickson-Davis et al., 2012), it would need to be further evaluated in larger trials incorporating a broader selection of PD patients spanning different clinical states (e.g., including patients in the OFF state). To our knowledge, no other studies have suggested an association of beta or theta oscillations with balance in PD, so this is an area that will also require further future investigation.

In a different neurofeedback paradigm, Fumuro et al. (2013) investigated the possibility of manipulating the Bereitschaftspotential (BP), otherwise known as the readiness potential, in PD patients. BP represents the negative slow cortical potential (SCP) activation, which precedes, by up to 2 seconds, voluntary, self-paced movements (Shibasaki & Hallet, 2006). This negative shift in SCP reflects the widespread depolarization and increased excitability of the superficial layer of apical dendrites from cortical pyramidal neurons (Birbaumer, Elbert, Canavan, & Rockstroh, 1990). Positive shifts in SCP, by contrast, reflect decreases in cortical excitability. BP is divided into an early and late component. Early BP predominantly reflects widespread bilateral increases in neuronal activity, initially in the pre-supplementary motor area (pre-SMA), followed by somatopically coordinated activation in SMA and lateral premotor areas (Ikeda, Luders, Burgess, & Shibasaki, 1992; Ikeda et al., 1995; Yazawa et al., 2000) bilaterally. Late BP is somatopically even more specific for the site of movement, and mainly arises from generator current sources in the contralateral primary motor cortex

(Shibasaki & Hallet, 2006). Dick et al. (1989) showed that the early BP component was significantly smaller in patients with PD in their OFF state, compared with healthy controls. Additionally, early BP displays an increase in amplitude following dopaminergic therapy in PD patients (Dick et al., 1987), suggesting that it may represent an attractive electrophysiological target potentially amenable to modulation via neurofeedback training.

This putative electrophysiological biomarker has recently been targeted by Fumuro et al. (2013). They performed a controlled EEG neurofeedback trial comparing 10 PD patients in the ON medication state with 11 age-matched healthy controls, all of whom were right-handed. EEG data (using the international 10-10 system) encompassing the BP shifts were recorded before and after neurofeedback training. During measurement of BP, all participants were instructed to perform self-paced, brisk button presses every 10 seconds with their right thumb while looking at a blank screen, over approximately 100 trials. The first BP recording session was followed, after a 5-minute break by neurofeedback training. During neurofeedback, subjects were instructed to produce negative SCP shifts (negativation) guided by their own mental strategies, but were provided with appropriate suggestions of mental introspection by the examiners, if required. Neural activity was transformed into a visual representation, and provided the means of feedback that guided participants' mental strategies so that they could learn to manipulate their SCP. BP was subsequently measured post-neurofeedback training in a similar manner as pre-neurofeedback, while participants simultaneously mustered introspective efforts to produce negative SCP shifts. This study showed that PD patients were capable of successfully manipulating early BP, as good neurofeedback performance was associated with a statistically significant increase in early BP amplitude. Unfortunately, the authors did not assess any symptomatic or clinical measures of PD, so the behavioral effects, or functional significance, of such manipulation of early BP could not be determined.

Volitional control of neural activity can also be achieved using real-time fMRI-based feedback, whereby data processing and analysis closely follow data acquisition. This enables fMRI results and images to be produced as the experiment unfolds in real-time (deCharms, 2007). The BOLD signals from a selected brain region can then be fed back to the participant, usually in the form of some dynamic visual representation, in order to permit learned up- or down-regulation of brain activity. The efficacy of real-time fMRI feedback has been extensively investigated in recent years, and it has been demonstrated that learned regulation of regionally circumscribed brain regions is frequently associated with predictable behavioral effects (Ruiz et al., 2014).

Subramanian et al. (2011) have evaluated the use of real-time fMRI neurofeedback in patients with PD as part of a non-randomized controlled clinical trial. Patients in the ON state, were allocated to an experimental neurofeedback group (NF, n = 5) or a control group (CG, n = 5), matched for clinical severity and medication. The target brain region selected for up-regulation in the NF group was SMA, owing to its extensive connections with the basal ganglia thalamocortical circuit implicated in PD. During neurofeedback training, NF patients received a visual display, consisting of a thermometer, which provided a representation of the level of neural activity within the SMA, translated from BOLD signal data in real-time. NF patients were trained to up-regulate SMA in two separate sessions, each consisting of two neurofeedback runs (6.5 minutes each). They were instructed that they may use motor imagery to achieve SMA up-regulation, but were not prescribed any specific strategies. CG patients, however, were also instructed to use motor imagery while undergoing fMRI scanning, but importantly did not receive veridical feedback on SMA neural activity. In the 2–6 months between the two neurofeedback or motor imagery sessions, patients were

instructed to use the same mental strategies at home on a daily basis, but to refrain from practicing overt hand movements. The authors showed that only NF patients were able to significantly up-regulate SMA BOLD activity to a level that was comparable to the activation that was achieved while performing overt hand movements during the initial localizer runs. In contrast, CG patients displayed significantly less BOLD activation in SMA during motor imagery, compared with localizer runs, and neural activity failed to increase during subsequent motor imagery runs. Successful SMA up-regulation following neurofeedback, was accompanied by significant improvements on UPDRS symptom scores (37% improvement in motor function) and on a finger-tapping task. The CG did not show any such clinical or behavioral improvements. This proof-of-concept study of fMRI neurofeedback in PD was limited by the small sample size and unblinded assessments.

A more recent pilot study, employing a similar real-time fMRI neurofeedback paradigm, undertaken by Buyukturkoglu et al. (2013) yielded conflicting results. The authors recruited one patient with PD and three healthy volunteers. Participants undertook pre- and posttest runs separated by a block of neurofeedback training trials. During neurofeedback training, all participants learned to up-regulate activation in bilateral SMA, while using their own cognitive strategies, guided by a visual display representing SMA BOLD signal changes in real-time. Up-regulation of SMA in all participants, including the PD patient, resulted in slower responses on a sequenced motor task. However, the authors did not assess the impact of this task on clinical symptomatology in the PD patient. Although our interpretation of these results is limited, given the small sample sizes, they highlight two important issues:

1) The potential variability in pathological activation levels in different PD patients.
2) The possible pleiotropic effects of real-time fMRI feedback.

Concerning the former, the evidence from the literature is inconsistent regarding the direction of pathological activation of SMA in PD. Some studies have reported hypoactivation of SMA (Jenkins et al., 1992; Wu, Wang, Hallett, Li, & Chan, 2010; Yu, Sternad, Corcos, & Vaillancourt, 2007), whereas others have reported hyperactivation (Catalan, Ishii, Honda, Samii, & Hallett, 1999; Rowe et al., 2002). This is complicated by the fact that regional differences in activation have also been observed within the SMA itself (Sabatini et al., 2000). Thus, attempting a simple method of grossly increasing or decreasing activation of regional brain activation, through region of interest (ROI)-based univariate analyses, may not necessarily achieve the desired therapeutic effects. In order to more accurately model the aberrant neural network connectivity patterns, spanning the basal ganglia thalamo-cortical loop, which characterizes PD, more sophisticated real-time fMRI algorithms would be required. One such example invokes the use of multi-voxel pattern analysis (MVPA). MVPA centers on training pattern classifier algorithms to decode the mental representation of a behavioral, cognitive, or emotional state based on discriminating between the underlying neural activation signatures. These trained pattern classifiers can then predict subsequent manifest cognitive states by reading the evoked neural activation patterns (represented by a spatially distributed set of activated voxels in the context of fMRI), based on the experience gained during training (for review, see Haynes & Rees, 2006). Pattern classifiers have been applied in real-time fMRI paradigms (Hollmann et al., 2011; LaConte, Peltier, & Hu, 2007; Sitaram et al., 2011) and have been shown to improve performance in perceptual discrimination tasks through neurofeedback training (Shibata, Watanabe, Sasaki, & Kawato, 2011). MVPA could potentially be utilized to overcome the limitations of univariate real-time fMRI methods in the context of PD, by avoiding an *a priori* approach and by closely modeling the complex network dynamics that underpin this pathology. For example, pattern classifiers could conceivably be trained to distinguish distributed neural activation patterns and functional connectivity states between PD patients and healthy volunteers (or between the

ON and OFF medication state in PD patients). This would be predicated on the principle that hypersynchronized network dynamics across basal ganglia thalamocortical loops underscores the pathophysiology of PD. The precise neurofeedback that PD patients would receive therefore, in this MVPA real-time fMRI paradigm, would be a measure of how closely correlated the patient's neural activation pattern is in comparison to the "healthy" state, or the ON medication state, for example. Thus, PD patients could be trained to replicate the spatially distributed activation patterns that are representative of the healthy or ON state, by breaking down inter-neuronal hypersynchrony, and correspondingly derive the associated behavioral and symptomatic benefits. This hypothesis remains to be tested, but the limitations of this potentially powerful tool grounds down to the very signals that fMRI measures, which brings us to the possible pleiotropic effects of real-time fMRI feedback.

BOLD signal dynamics reflect a combination of changes in blood flow, volume and oxygenation, within a brain region, in response to a perceptual, cognitive or motor demand (Logothetis, 2003, 2008). A major limitation to interpreting increases in BOLD signals, therefore, is that it can arise as a consequence of overall increases in excitation, inhibition or a combination of both (Buzsáki, Kaila, & Raichle, 2007; Logothetis 2008). The corollary of this is that modulating BOLD activity through real-time fMRI feedback could produce differential effects, as the participant is given no control over whether, and in what proportion, excitatory or inhibitory networks are activated. Consequently, up- or down-regulating BOLD signals in brain regions, in different times or in different people, may not necessarily yield consistent results, and so may partially explain the conflict in findings between the aforementioned real-time fMRI feedback paradigms (Buyukturkoglu et al., 2013; Subramanian et al., 2011). The other major limitation of real-time fMRI is the poor temporal resolution owing to the unavoidable haemodynamic lag between neural activation and the onset of BOLD signal changes (ca. 3–6 seconds; Logothetis, 2008). This ultimately prevents any real-time fMRI feedback paradigm from probing into the millisecond-range temporal dynamics that is pivotal in determining neuronal oscillatory synchrony. However, it has been suggested that BOLD signals closely correlate with changes in LFP oscillations, particularly in the gamma band (Magri, Schridde, Murayama, Panzeri, & Logothetis, 2012), which could prove useful for attempts at manipulating oscillatory synchrony in PD through neurofeedback.

Conclusions

Although neurofeedback offers a promising, noninvasive means of modulating regional brain activity, or enhancing compensatory networks, the evidence supporting its potential therapeutic efficacy in disease states remains in its infancy. In the context of PD, there is insufficient evidence at present to support the application of neurofeedback in routine clinical practice. Extant studies have suffered from small sample sizes and/or methodological limitations. The EEG feedback studies have trained participants to manipulate oscillations within targeted frequency ranges in a manner that is inconsistent with the mechanisms that have been suggested to contribute to the pathophysiology of PD, which may explain certain null findings (Erickson-Davies et al., 2012). In the EEG study that has yielded positive results of neurofeedback in PD (Azarpaikan et al., 2014), the precise mechanism for this is unclear as the role of cortical oscillatory dynamics in balance in PD patients has not before been explored. Modulating the BP could potentially offer another promising avenue for treating PD (Fumuro et al., 2013), but the behavioral and clinical effects of such modulation will need to be investigated.

Real time fMRI neurofeedback offers a promising prospect for modulating regional brain activity. Initial evidence on the effects of SMA modulation has been promising, but this would need to be verified in larger, controlled clinical trials. By providing brain-wide access with a high spatial resolution on the order of a few millimeters, real time fMRI could enable activity of subcortical basal ganglia structures to be controlled, which cannot be easily accessed using EEG-based paradigms. The effects of modulating activity in various basal ganglia nuclei, or their combinations, on behavior and PD symptomatology will need to be evaluated if this field is to make progress.

Finally, future well-designed robust studies, recruiting larger sample sizes, and applying methodological principles predicated on the existing evidence base for the neural mechanisms implicated in PD are required before neurofeedback could be recommended in clinical practice.

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NeuroRegulation

Proceedings of the 2014 ISNR Conference

Selected Abstracts of Conference Presentations at the 2014 International Society for Neurofeedback and Research (ISNR) 22nd Conference, Bayside San Diego, California, USA

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KEYNOTE PRESENTATIONS

Harnessing Brain Plasticity: The Future of Neurotherapeutics

Adam Gazzaley, MD, PhD
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Principal Investigator, Gazzaley Lab, University of California, San Francisco, California, USA

A fundamental challenge of modern society is the development of effective approaches to enhance brain function and cognition in both healthy and impaired individuals. For the healthy, this serves as a core mission of our *educational system* and for the cognitively impaired this is a critical goal of our *medical system*. Unfortunately, there are serious and growing concerns about the ability of either system to meet this challenge. I will describe an approach developed in our lab that uses custom-designed video games to achieve meaningful and sustainable cognitive enhancement (e.g., Anguera et al., 2013), as well the next stage of our research program, which uses video games integrated with technological innovations in software (e.g., brain computer interface algorithms, Neurofeedback, GPU computing) and hardware (e.g., virtual reality headsets, mobile EEG, transcranial electrical brain stimulation) to create a novel personalized closed-loop system. I will share with you a vision of the future in which video games serve as an underlying engine to enhance our brain's information processing systems, thus reducing our reliance on non-specific drugs to treat neurological and psychiatric conditions and allowing us to better target our educational efforts.

Online, Voluntary Control of Individual Neurons in the Human Brain

Moran Cerf, PhD
Assistant Professor of Business and Neuroscience, Kellogg School of Management, Illinois, USA
and the University of California, Los Angeles, Department of Neurosurgery, California, USA

Recording from single neurons in patients implanted with intracranial electrodes for clinical reasons, I will demonstrate that humans can regulate the activity of their neurons in the medial temporal lobe (MTL) to alter the outcome of the contest between external images and their internal representation using feedback from these neurons. I will discuss some of the works in real-time recording of the activity of individual neurons in the brains of humans in the last decade, and how these can be used to access the underlying mechanisms of our decisions, emotion, memory, and free will.

KEYNOTE PANEL

Experiences and Opportunities of Brain Training for Athletes

Moderator: *Leslie Sherlin, PhD,* SenseLabs
Panel: *Carlos Quentin,* Outfielder, San Diego Padres
Kaya Turski, Canadian Freestyle Skier, X Games
Dan Buchner, Professional Golfer

Athletic performance has in the past focused on training the body. However, an athlete who can exercise volitional control of the levels of cognitive engagement and arousal of brain and body has an advantage during competition. A growing interest in a quantified self and the technological advances contributing to wearable sensors are creating a new opportunity in the field of sport psychology. The panel moderator will provide a brief overview of the most recent trends in the implementation of electroencephalography evaluation and training methods in professional and world-class level athletics. The panel will host professional athletes of wide-ranging demographics who have utilized these techniques to help them achieve the highest levels of success in the world of sports. Case-specific data of psychophysiological and performance outcomes will be elaborated upon as each athlete will describe his or her experiences of the evaluation and training. Through interview style discussion, they will discuss the reasons they chose to implement these techniques and the benefits they received from the training.

INVITED PRESENTATIONS

An Introduction to Misophonia: Case Reports and Physiological Findings

Miren Edelstein, Doctoral Student
University of California, San Diego, California, USA

Misophonia, literally translated to “hatred of sound,” is a little known condition in which certain sounds trigger an involuntary “fight or flight” response in certain individuals. These sounds are often repetitive, innocuous, and social in nature (gum chewing, pen clicking, lip smacking). When exposed to these trigger sounds, misophonic individuals report intense feelings of anger, panic, and rage to the point where their everyday lives can be severely impacted. Here, I describe case reports and provide preliminary physiological evidence (via skin conductance) suggesting that misophonia evokes heightened autonomic responses to certain sounds not seen in control subjects.

Altered Cerebral Connectivity and the Corpus Callosum: Adaptation or Dysfunction

Elliott Sherr, MD, PhD
Professor of Neurology and Pediatrics, University of California, San Francisco, California, USA

Agenesis of the corpus callosum (ACC) is a common disruption in brain development, which is often associated with cognitive and behavioral deficits including autism, intellectual disability, and disruptions in emotional regulation. There is also considerable variation in the expression of these phenotypes in different cases of ACC, suggesting that understanding the

pathways implicated and the genetics that underlie the causes of ACC may shed light on how altered connectivity leads to both pathology and adaptive responses.

Double-Blind Randomized Clinical Trial of Neurofeedback for ADHD: Rationale and Strategy

Roger deBeus, PhD
University of North Carolina, Asheville, North Carolina, USA

Objective. Current established, evidence-based treatments for attention-deficit/hyperactivity disorder (ADHD) are incompletely effective and not universally acceptable, and appear to wane in effect over time despite significant immediate benefit. Additional treatments are needed that are effective with persisting benefit, preferably related to a biomarker predicting treatment response. A good candidate is neurofeedback (NF) based on observations that patients with ADHD often have excessive theta band (4–8 Hz) quantitative EEG power, low beta band (13–21 Hz) power, and excessive theta-beta ratio (TBR). Although there has been an explosion of ADHD NF research over the past decade, overall efficacy remains inconclusive.

Method. Experts in NF, ADHD, clinical trials, statistics, and data management have joined to design a double-blind sham-controlled randomized clinical trial. This 5-year study has been funded by the National Institutes of Mental Health (NIMH). At each of two sites (one university and one NF clinic) 70 children (total $N = 140$) ages 7 through 10 with rigorously diagnosed moderate to severe ADHD and TBR > 5 will be randomized in a 3:2 ratio to active TBR down-training by NF versus a sham training of equal duration, intensity, and appearance. Multi-domain assessments at baseline, mid-treatment, treatment end, and follow-ups at 6 months, 1 year, and 2 years will include parent and teacher ratings of symptoms and impairment, neuropsychological tests, clinician ratings, and quantitative EEG, as well as tests of blinding and of sham inertness.

Results and Conclusions. Hypotheses include that NF will improve parent- and teacher-rated inattentive symptoms (primary outcome) and other outcomes more than sham, that benefit will persist for 2 years after training, that initial TBR will moderate treatment response, and that change in TBR will mediate response. Research Domain Criteria and EEG brain changes will be explored, including relationship of TBR to clinical symptoms, executive-function impairment, and sleep.

STUDENT TRAVEL AWARD WINNERS

Prefrontal Neurofeedback Training Approaches in Autism

Yao Wang, MS, Estate Sokhadze, PhD, Ayman El-Baz, PhD, Lonnie Sears, PhD, Allan Tasman, MD, and Manuel Casanova, MD
University of Louisville, Kentucky, USA

Background. Neurofeedback (NFB) has a potential as an intervention for autism spectrum disorder (ASD) treatment. Neurofeedback applications for attention-deficit/hyperactivity disorder (ADHD) has recently been reviewed extensively (Arns, de Ridder, Strehl, Breteler, &

Coenen, 2009; Gevensleben et al., 2009; Sherlin, Arns, Lubar, & Sokhadze, 2010). Several publications suggest that ADHD protocols may also be efficacious for the treatment of autism (Kouijzer, de Moor, Gerrits, Buitelaar, & van Schie, 2009; Kouijzer, van Schie, de Moor, Gerrits, & Buitelaar, 2010). However, strategies commonly used in ADHD treatment (suppression of the frontal theta, enhancement of the SMR or slow beta) cannot be duly transferred to ASD treatment. Coben's studies (Coben & Padolsky, 2007; Coben & Myers, 2010; Coben, Linden, & Myers, 2010) along with Scolnick (2005) and Jarusiewicz (2002) findings point at the preference of individualized protocols that use qEEG-guided intervention targets.

Objectives. We believe that approach in autism neurotherapy should focus on prefrontal NFB training, which may result in better executive functions. We selected two protocols: one targeting suppression of high amplitude (Inhibit All protocol [IA], i.e., wideband suppression) across all EEG bands (typically used in ADHD), while the second one targeting increase of the relative power of 40 Hz centered gamma (gamma [RG]) in addition to Inhibit All at FPz site. The current pilot study is aimed to compare effectiveness of two prefrontal NFB protocols in autism. Development of more refined prefrontal NFB protocol for ASD was one of the main goals.

Method. The study participants with ASD diagnosis were recruited from Department of Pediatrics. In first group (N = 8, M = 14.9 years) we used twelve 25-minute long sessions of weekly IA protocol, while in the second group (N = 15, M = 13.6 years) we used gamma up-regulation [RG] protocol (25 min, 12 sessions) using PAT device. Feedback was arranged in a form of auditory feedback and visual feedback (bars, lines) with information about values of controlled parameters, their relation to the threshold (e.g., % over threshold), and other descriptors of the training progress. We also used control of DVD by training parameters (e.g., controlling size and brightness of video by EEG feedback). Analysis of EEG during NFB training was conducted for relative power of bands and several ratios during 25 minutes of each session and across the course of neurotherapy as it was reported in our study on ADHD (Hillard, El-Baz, Sears, Tasman, & Sokhadze, 2013).

Results. The trainees in IA protocol were successful in reducing theta/beta and theta/alpha ratios, but effects were not transferred from session to session. The second protocol—aimed to target increase of relative gamma power—resulted both in reduction of slow wave proportion and increase of higher frequency rhythms power (high beta, gamma) and decrease of theta/beta and theta/gamma ratios, not only during the session but also across sessions. This group also showed EEG changes that were typical for the IA group (i.e., decreased theta/slow beta ratio).

Conclusion. Application of prefrontal neurofeedback protocols aimed to increase higher frequency activity (beta, gamma) along with suppression of slow frequencies (theta) could be considered as a promising approach that warrants further investigation using as outcome measures not only EEG changes during NFB, but also clinical behavioral measures and quantitative EEG outcomes.

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Transcranial Direct Current Stimulation Improves Visual and Spatial Aspects of Memory in Major Depression

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With a lifetime prevalence estimated at 16%, major depression (MD) is a major public health issue. Previous studies have shown that depression has been associated with a variety of cognitive impairments. In addition to cognitive impairments, MD is usually accompanied by alterations of cortical activity, especially in prefrontal areas. Recent studies have highlighted the importance of noninvasive brain stimulation as a means of modulating cortical excitability. Recent studies on MD have revealed that transcranial direct current stimulation (tDCS) induces cortical excitability, which facilitates memory and especially working memory. On the other hand, visual aspects of memory in MD have not been yet investigated.

Objective. This study aimed to investigate whether anodal and cathodal tDCS applied over dorsolateral prefrontal cortex (DLPFC), would significantly improve visual memory in patients with MD.

Methods. Thirty (*N* = 30) patients with MD were randomly assigned to receive either experimental (active) or control (sham) tDCS. The participants underwent a series of visual memory tasks before and after 10 sessions of tDCS. The parameters of active tDCS included 2 mA for 20 minutes per day for 10 consecutive days, anode over the left DLPFC (F3), cathode over the right DLPFC (F4) region.

Results. After 10 sessions of anodal and cathodal tDCS, patients showed significantly improved performance in visual and spatial aspects of memory tasks. Specifically, anodal stimulation improved visual memory performance for the experimental group relative to baseline, whereas sham stimulation did not differentiate performance from baseline in the control group.

Conclusion. This study showed that anodal tDCS over DLPFC concurrently with cathodal tDCS over right DLPFC improved visual and spatial aspects of memory in patients with MD. This finding is in generally consistent with previous findings about effectiveness of tDCS on cognition in MD, while additionally provides support for effectiveness of tDCS on visual memory in MD.

ORAL PRESENTATIONS

Successful Treatment of 25 Consecutive Clients with Post-Concussion Syndrome using Neurofeedback and a Variety of Adjunctive Interventions

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In 2013, the National Football League agreed to a $765 million settlement to pay for professional football player post-concussion brain injuries. The recognition that all head

injuries can have delayed—sometimes decades of delay—impact on cognitive, emotional, and behavioral functioning. Head injuries from all sports, traffic accidents, and military service are being more closely scrutinized for the delayed symptoms of post-concussion syndrome disabilities.

This presentation will offer proven assessment and treatment methods for post-concussion syndrome and other head injuries. It will apply the Quadrant Brain Theory of individualized Brain Quadrant EEG assessment and other neuropsychology assessments as the basis for treatment. Post-Concussion Syndrome and most head injuries are not a unitary condition, but instead are multifaceted with various constellations of symptoms. This necessitates careful assessment protocols that are linked with specific treatment methods that address the symptoms and clusters.

The results of a study of 25 consecutive clients with Post-Concussion Syndrome brain injuries will be presented. The application of the Quadrant Brain Theory was previously demonstrated as successful in the neurofeedback treatment of chronic inpatient schizophrenia (Bolea, 2010) and severe, suicidal depressed outpatient clients (Bolea & Romig, 2013).

Quadrant Brain Theory-based neurofeedback was expanded with the post-concussion clients to include adjunctive modalities of intervention: respiration biofeedback, exercise, nutrition, sleep hygiene practices, and other behavioral and cognitive methods. The diagnostic characteristics of the clients will be described including the number of head injuries, the presence of psychological trauma, the variety of symptoms at intake, and the length of time between head injuries and onset of symptoms.

Of the 25 clients, 21 were completely rehabilitated, while the other 4 clients went from total disability to only partial disability. The neurofeedback protocols and the adjunctive treatments that were found most useful will be presented. The principles for which adjunctive modalities to use based upon the severity and co-morbidity of symptoms will be outlined.

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The Effects of Neurofeedback in the Default Mode Network: Pilot Study Results of Medicated Children with ADHD

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Children with attention-deficit/hyperactivity disorder (ADHD) have difficulty activating the Default Mode Network (DMN) in a resting or quiet state. The DMN function assists in processing and understanding a person's internal, reflective world and the world of self and others. Neurofeedback (NFB), a type of EEG operant and classical conditioning, trains self-regulation skills using a brain-computer interface. The hardware and software have audio-video capabilities to correct irregular brainwave patterns and regional cerebral blood flow

associated with mental health and cognitive concerns. Individual treatment sessions usually last 20 minutes to gain the largest overall treatment effect. Typically NFB users need to experience about 30 to 40 sessions.

This experimental pre-post test design pilot study randomly assigned 12 children diagnosed with ADHD and currently on a stimulant medication to a treatment and control group. Subjects in the treatment group completed 40 NFB sessions. Pre- and posttest fMRIs were administered on the treatment and control groups. Evidence showed that forty 20-minute sessions of Sensory Motor Rhythm (SMR) NFB consolidated the DMN allowing for appropriate activation in the posterior cingulate, precuneous, the temporoparietal junction and the cerebellar tonsils. In addition to regulating and increasing SMR at 12–14 Hz, our research results showed activation of the DMN in a resting state after 40 NFB sessions. Symptom checklists were completed after each NFB session, and our results showed significant overall symptom reduction. Our study showed that NFB treatment of medicated ADHD subjects led to clinical improvement that was accompanied by improvement in the DMN functions. Of additional interest is the group average pretreatment fMRI. This image suggests the limited anterior to posterior connectivity and the ectopic DMN components that are seen in the 9- to 15-year-old brain. The absence of these findings posttreatment suggests that the NFB also resulted in maturation of the DMN toward that of the adult brain in a period of several months.

Assisting children with ADHD to appropriately activate the DMN may help them be more adaptive and reflective and to better understand their own internal world and the world of others. A discussion of these research results and the maturation of the DMN will be at the core of this workshop.

The limitation of this study was the small sample size and male to female ratio of subjects. To address this concern, a large PCORI grant has been written and submitted to conduct further research on the effectiveness of NFB and the DMN with a sample size of 75 children with ADHD. A sham control has also been added.

LORETA Z-Score Neurofeedback is Effective and Specific in the Treatment of Post-Traumatic Stress Disorder and Traumatic Brain Injury in Combat Veterans

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Post-traumatic stress disorder (PTSD) is becoming understood as a set of dynamic functional neural network disturbances through the advancement of increasingly accurate and available neuroimaging techniques. These same techniques are contributing to the understanding of traumatic brain injury (TBI) and the mechanics of both the common characteristics of mild TBI (mTBI) and the possible idiosyncratic neural disruptions. Since both PTSD and TBI can involve a wide range of neural dysregulations, maximal treatment outcome will result from optimal specificity of assessment and treatment. Combat veterans often suffer from both PTSD and mTBI resulting in numerous, complex, difficult to treat, and often disabling symptoms. This study reports on an ongoing project providing treatment to U.S. combat veterans suffering from both PTSD and mTBI utilizing 3-D tomographic EEG (tEEG) Low Resolution Brain Electromagnetic Tomography (LORETA) Z-Score Neurofeedback (LZN) driven by a symptom check list, functional neural network match (SCL-FNM) method. Objective and subjective results of the first 16 cases indicate this method is effective and

specific. Subjective responses to the training indicate relatively rapid remediation of symptoms in many cases. Quantitative analyses using paired *t*-tests demonstrate significant normalization of current density and network connectivity measures in the targeted cortical and subcortical regions of training. Large effect sizes, based on Cohen's analysis, are observed in current density, phase, coherence, phase shift, and phase lock. A negative correlation between effect size and psychotropic medication was found along with a trend toward requiring less medication as training progressed. These interactions between LZN and psychotropic drugs provide a rationale for optimal cooperation among the trainee, LZN trainer, and prescribing physicians to maximize treatment efficacy. LZN based on the SCL-FNM method is evidently both effective and specific in the treatment of combat veterans with PTSD and mTBI. Based on the content of this presentation, the participant will be able to identify functional neural network models of PTSD and mTBI and understand and justify the use of LZN in the treatment of these disorders through the operant conditioning of current density, phase, coherence, phase lock, and phase shift.

Unlocking Parkinsonian Resting Tremor with Neurofeedback

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Parkinson's disease is the second most common progressive degenerative disease affecting as many as one million individuals in the US and four million worldwide. Approximately four percent of people with Parkinson's are diagnosed before the age of 50 and the incidence increases with age. Currently there is no cure for Parkinson's disease and finding the right treatment as symptoms change over time is important and requires the expertise of specialized health care professionals.

Resting tremor, the most common symptom of Parkinson's disease, is treated with dopamine replacement therapy, surgical ablation, or with electrode implantation called Deep Brain stimulation. Innovative studies have discovered that tremor-correlated cortical activity can be detected by electroencephalography (Hellwig et al., 2000; Muthuraman, Raethjen, Hellriegel, Deuschl, & Heute, 2008). The findings underline that the motor areas of the cerebral cortex are involved in the neuronal network generating resting tremor in Parkinson's disease. Further studies have confirmed the pathological neural network for Parkinsonian resting tremor (Brittain, Sharott, & Brown, 2014; Brown, 2003; Helmich, Hallett, Deuschl, Toni, & Bloem, 2012; Timmerman et al., 2003; Timmerman & Fink, 2011). Understanding the cortical representation and localization of tremors makes it possible to develop Neurofeedback training protocols to decrease resting tremor.

This presentation will review scientific literature outlining the pathological network activity of Parkinsonian resting tremor. Case studies will be presented to demonstrate the decrease of Parkinsonian rest tremor during Neurofeedback training. The resting tremor is measured by Digitrac, a triaxial accelerometer that calculates and displays movement frequency and amplitude using Fast Fourier Transform (FFT). The tremor frequency and its three harmonics will be discussed. One case study of sham Neurofeedback for resting tremor with accelerometer recordings will be presented and discussed. Accelerometer recordings will also be provided to show the starting and stopping of rest tremors in a single session. Easily accessible tools for measuring resting tremor will be provided so that a clinician can monitor progress within a session and between sessions.

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Brain SPECT Scans, What is it, How May it Be Used to Help My Patients in My Practice

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A brain single-photon emission computerized tomography (SPECT) scan is a nuclear diagnostic test that provides information about the circulation of the brain and, to a lesser extent, the anatomy of the brain. The scan is performed by injecting a radioisotope intravenously and placing a camera around the patient's head. Radioactivity exits the patient's head and is absorbed by a special camera, and then that data is transferred into a computer. The computer has a program that converts the radioactivity into pictures of the brain similar to a MRI, as slices in both 2-D slices (axial, coronal, and sagittal) and 3-D surface maps unique to SPECT technology. The 3-D images are extremely useful in assessing trauma cases where the maximum injury is, to anterior poles of frontal and temporal lobes, which are easily imaged unlike in MRI, CT, or PET scans. Other advantages are that SPECT scans provide metabolic information about the circulation, and the results can be abnormal even though the structural imaging tests such as CT/MRI/PET are

completely normal. Hence the notion of normal brain anatomy yet abnormal metabolic function is firmly established, and analogous to the well-documented example of an abnormal EEG and normal CT/MRI/PET of the brain. Another major clinical advantage is that it can provide the basis for a medical treatment in a variety of common clinical conditions, such as traumatic brain injury, ADD/ADHD, autism, and more. A common finding in many of the conditions listed above is a reduction of blood flow known as hypoperfusion. Since the brain is such a highly active metabolic organ, a very small degree of hypoperfusion can have a very deleterious consequence. Thus any treatment that either increases blood flow and/or increases oxygen content in the blood/brain will improve brain function. A medication with the specific purpose of increasing cerebral blood exists, and has been found to improve clinical symptoms in the conditions listed above. It is a safe FDA-approved drug in the category of calcium channel blockers, and is very well tolerated with minimal or no side effects. Hyperbaric oxygen has a similar beneficial effect on improving function of the existing population of neurons that were ischemic secondary to the cerebral hypoperfusion.

Comprehensive Neuro-Programming in a Residential Context

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In February 2014, the Cooper Riis Healing Community, a non-profit mental health facility in Asheville, NC, began offering its residents the opportunity to participate in what may be the most comprehensive neuromodulation and assessment program offered anywhere in the world within the context of a residential or inpatient setting. On-site qEEG assessments (using Deymed hardware and Neuroguide software) and computerized neuropsychological testing, various forms of neurofeedback (including Neuroguide LORETA and 19-channel Surface Z-score Training, Neuroguide Brainsurfer, EEG-driven audio-visual stimulation combined with neurofeedback, TAG Synchrony training, standard neurofeedback using Bio explorer and Deymed software and nirHEG), several different biofeedback modalities, as well as neurostimulation approaches such as CES, tDCS and AVS, are all part of the CooperRiis Neuroenhancement Program (CNEP). In addition, residents are encouraged to use BrainHQ computerized cognitive training exercises as well as to receive instruction in breathing techniques and mindfulness practices. Participants typically start with between 2 and 4 neurofeedback training sessions per week, in addition to ongoing opportunities to use CES, BrainHQ, and personalized breathing and mindfulness practice.

Our hypothesis is that this approach, offered within the context of a holistic program that encourages good nutrition, exercise, community involvement, as well as “standard” individual and group psychotherapeutic approaches, is uniquely suited to promote rapid healing, because positive neuroplastic change is encouraged in a comprehensive and integrated manner. We will examine the role a neuromodulation program plays within an already progressive model, how we promote it and educate other clinicians about its utility. Moreover, the unique advantages and challenges of being an integral part of a larger treatment team including psychiatrists, therapists, social workers, nutritionists, wellness coaches, and others, will be explored. We will discuss how we conceptualize utilizing nutrition to optimize brain training.

We will explain how we use each of the neuromodulation modalities in conjunction with each other, how we decide what to use, and how we monitor progress. We will discuss our impressions of the use of LORETA and surface z-score training combined with more

"traditional" neurofeedback protocols, and how they can be used complementarily. Finally, we will present brief case summaries and an overview of both the clinical successes and failures to date. Specifically, we plan to present a case of possibly undiagnosed TBI and how we used many of the approaches mentioned above (HEG, qEEG-informed use of tDCS, CES, "standard" qEEG-informed bioexplorer neurofeedback, HRV training and BrainHQ training) and achieved remarkable results (as measured by follow-up qEEG and neuropsychological testing) in 4 weeks of training. We also hope to present cases featuring the combined use of qEEG-informed neurofeedback and tDCS to help with the challenges inherent in thought disorders such as schizophrenia.

Connectivity in Adult Attention-Deficit/Hyperactivity Disorder

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Objectives. Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder characterized by symptoms of inattention, impulsivity, and hyperactivity that are observed across the lifespan. Despite the misconception that most children with ADHD mature out of the disorder, 30–65% take their symptoms into adulthood (Faraone, Biederman, & Mick, 2006). Deviant electroencephalographic (EEG) patterns of activity have been repeatedly observed in adults with ADHD during resting-state conditions (Bresnahan & Barry, 2002; Bresnahan, Barry, Clarke, & Johnstone et al., 2006; Clarke et al., 2008; Hermens et al., 2004; Koehler et al, 2009; Loo et al., 2009; Woltering, Jung, Liu, & Tannock, 2012). However, the investigation of coherence and connectivity differences in adult populations is limited. Clarke et al. (2008) was the first to investigate pairwise coherence differences among male adults with ADHD and age- and gender-matched controls, during an eyes-closed condition. The study findings revealed reduced hemispheric differences in the delta band at long inter-electrode distances and reduced alpha coherence at short-medium inter-electrode distances among ADHD participants. Further research is needed to replicate these findings and address the limitations of the initial study. In addition to addressing the issues of a small sample size, lack of female participants, and lack of an eyes-open condition, the current study will also investigate the known limitations of using pairwise coherence measurements (for review see, Coben, Mohammad-Rezazadeh, & Cannon, 2014).

Methods. Continuous 21-channel EEG, in accordance with the International 10-20 System, was acquired from 46 adult participants (27 male, male-to-female ratio, 1.4:1.0) with DSM-IV defined ADHD and 46 age- and gender-matched healthy controls. For each frequency band (delta, theta, alpha, beta) and condition (eyes-closed and eyes-open), a mixed ANOVA was used to examine between group differences in coherence calculated with pairwise (per Clarke et al., 2008) and multivariate (Coben et al., 2014) approaches. Partial correlation coefficients were calculated between ADHD behavioral measures and coherence data.

Results. This investigation is currently in progress. The most current data will be presented at the time of the ISNR conferences.

Conclusion. This study is the first to investigate EEG coherence differences in a mixed gender adult ADHD sample during eyes-closed and eyes-open conditions using pairwise and

multivariate coherence estimates. Treatment implications, study limitations, and future directions in research will be addressed.

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Accelerated Recovery from Traumatic Brain Injury (TBI) with Z-score Neurofeedback Therapy

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Multi-case study. Forty patients diagnosed with mild to severe TBI were subjected to Z-score LORETA neurofeedback (NFB) therapy in our practice since 2011. Most of patients were diagnosed with mild TBI (mTBI) and were treated within the first year after their brain injury. Few patients were diagnosed with more severe TBI and were subjected to delayed outpatient NFB therapy (more than one year since their TBI). Most of the patients complained of headaches and cognitive problems while some also suffered from dizziness and overlapping depression. Those who complained of cognitive problems were subjected to analysis with computerized cognitive testing (Neurotrax, Inc) before and after 10 sessions of NFB. During NFB therapy a subjective response from patients was collected to discern whether or not there was an improvement of symptoms. In addition, QEEG maps were obtained before each NFB session initiation in order to see an objective improvement of QEEG abnormalities. Subsequent analysis revealed that all patients (100%) noticed subjective improvement of their symptoms during 10 sessions of NFB therapy, out of which most of them reported initial improvement after only 1 to 3 sessions. Thirty-five patients also had an objective improvement (87%) of QEEG maps manifesting as reduction of excessive beta activity and/or normalization of delta or theta power. Fifteen patients completed pre- and post-NFB cognitive testing with 14 patients (93%) having significant cognitive enhancement (Global Cognitive Score increased 3–23 points with 12 points on average) after 10 sessions of NFB therapy. When compared to another cognitive enhancement group from our practice (consisting mostly of ADD patients) the TBI group resulted in even greater cognitive recovery. These results are very encouraging and indicate high potential of Z-score LORETA NFB in rehabilitation of patients suffering from TBI.

Rocking the Brain: Bi-directional Training as a Strategy to Facilitate Progress in Neurofeedback Training

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Brain training traditionally has focused heavily on training to inhibit or reward amplitude at certain frequencies or level of synchrony between two sites at a frequency. Our experience with multiple-clinician supervision groups is that certain types of such training (e.g., down-training eyes-open alpha or high-coherence fast frequency activity) can be extremely difficult for the client to achieve. Resistance to releasing an established pattern can slow or even block results.

Nearly two decades ago we discovered, somewhat by accident, that training such a pattern in the “wrong” direction first, then reversing the direction of training—as one would “rock” a stuck automobile to get out of a snowdrift or mud—can have a very positive effect.

During the last 2 years we have implemented “rocking” approaches to training amplitude, synchrony, and nIR HEG with impressive results.

1. Training designs for coherence work—two- and four-channel—allow the trainer to shift from increasing to reducing coherence at the click of a button in the software.
2. Training designs for amplitude also allow the trainer to switch direction in a matter of moments.
3. The LIFE game, developed for use with nIR HEG training, actually trains the client to increase blood supply—then reduce it—in the PFC.

It appears that many brains move more easily in a direction to which they are already committed. Challenging a brain that is already excessively locked in a beta frequency to lock tighter is less threatening than asking it to unlock. When, after 2 minutes, the brain is asked to let go and move back to the state it normally maintains, it tends to do so easily and often "overshoots" on the low side. After 2 minutes of this approach, we reverse again, and push the brain back up for 2 minutes, then again nudge it back down. This, in a sense, builds a "momentum" that frequently breaks the longstanding pattern.

We have discovered that the same approach used in amplitude training (e.g., training UP theta when it is already high before reducing it) also has a positive effect—especially in the first 1 to 3 sessions.

The LIFE nIR HEG game provides feedback for alternating between single-pointed focus (maximizing oxygenated blood flow) and open-focus states of pure consciousness. The client is trained in the first 2 to 4 sessions to learn to apply each state for a full session, and then he is trained to shift back and forth.

One of the great benefits of this approach has been the ability to use nIR HEG with OCD, anxiety, and other states where executive function may be excessive. It has also had the positive effect of minimizing over-training effects, allowing clients to train generally a minute longer per site than they normally would because of the "warm-down" period that follows the intense focus. We teach the client the same techniques we would use with HRV for shifting from closed to open focus.

The Use of a Neurofeedback Intervention in the Remediation of Neurophysiological Impairment Following Chemotherapy in Breast Cancer Patients

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Cognitive impairment—including declines in attention, learning, memory, and processing speed—is reported by as many as 50% to 70% of patients experiencing cancer chemotherapy (Dietrich, Monje, Wefel, & Meyers, 2008; Vardy & Tannock, 2007). This impairment does not appear to remediate over time (Koppelmans et al., 2012) and has no effective treatment. While neuroimaging has revealed organic damage (Saykin et al., 2003; Stemmer, Stears, Burton, Jones, & Simon, 1994), neurophysiological impairment has not been shown.

Neurofeedback has been shown to be helpful in improving cognitive performance (e.g., Monastra, Monastra, & George, 2002; Nelson & Esty, 2012). The present study was performed to determine what neurophysiological impairment can be shown in this population and if a neurofeedback intervention can normalize these impairments.

Nine breast cancer subjects, with significant cognitive impairment as demonstrated by neurocognitive testing, and at least 1-year post-chemotherapy, were given a neurofeedback training intervention. The intervention consisted of eighteen 30-minute sessions of neurofeedback at two training sites, C3 and C4 simultaneously and occurring three times per week. Pre- and post-training QEEGs were performed to determine initial neurophysiological functioning as well as improvement following training.

In examining the pre-training eyes closed EEG, we found that all nine subjects demonstrated abnormal spectral magnitude across five bands compared to healthy adults (SKIL normative database; $p < 0.05$, using a Z-score comparison of the individual to the normative data base; *t*-distribution comparison). Bands were 2–4, 4–8, 8–13, 13–34, and 34–39 Hz, respectively. Excessive (two or more standard deviations from norm) delta activity was shown by 4 of the 9, and 7 showed deficit theta activity. I n addition, 6 of the 9 also demonstrated excessive gamma activity.

We compared integer frequency bands (e.g., 2 Hz, 3 Hz, 4 Hz) pre-training to post-training eyes-closed QEEG spectral magnitudes. All comparisons were between an individual and the database norm with two or more Z-score deviations from normative values indicating abnormality. Of the 9 subjects, 7 showed significant normalization of band activity, and the remaining 2 showed significant compensatory changes.

Average improvement across subjects was 40%, meaning that 40% of the abnormal frequency bands were in normal range after training. Half of the individuals who normalized, normalized completely for the entire frequency band; whereas the other half normalized for half of the range of the band, on average. Of the 4 subjects with excessive delta wave activity prior to training, 3 normalized in delta activity; and of the 7 subjects with diminished theta activity, 3 normalized in theta activity. Out of the 6 subjects with gamma excesses, 2 significantly improved (normalized) but 2 showed more excessive gamma activity after training. Improvement of symptoms such as anxiety and sleep will also be presented. Positive neurocognitive testing results will be presented separately.

Conclusions. Pre-training baselines demonstrated significant neurophysiological impairment. This impairment, particularly elevated delta frequency band, is consistent with cognitive deficits. The neurofeedback intervention produced significant improvements in the direction of normalization of the EEG. Discussion will include implications of using a standardized neurofeedback protocol, and suggestions for study replication, facilitated by specific protocol parameters.

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Functional Connectivity, Diffusion Tensor Imaging (DTI) AND LORETA Coherence, Phase and Co-Modulation

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Objectives. The purpose of this study was to explore the relationship between diffusion tensor imaging (DTI) measured cortico-cortical 'U' shaped fibers and association fasciculi and electroencephalogram (EEG) source localization using LORETA. Another objective was to link the Talaraich Atlas spacings measured using DTI and the observed spacings between maxima and minima of cross-correlations and coherence of LORETA derived EEG time series.

Methods. The EEG was recorded from 19 scalp locations from 71 subjects ranging in age from 13 years to 16 years in the eyes-open and eyes-closed conditions. LORETA time series from 88 Brodmann areas was computed. Coherence between the within hemisphere Brodmann areas and homologous Brodmann areas was computed with special emphasis on phase differences that demonstrated network functional connectivity and not volume conduction. Similarly, the Pearson product correlation was computed between current density produced by Brodmann areas located within each hemisphere as well as hemispheric homologous Brodmann areas. Spatial heterogeneity between Brodmann areas cannot be

accounted for based on volume conduction and the comparative consistency between coherence and co-modulation of current sources was evaluated.

Results. An spatial pattern of increasing and decreasing coherence and correlations were present with a spacing of approximately 2–3 cm in registration with the 'U' shaped fibers as measured by DTI (Catani & de Shotten, 2012). Co-registration with longer distant cortical fasciculi were also present, especially in the frontal-occipital and frontal-temporal fasciculi. The presence of long phase differences and spatial heterogeneity that corresponds to the DTI Atlas heterogeneity shows that the results are due to network connectivity and not volume conduction.

Conclusions. There is reasonable cross-validation of the spatial distance between LORETA functional connectivity and the 'U' shaped fibers and association fasciculi. This is important because it emphasizes the underlying covariance between the wiring diagrams of the brain and the EEG sources that are dependent on cortico-cortical connections between Brodmann areas. DTI fiber connection location and heterogeneity is gives rise to different conduction velocities and different densities of connections between Brodmann areas. The linkage of the underlying wiring patterns of the brain by DTI and EEG LORETA provides a richer view of the EEG than if this linkage was not demonstrated. The objective is to learn how one can validate the spatial structure of the human EEG based on the density and distance of connections between Brodmann areas

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Enhancing Your Practice to Work with Military Personnel

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Because of the increased demands on the military since 11 September 2001, many of us have and will treat current and former military members, providing clinical and peak performance services. The military culture with its rapid deployment cycles, high potential for traumatic brain injury (TBI) and post-traumatic stress disorder (PTSD), unique family dynamics, and stressful work environments require all providers working with military members to be well informed to be effective. Without knowledge of these areas, the provider risks making errors that may alienate or even harm military members.

This presentation will help providers be more confident in working with military members by providing a practical understanding of the unique aspects of military culture and exposing them to clinical techniques that are effective in these populations.

The presentation will consist of two parts. In the first part, focus will be placed on the cultural aspects of working with the military, relevant to effective treatment of its members. Emphasis will be given to aspects of the military work environment, subtle but highly relevant stressors of deployment, unusual diagnostic considerations for military members, and the

characteristics of family dynamics. During the second part, findings from EEG, ERPs, and ECG in PTSD and mTBI cases, as well the effective use of Brodmann area and surface amplitude neurofeedback with these patients will be discussed. Particular emphasis will be give on how the multifactorial nature of cognitive impairment and peak performance necessitates rapid, inexpensive, and easily applied multimodal analysis methods that can offer greater sensitivity and specificity for typical military cases. Case analysis will emphasize ease-of-use and accessibility for a clinical practice or peak performance program.

Case presentations, pictures from the deployed and "in garrison" military environments will be used to make this presentation applicable, and this often misunderstood portion of the population comprehensible, thus increasing competence in providers working with this population. Understand better how to work with military members in a clinical and peak performance practice settings. Deployment stressors and their effects.

LORETA Neurofeedback in Precuneus for Substance Use Disorders

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Introduction. Substance use disorders continue to exact substantial negative effects on society and the individual. This clinical study was conducted in an outpatient program. LORETA neurofeedback was utilized to target alpha current source density in a three-voxel cluster of neurons in the left precuneus. Patients completed between 3 and 5 sessions per week and 17 sessions total.

Methods. This study was conducted with 20 individuals, 10 male, mean age of 27.17. All patients were diagnosed with a substance use disorder including heterogeneous comorbid syndromes including depression, anxiety and attention-deficit/hyperactivity disorder. Outcomes were evaluated using neurophysiological and neuropsychological assessments. Clients were given pre-post testing assessments including the Integrated Visual and Auditory Continuous Performance Test (IVA+/CPT), Verbal Fluency and Color Word Interference Tasks from the Delis-Kaplan Executive Function System (DKEFS), Personality Assessment Inventory (PAI), Pittsburgh Sleep Quality Index (PSIQ) and a Brief Substance Craving Scale (BSCS).

Results. LORETA current source increased significantly at the region of training (ROT) as well as in specific nodes shown to be associated with reward and addiction (e.g., right insula, medial prefrontal, and parietal regions). The default and salience networks also show significant shifts in both CSD levels and connectivity. Attention and executive function scales improved significantly or in the desired direction post-training, with the clinical scales on the PAI showing dramatic and significant reductions. There was notable increase in sleep improvement and reductions in cravings.

Discussion. Data are still being collected and analyzed; however, perceptible post-training changes are expected to increase across sample size. Treatment of SUD shows mixed results concerning outcomes and pre- and posttreatment measures. LORETA neurofeedback may provide an important mechanism for treatment of SUD in conjunction with other therapeutic methods. The authors will discuss these complimentary methods in

terms of self-perception, neural plasticity, and efficiency and the importance of brain-based treatment for addictive disorders.

Investigating Efficacy of 19-Channel Z-Score Neurofeedback in a Clinical Setting

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Objectives. In recent years new neurofeedback (NF) models have emerged based on the incorporation of live normative database derived z-scores, termed z-score NF (ZNF) (Collura, Thatcher, Smith, Lambos, & Stark, 2009; Thatcher, 2012). Among these, the surface montage 19-channel z-score neurofeedback (19ZNF) is a new NF model. Yet, peer-review literature lacks research using quantitative-methodologies, with group means statistical analysis of outcome measures, to evaluate 19ZNF. Currently, case studies (Hallman, 2012; Koberda, Moses, Koberda, & Koberda, 2012) and descriptive/clinical reviews (Thatcher, 2013; Wigton, 2013) make up the majority of peer-reviewed 19ZNF literature. The purpose of this quantitative study was to investigate the efficacy of 19ZNF, in a clinical setting, using archival data, with statistical analysis of group means data. This study also proposes an original method for measuring overall normalization of QEEG z-scores suitable for group means research designs.

Methods. This study implemented a retrospective one-group pretest-posttest design, and used outcome measures of QEEG z-scores and the clinical assessments of the Integrated Visual and Auditory continuous performance test (IVA), the Behavior Rating Inventory of Executive Functioning (BRIEF), and the Devereux Scale of Mental Disorders (DSMD). Each outcome measure framed a group such that 19ZNF was evaluated as related to electrocortical functioning (n = 21), as well as the neuropsychological constructs of attention (n = 10), executive function (n = 12), and behavior (n = 14). Thus, the four research questions were as follows: Does 19ZNF improve (1) electrocortical function as measured by QEEG z-scores, (2) attention as measured by the IVA, (3) executive function as measured by the BRIEF, and (4) behavior as measured by the DSMD? One-tailed t-tests were performed to compare the pre-post scores for the selected QEEG metrics and the included scales of the clinical assessments.

Results. The direction of change, for all pre-post comparisons, was in the predicted direction. For all clinical assessments and z-scores, the group means were beyond the clinically significant threshold before 19ZNF, and no longer clinically significant after 19ZNF. The pre- to post-difference for all scales/metrics for all outcome measures were statistically significant, with results ranging from p = .000 to p = .008; and effect sizes ranging from 1.29 to 3.42. In general, the number of session for all groups was approximately 10 (ranging from 9.70 to 11.83 sessions) at a session frequency of on average of once per week.

Conclusions. Results suggest 19ZNF improved electrocortical function, attention, executive function, and behavior. Moreover, these findings support 19ZNF as having potential to bring about both QEEG normalization and symptom improvement. While clearly more research is needed, this study provides beginning evidence of the efficacy of 19ZNF. Additionally, this study supports prior clinical reports (Thatcher, 2013; Wigton, 2013) that 19ZNF results in improvement of clinical symptoms in fewer sessions than traditional NF. In sum, this

research adds to what is known about 19ZNF and offers a methodology for using QEEG metrics as outcome measures. Based on the content of this presentation, the participant will be able to understand the parameters of the efficacy of 19ZNF, within the context of this study, regarding how 19ZNF improved electrocortical function, attention, executive function, and behavior.

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Training Attention, Working Memory, and Information Processing Speed Using 3D-MOT

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3D-MOT is a perceptual-cognitive training system based on multiple object tracking (MOT) in a three-dimensional (3D) virtual environment. The current presentation will discuss 3D-MOT as an adjunct to neurofeedback intervention for healthy individuals looking to improve cognitive performance, individuals with attention deficits, individuals recuperating from head trauma, high-level athletes, and aging populations.

To this end, current research will be presented that shows that 3D-MOT training can enhance attention, working memory and visual information processing speed in healthy university

students; correlates of brain functions are also assessed using quantitative electroencephalography (Parsons et al., in press). Further research demonstrates attention improvements in school-aged children with attention deficits (Parsons, Bates, & Faubert, in press). Research currently underway has demonstrated the potential of using 3D-MOT has a rehabilitation tool for those suffering from head trauma (Romeas et al., in press). Research has also shown a strong link between athletic performance levels and learning capacity on this task (Faubert & Sidebottom, 2012; Faubert, 2013). Finally, research has demonstrated transfer of this training to socially relevant tasks in aging populations (Legault & Faubert, 2012; Legault, Allard, & Faubert, 2013).

As a clinical tool, 3D-MOT offers relatively faster gains than neurofeedback; yet research into the longevity of training effects is currently lacking. Neurofeedback studies, however, have demonstrated the long-term retention of training effects. Combined, the two methods offer the best of both worlds: quick gains and long-term consolidation. A 3D-MOT session takes approximately 8 minutes to administer, and thus the length of a typical neurofeedback session would not have to be significantly lengthened.

Further, a novel research study used a hybrid, integrated EEG-based neurofeedback intervention and demonstrated enhanced cognitive functions similar to the study of Parsons and colleagues (in press) as well as augmented learning within the task in healthy university students (Parsons & Faubert, in press).

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The Combined Effects of Neurofeedback and Biofeedback Training for Treating Children with Autism Spectrum Disorder

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Autism Spectrum Disorder (ASD) is characterized by deficits in social communication and restricted, repetitive behaviors. Research suggests that the Mirror Neuron System (MNS), which is thought to mediate empathic behavior, may be dysfunctionally operating in this population, thus contributing to some of these deficits. Neurofeedback Training (NFT) has

been able to improve MNS activity by targeting mu rhythms (8–13 Hz oscillations over the sensorimotor cortex). Peripheral physiological activity in ASD has received less research attention, yet also shows deficits and potential for clinical intervention. Heart Rate Variability (HRV) vagal activity is a key example of this. Moreover, these physiological mechanisms have been linked to social behavior and are believed to be highly integrated with central, neurological activity. An approach that targets this system as a whole could have a greater impact on behavior. This presentation will discuss a clinical intervention that uses a combination of NFT and HRV biofeedback to improve symptoms of autism. The study is currently taking place and is projected to be completed by September, 2014. Of the 24 participants, each will either receive (a) HRV Biofeedback only (Group 1), or (b) HRV Biofeedback and NFT (Group 2). Both groups will first learn how to diaphragmatic breathe, which is pivotal for conducting HRV Biofeedback. Then, one group will continue to receive HRV Biofeedback (through operant reinforcement of a DVD movie), while the other receives HRV Biofeedback and NFT (also through operant reinforcement of a DVD). It is hypothesized that Group 1 will show positive changes in HRV, vagal tone, and social behavior. It is hypothesized that Group 2 will show positive changes in HRV, vagal tone, mu suppression, long-range neural connectivity, and social behavior. Outcomes will be assessed through EEG and magnetoencephalographic (MEG) scans, physiological measurement (e.g., HRV and vagal tone), and behavioral questionnaires. This presentation aims to convey the basic neurological and physiological mechanisms thought to underlie autism and social behavior, and potential avenues to improve these mechanisms. Learn about the integration of biofeedback and neurofeedback.

Insomnia in Returning Service Members an Early Intervention in Combat Stress

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Introduction. Disrupted sleep, mood disorder and performance deficits have been observed among service members returning from combat. Insomnia is a crucial symptom in the post-traumatic stress disorder (PTSD) cluster (Maker, Rego, & Asnis, 2006) and efforts to quickly address disrupted sleep in newly returning soldiers may enhance amelioration of the emergence of PTSD. EEG Biofeedback has been demonstrated to be of value in the treatment of PTSD symptoms (Peniston & Kulkosky, 1991; Peniston, Marrinan, Deming, & Kulkosky, 1993) but to date is not well established as a treatment option for active duty soldiers.

A program evaluation was designed to assess the effectiveness of insomnia focused CBT/CPT group psychotherapy in conjunction with EEG (neurotherapy). This pilot study was designed to further evaluate the EEG/QEEG, Continuous Performance and self-report measures pre- and post-initial intervention at an Embedded Behavioral Health unit with newly returning combat Infantry soldiers.

Methods. Twenty-eight service members returning from deployment in 2013 with combat stress symptoms were recruited for a program evaluation as part of routine outpatient care at an Embedded Behavioral Health clinic at Fort Bliss, Texas. All service members in the evaluation received group psychotherapy in an intensive workshop format. The insomnia workshop consisted of four weekly 120-minute sessions. The initial two sessions of this workshop consisted of CBT Stimulus Pairing training and cognitive re-appraisals about

insomnia. The remaining two sessions of this workshop focused on CBT methods to address nightmares in accordance with research (Davis & Wright, 2005).

After completion of the workshops, service members could elect a course of 20 sessions of EEG assisted biofeedback. Those who elected this modality received an analog/QEEG (digital) 19-channel baseline with attentional testing at initiation of biofeedback along with standard clinic measures PCL-M, GAD, and PHQ pre- and post-EEG biofeedback treatment. Participants were assigned to 1 of 3 EEG Biofeedback systems that included either the Brainpaint, LENS (Low Energy Neurotherapy System), or a task activated EEG Biofeedback program developed by Marvin Sams, ND. All participants completed weekly sleep logs and reported changes in medication utilization and tobacco/alcohol use.

Results. An analysis of outcome measures was performed for all members who completed self-report inventories after 20 EEG biofeedback sessions. Data analysis of the IVA continuous performance measures demonstrated statistical significance on pre vs. post measures of the Full Scale Attention Quotient as well as the Visual Attention Quotient. All means scores of each of the IVA variables improved from the average pretest scores. Data analysis of the EEG QEEGs is currently underway, as is clinic follow-up on the treated service members and those that dropped out of treatment during the course of the evaluation.

Conclusions. Disrupted sleep is postulated as an early marker of combat related stress disorders (PTSD). The authors have sought a pilot program to assist with objective measures to enhance treatment outcomes in service members at risk to develop PTSD. It is hoped that this initial pilot study may provide a stimulus for further research that demonstrates that timely rapid and effective relief of symptoms will improve outcomes. Introduce treatment outcomes for interventions for Insomnia in recently redeployed combat soldiers.

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Gamma ERS/ERD Development Cycles of Abstractions in Math Learning

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The present study describes an event-related synchronization and desynchronization (ERS/ERD) in the gamma band (31–50Hz) induced by the onset of abstractions in math learning stimuli. Three levels of tasks in abstractions are presented with spontaneous and comprehensions conditions in which abstract mappings (A2) with abstract systems (A3) and systems of abstract systems (A4) were held in math learning. The tasks are all based on the four mathematical operations of addition, subtraction, multiplication, and division. Dynamic growth models show the complex, interrelated changes that take place during brain growth, cognitive development, and learning (Fischer, Bernstein, & Immordino-Yang, 2007). Math learning requires flexibility to adapt existing brain function in selecting new neurophysiological activities to learn desired knowledge. Flexibility exists over multiple brainwave scales as performance of a skill change from slow to fast and automatic learning. This study investigated the brainwave structure and connectivity by identifying dynamic changes of brain modularity. This research shows the complex network of interactions, convergent and divergent paths, nested cycles, stabilities and instabilities, progressions and regressions, clusters of discontinuities, and stable levels of performance (Fischer et al., 2007). It represents the relationships between brainwave activities and cycles of cognitive performance, which becomes most visible with optimal functioning of the cognitive system when the effective supports a student's performance. A series of discontinuities in optimal cognitive growth defines an abstract level scale. Brain development characteristics—such as number of neurons and synapses, brain mass, myelination, and brain activity—change by the cycles of cognitive performance. The brain changes by the simultaneous cognitive and emotional factors, in parallel with the individual difference and age. This study neurologically proves the framework of the dynamic systems theory for analyzing complex patterns of the spurts and drops of the development of abstractions. Neurologically proved complexity scale provides a useful ruler to track the individual differences in learning a specific task for math learning. The participant will be able to understand the event-related synchronization and desynchronization (ERS/ERD) in the gamma band (31–50 Hz) induced by the onset of abstractions in math learning stimuli.

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On the Relationship Between EEG Phase Reset in the Time Domain and EEG Power in the Frequency Domain

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Objectives. The purpose of this study was to explore the relationship between EEG phase reset in the time domain and EEG absolute power in the frequency domain. Of special interest was how a synchronous group of neurons in one frequency band correlates with power in the same and different frequency bands.

Methods. The electroencephalogram (EEG) was recorded from 19 scalp locations from 326 subjects ranging in age from 5 years to 17.6 years in the eyes open and eyes closed conditions. Complex demodulation was used to compute instantaneous EEG absolute power in the frequency domain (delta, theta, alpha, beta1, beta2, beta3 & hi-beta). The first derivative of straightened phase differences for all electrode combinations for each frequency band and then phase shift duration and phase lock duration was computed between pairs of electrodes in the time domain. Correlations were then computed between EEG power and the duration of phase shift and phase lock in different frequency bands. Cross-frequency correlations were also computed to compare amplitude and phase reset relations across frequency bands.

Results. Positive correlations were present between phase shift and lock duration and absolute power in all frequency bands except the delta (1–4 Hz) frequency band. Inverse or competitive relationships were present between alpha1 (8–10 Hz) and alpha2 (10–12 Hz) and theta (4–7 Hz) frequency bands and between beta1 (12–15 Hz) and alpha (8–12 Hz) as well as between alpha1 (8–10 Hz) and alpha2 (10–12 Hz) frequency bands. Positive correlations were present between theta and beta1 frequency bands as well as between alpha2 and beta3 (18–25 Hz) frequency bands.

Conclusions. The spontaneous EEG is the summation of phase shifting and phase locking of competitive thalamocortical pacemaker systems. Cross-frequency coupling occurs by reciprocal inhibition of competing frequency resonance bands of synchronized groups of neurons. Learn how groups of neurons that synchronize in one frequency band affect EEG power in different frequency bands. Learn about cooperation and competition between groups of synchronous neurons in different frequency bands.

QEEG and Neurofeedback for Elementary Students with ADHD Inattentive Subtype

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Beginning with the first study on the use of neurofeedback (NF) as an intervention for attention-deficit disorder (ADD) and attention-deficit/hyperactivity disorder (ADHD; Lubar & Shouse, 1976), improvements in school performance and positive outcomes on cognitive and academic measures have been consistently reported (Linden, Habib, & Radojevic, 1996; Lubar, 1991; Thompson & Thompson, 1998; Thornton, 2000). Research has demonstrated that NF is most efficacious for ameliorating symptoms of inattention (Arns, de Ridder, Strehl, Breteler, & Coenen, 2009; Monastra, Monastra, & George, 2002). Studies provide evidence that inattention is associated with reduced processing speed on school-related tasks (Weiler, Bernstein, Bellinger, & Waber, 2000) and impaired processing of visual information (Barkley, Grodzinsky, & DuPaul, 1992; Swanson, Posner, Potkin, & Bonforte, 1991). Willcutt and Pennington (2000) found that the relationship between ADHD and reading disorders (RD) was stronger for students with symptoms of inattention than hyperactivity/impulsivity. A recent review of the literature (Sexton, Gelhorn, Bell, & Classi, 2012) suggests that ADHD and RD are not just frequently associated comorbid conditions but are, indeed, co-occurring conditions that result in significant deleterious and chronic impairments that begin in childhood and extent into adulthood.

The extant literature on NF now lends support that it is an efficacious intervention for ameliorating symptoms of ADHD (Arns et al., 2009; La Marca, 2011). This is evidenced by the recent decision of the American Academy of Pediatrics to state that the research on biofeedback (including neurofeedback) has met criteria to justify their highest level of support (Level 1: Best Support) as an evidenced-based psychosocial intervention for attention and hyperactivity (American Academy of Pediatrics, 2012). However, there remains a veritable dearth of research that specifically examines the use of NF to directly address academic achievement; the literature deals primarily with symptoms of attention.

This presentation will examine some of the findings of the first study to directly investigate the impact of NF on measures of reading achievement (La Marca, 2014). An examination of pre- and post-intervention QEEG and IVA CPT data will be presented. The role of theta/beta ratio reduction, particularly as it applies to addressing symptoms of inattention, will also be discussed.

In addition, we will review the history of using QEEG Ratio/Scans and QEEG Brain Mapping to assist in the diagnosis of ADHD and subtypes of ADHD. We will present different types of QEEG measures, such as theta/beta and theta/high beta ratios that differential inattentive ADHD from hyperactivity/impulsivity. Furthermore, we will present data that indicates that certain QEEG measures and ratios are more sensitive to the effects of neurofeedback on measures of attention and reading achievement.

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Early Cognitive Decline and Alzheimer's Disease: Detection and Intervention Using sLORETA Z-scored Imaging

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This presentation will review the history of MRI research related to detecting early cognitive decline and Alzheimer's disease, compare and contrast MRI and sLORETA Z-scores as imaging techniques, and discuss potential applications of sLORETA Z-score imaging technology to both the categories of early detection and intervention with regard to cognitive decline and Alzheimer's disease in aging populations.

An increasing body of research on Alzheimer's disease and early cognitive decline using magnetic resonance imaging (MRI) has pinpointed the hippocampus and parahippocampal gyrus as the brain regions most likely to show functional abnormalities in the initial stages of cognitive decline.

Early MRI studies demonstrated a significant deficit in the volume of the parahippocampal gyrus in subjects with aging-associated cognitive decline. Compared with cognitively intact subjects, subjects with aging-associated cognitive decline had a significantly smaller mean volume of the right parahippocampal gyrus. Additional research indicates the significant association between parahippocampal gyrus volumes and severity of cognitive deficits in aging-associated cognitive decline groups. This finding is in line with the view that pathological alterations in the parahippocampal gyrus, leading to a disconnection of the hippocampus from neocortical association areas, represent the neuroanatomical correlate of cognitive dysfunction in early dementia.

Consistent, although much more pronounced, changes were found in Alzheimer's disease patients, indicating that, with respect to parahippocampal gyrus volume, the subjects with aging-associated cognitive decline took an intermediate position between the Alzheimer's disease patients and the cognitively intact comparison subjects.

Neuropathological evidence supports the premise that the earliest changes in Alzheimer's disease selectively affect the parahippocampal regions of the brain. Studies conducted to determine if otherwise healthy elderly subjects with mild cognitive impairment had structural volume deficits affecting the parahippocampal gyrus resulted in findings indicating that parahippocampal atrophy underlies the observed cognitive deficits in aging-associated cognitive decline. These findings corroborate the hypothesis that aging-associated cognitive decline represents a preclinical stage of Alzheimer's disease.

Standardized low-resolution brain electromagnetic tomography (sLORETA) uses surface EEG readings to project a three-dimensional cortical image composed of 6,239 voxels approximately 5 millimeters in size. Each voxel is converted instantaneously into a current source density, and three spatial values (vectors) that describe the dipole. Thus, each voxel represents an "analog" number, the local level of activity. This is done in 16 frequency bands simultaneously, in real time.

All projected signals are time-domain signals, providing superior speed and accuracy compared to FFT-based approaches, which introduce delays and possible distortion, due to the epoch size limitations. FFT-based methods also show only averaged activity, not instantaneous values. The software computes the sLORETA projection instantaneously using high-speed time-domain methods, and accurately shows the momentary changes in EEG signals, in real time.

sLORETA has been extensively validated against MRI with regard to the accuracy of its localization methods, and can be used to not only evaluate parahippocampal activity relative to age-referenced normative values, but also to provide feedback to the brain based exclusively on activation patterns observed in the parahippocampal gyrus. Evaluate emerging trends in sLORETA imaging technology in the field of neurofeedback, with regard to applications potentially assessing and treating elderly populations exhibiting signs of early cognitive decline and preclinical Alzheimer's disease.

Four-Channel Multivariate Coherence Training: Rationale and Findings

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Coherence training has been used as a form of neurofeedback for over 10 years. Empirical findings have shown it to be effective in the treatment or management of Traumatic Brain Injury (Walker, Norman, & Weber, 2002), Autism Spectrum Disorder (Coben & Padolsky, 2007), Learning Disabilities (Nazari, Mosanezhad, Hashemi, & Jahan, 2012), and potentially many more conditions including Epilepsy (Coben, Wyckoff, & Hudspeth, in press). However, all such coherence training is based on training coherence in pairs (sets of two electrode signals).

There is considerable research over the past decade to suggest that measuring coherence in pairs can lead to spurious errors and findings (Blinowska, 2011). Rather, there are forms of advanced multivariate statistical methods that can be used to calculate coherence that are more accurate and precise in depicting such relationships and even their directionality. In fact, one study has shown that using pairwise estimates can lead to up to 50% more errors than multivariate approaches. Given these findings, one wonders how this might impact neurofeedback training, as this has been the only way coherence training has ever been done. More so, could we develop another approach that would lead to even more effective treatments?

We developed a new methodology for performing coherence training that uses four channels of EEG data and bases calculations of coherence on multivariate methods. In this approach, four sensor locations are selected based on QEEG data and ongoing calculations are made such that each location is the average coherence between that electrode and the three others, and the same is done for each location. A summed coherence value is then derived that represents the average of all the averages and is considered to average multivariate coherence value of all channels. It is our belief that calculating and feeding back multivariate coherence estimates to the client will lead to more accurate information and enhance efficacy.

To test this hypothesis we designed a research program that has used this approach for more than 200 patients. Of these patients, more than 75 had also previously had two-channel coherence training and served as their own controls. We have then compared the effects of these trainings (two-channel coherence vs. four-channel multivariate coherence) on various clinical and EEG variables across multiple diagnoses. Preliminary analyses of a smaller cohort has already shown significant differences such that four-channel training leads to greater coherence and power changes in about half the number of sessions.

Our goal during this presentation is to present this methodology, study findings, and discuss the ramifications of how this can enhance efficacy in neurofeedback training. This large database also enables us to discuss how these issues interface with different diagnoses, clinical setting, gender, age, and many other variables. The participant will learn about a new form of neurofeedback and how it compares to more traditional two-channel coherence training.

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The Internalization of Neurofeedback Training as Demonstrated by Repeated QEEGs

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In general, neurofeedback training can be described as the process of using high-tech computer equipment to measure the electrical patterns of the brain (brainwaves) and then provide instantaneous feedback about those patterns for the purpose of enabling an individual to gain some level of influence or cognitive control over them (Hammond, 2011). In essence it is a process of self-remediation of unwanted activity in the brain. Typically changes in brainwave patterns occur gradually over time and with frequent rehearsal the changes become more enduring.

According to this conceptualization of the neurofeedback process, the first objective would be to learn the new, more desirable pattern of brainwave responses through a process of trial and error by relying on the feedback (audio and/or visual) for guidance. Once the individual has learned the intended brainwave pattern, it then becomes a matter of repetition of that pattern so that it may be properly "imprinted" in the brain in order to have some lasting result. Somewhere between the learning phase and the rehearsal phase, it can hypothesized that there is an internalization of the intended brainwave pattern by the individual in training and that they could learn to produce this pattern, at will, even without the aid of the feedback. It would then be reasonable to assume that the repetition of this newly internalized pattern would have increasingly more and more influence over the resting brain that was not actively engaged in the neurofeedback training process at the time, thereby indicating a lasting effect of a reconditioned brain.

The purpose of this presentation is to provide empirical evidence of the internalization and remediation process that occurs during the course of neurofeedback training and treatment. Using multiple case examples and creative applications of Quantitative Electroencephalography (QEEG) analysis, this process can be illustrated in stages as they occur, providing a vivid cross-section of the changing brain showing both the internalization of normalized EEG patterns and the global improvements that occur along the way as compared to the resting state baseline QEEG measures. The clinical data presented will serve to negate any possible influence of the placebo effect as well as other therapeutic implications for accelerating treatment regimens and increasing efficacy of therapeutic outcomes from neurofeedback training efforts. One objective of this standing presentation is to demonstrate to the audience a creative manner of using QEEG for diagnostic purposes that may not have been commonly thought of before.

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Offering Hope: The Efficacy of Neuroptimal Training in the Treatment of Anxiety and Depression

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Kingston Institute of Psychotherapy and Neurofeedback was established in 2010 with the goal of making EEG-neurofeedback accessible in the Kingston community. Kingston is a small city with a large military base and is home to many large penitentiaries. Not surprisingly, there is a strong need for mental health care directed toward treatment for depression, anxiety, substance dependence, and post-traumatic stress disorder (PTSD). Neuroptimal is the mainstay of treatment at this multi-system clinic, which treats over 100 patients daily. The clinic also uses qEEG, targeted EEG, Hemo-encephalography, BAUD and Alpha-Stim. The 10 neurofeedback systems are supervised by two physicians.

Our patient population includes civilian, first responders, active serving military, and veterans of the Canadian Forces. Diagnostic groups routinely treated at the clinic include sleep disorders, PTSD, mood and anxiety, ADHD, and neurocognitive dysfunction. These patients invariably describe symptoms of anxiety and depression. For this reason, the clinic uses the Beck Depression Inventory (BDI) and the Beck Anxiety Inventory (BAI) to determine the effectiveness of intervention over time.

Study Design: This is a retrospective case series, which reviewed self-report outcome measures (BDI, BAI) at 0, 8, and 16 weeks. Patients were included only if they had no medication changes during this time period. Results for more than 200 patients demonstrate clear efficacy in decreasing disability symptoms of anxiety and depression. Results are analyzed based on:

- Symptom severity
- Age
- Gender
- Primary diagnosis

Data from this retrospective case series suggest that Neuroptimal neurofeedback can be used as a primary intervention to alleviate symptoms of depression and anxiety. Individuals who suffer from PTSD describe high levels of anxiety and struggle with feelings of depression. Neuroptimal training benefits our patients with PTSD by decreasing subjective symptoms of anxiety and depression within this group.

Effectiveness of Pulsed, Ultra Low Power Electric Current EEG Biofeedback in Treating Symptoms of Concussions in Former NFL Players

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Football related traumatic brain injury (TBI) is one of the signature sports injuries. Even one concussion can cause a lifetime of problems. TBI can result in a myriad of symptoms, including hypersensitivity, headaches, cognitive dysfunction, depression, and many more. Currently there is no adequate treatment. Little more than rest is generally prescribed. Residual symptoms can leave a person appearing outwardly normal but still have devastating consequences on the person and his family. While it has not yet been conclusively proven, it may be that repeated concussions lead to CTE, chronic traumatic encephalopathy. There have been numerous reports of mild/moderate TBI responding very well to this treatment. This has certainly been the experience of three Investigators on this project.

This project seeks to determine whether pulsed ultra-low power electric current EEG biofeedback is an effective treatment for symptoms of TBI. It is hypothesized, based on informal clinical observations, that this form of EEG biofeedback can have a significant and beneficial impact on problems associated with TBI such as anxiety, depression, sleep disorders, headaches, cognitive dysfunction, emotional ability, and other symptoms of TBI. This project will track across-session subjective symptoms ratings over the course of 20 sessions. There will also be pre- and post-testing using brain maps (qEEG), the Rivermead Post Concussion Questionnaire, the King-Devick test for cognitive functioning, a Sway/Balance for equilibrium and the ImPact test for cognitive function (memory, etc.). There will be follow-up testing 3 and 6 months after treatment has ended.

This study could be an important step toward validating a safe, effective, non-pharmaceutical intervention for a growing population of athletes who have sustained concussions and have residual symptoms. These symptoms can last a lifetime and have catastrophic effects on the athlete and his family. In addition, TBI resulting from repeated sports concussions is likely to not be so different from other causes, with the exception of blast injuries, which have their own particular mechanism of injury and pathophysiology. Verifying an effective treatment for this disorder would be a huge benefit to the approximately 1.7 million people each year who sustain a significant TBI. The ramifications of the study go far beyond treating athletes and even those in the military who are injured. Attendees will gain an understanding of the unique dynamics of reaching, evaluating, and treating former NFL players.

POSTER PRESENTATIONS

Lateralized Readiness Potential (LRP) as a Premotor Preparation Abnormality Biomarker in Autism and ADHD

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Background. Motor abnormalities in autism bear the characteristics of precedence and universality that characterize a core symptom. Underlying the motor skill deficits in autism is a dyspraxia that by definition is an inability to plan, organize, and execute movements in the absence of any known physical and/or neurological condition (Mostofsky et al., 2006). This deficit is more apparent in autism spectrum disorders (ASD) when compared to ADHD.

Objectives. The lateralized readiness potential (LRP) is an index of motor processes and it is assumed that this brain potential is generated by a source within the motor cortex (Leuthold, Sommer, & Ulrich, 2004). LRP reflects the response-specific involvement of the left and right cortices of the brain and enables the determination of the point in time at which the activation of the motor cortex controlling one hand surpasses the activation of the motor cortex controlling the other side (Eimer, 1998). The LRP is assumed to be related to selective response activation. It captures the asymmetric portion of the late Bereitschaftspotential preceding hand movements and helps to determine exact point in time when sensory information starts affect motor processing and execution.

Method. Our goal was to compare differences in LRP during a modification of a cued Posner spatial attention task between group of children with ASD, ADHD, and neurotypical (NT) children (N = 14/group, mean 14.9 years). Each subject participated in a Posner spatial attention task with congruent (75%) and incongruent (25%) trials with 128-channel EEG recording. We used stimulus-locked LRP method of waveform calculation and since S1–S2 interval was set on 1s computed mean LRP and integrated LRP values for two windows (early 600–800 ms post-S1; and late 800–1200 ms post-S1). In addition we calculated N100, P200, and P300 to S2 stimuli.

Results and Conclusions. In our sample of ASD, ADHD, and NT controls, differences were not significant at the early stage, but became significant at the late stage of the LRP yielding Time (early, late) X Group interaction (F = 6.77, p = 0.012). Post-hoc analysis showed group differences at 800–1200 ms post-S1 window (F = 4.81, p = 0.033) between ASD and NT groups. Autism group also showed more pronounced differences of LRPs in incongruent trials. Frontal N100 and P200 components showed delayed latencies in both ASD and ADHD as compared to controls (F = 4.72, p = 0.03). Our study suggests more deficient motor preparation during task congruency manipulations in ASD as compared to NT and even ADHD, which was manifested in late LRP reflective of abnormal motor preparation processing, and delayed middle latency ERP components in response to incongruent targets. Premotor potentials such as LRP can be considered as useful biomarkers of motor coordination deficits and dyspraxia in autism.

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Neuromodulation Based on 18 Session rTMS Course Improves Functional Measures of Information Processing and Behavioral Responses in Autism Spectrum Disorder

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Background. The study is based on an underlying neuropathology model of autism, which emphasizes minicolumnar pathology and lateral inhibition deficits resulting in behavioral and executive dysfunctions. We proposed that neuromodulation based on low frequency repetitive Transcranial Magnetic Stimulation (rTMS) will enhance lateral inhibition through activation of inhibitory double bouquet interneurons and will be accompanied by improvements in the prefrontal executive functions. In addition, we proposed that rTMS will improve cortical excitation/inhibition ratio and result in changes manifested in electrocortical responses such as event-related potentials (ERP) and evoked and induced EEG gamma oscillations during cognitive tests.

Objectives. TMS offers a noninvasive method for altering excitability of the neural circuits and induction of functional reorganization in the cortex. We reported earlier positive effects of rTMS in autism spectrum disorders (ASD) in our pilot studies using shorter 6- and 12-session long rTMS courses (Sokhadze et al., 2009, 2010, 2012; Casanova et al., 2012; Baruth et al., 2010). Along with traditional behavioral evaluations in current study we used ERPs and induced gamma responses power and coherence in a visual oddball task with illusory Kanizsa figures. We compared clinical, behavioral, and electrocortical (ERP, single trial EEG) outcomes in two groups of children with autism (TMS, wait-list group). We predicted that 18-session long course in ASD patients will have better behavioral and ERP/EEG outcomes as compared to an age- and IQ-matched wait-list ASD group.

Methods. We used 18 sessions of 1 Hz rTMS applied bilaterally over the dorso-lateral prefrontal cortex (DLPFC) in 27 individuals with ASD (10–21 years old). The wait-list group was comprised of 20 age-matched subjects with ASD tested twice. Both the TMS and wait-list (WTL) groups were assessed at (1) the initial baseline using clinical behavioral questionnaires (i.e., Aberrant Behavior Checklist [ABC], and Repetitive Behavior Scale-Revised [RBS-R]) and during performance on visual oddball task with Kanizsa illusory figures, and (2) post-completion of 18 sessions of TMS (or wait period).

Results. Post-TMS evaluations showed decreased irritability and hyperactivity on the ABC, and decreased stereotypic behaviors on the RBS-R. Following rTMS course we found decreased magnitude of the frontal N100 ERP component and evoked gamma power to non-targets, and shorter of the fronto-central P2a and P3a ERPs and reduced induced gamma oscillation power to non-targets in TMS group as compared to the WTL. These ERP changes along with increased P3b and enhanced induced gamma oscillations (higher power, phase coherence) to targets are indicative of more efficient processing of information post-treatment. Enhanced information processing was also reflected in such behavioral response measures as reaction time (RT) and error rate.

Conclusion. These results could be considered as a replication of our prior studies using shorter rTMS course. Collected data support our concept that rTMS improves executive functioning as evidenced by normalization of ERP and EEG responses and behavioral reactions during executive function test, and also by improvements in clinical behavioral evaluations. The study links behavioral, clinical, and ERP/EEG responses during cognitive tests and TMS outcomes with an underlying developmental neuropathology model derived from investigations in our laboratory.

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EEG Biofeedback Benefits Acquisition and Consolidation of Motor Sequence

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The evidence from different studies showed the importance of night sleep in consolidation of procedural memory. Our study tries to find out whether consolidation processes can be achieved by neurofeedback training.

It is hypothesized that consolidation of motor learning during sleep is associated with theta band activity. We used EEG-biofeedback (NF) to train participants (30 subjects, divided into two groups) to selectively increase either their relative theta or beta band power following the acquisition phase of motor sequence learning. We employed the keyboard version of the finger-tapping task (FTT) for motor learning. The participants were instructed to type the predefined order of letters as quick and accurate as possible. We tested performance on a motor task before and after motor training, after one NF session, one day, and one week.

By testing the performance in each those phases we could explore the interaction between immediate NF and subsequent sleep-dependent consolidation processes. Results revealed a significant improvement in performance immediately after NF in the theta group, but not in the beta group. The rate of improvement was significantly higher at theta group (more than 10%) in compare to beta group (4%).

Although participants were provided with feedback regarding the change in target band power using a single electrode (Fz for the beta group; Pz or Fz for the theta group, which did not differ in their modulation of theta power), in the current study we were able to measure the extent to which that single-channel feedback caused more widespread changes in the other 18 electrodes from which EEG was recorded.

The theta effect was kept up to at least one week following training: the performance slightly improved on the tests of second day and seventh day in the theta group and slightly decreased in the beta group. Across participants, post-NF improvement positively correlated with theta/beta ratio achieved during NF training in the theta group, but not in the beta group, indicating a clear relationship between memory consolidation, as reflected in motor performance, and theta NFT. Additional analysis showed correlation between EEG changes at target electrode with changes at other electrodes. Thus, regulating theta power may yield contributions to the initial performance and subsequent consolidation of an acquired motor skill.

A Pilot Study on the Correlates of Sleep Problems in Chronic Pain Patients

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Although traditional medical treatment has been relatively successful for treating acute pain, its record for treating chronic pain has been quite limited in efficacy and the findings inconsistent and inconclusive. Alternative treatments have been introduced and empirically

validated, many with promising results. Among these are non-pharmacological neuro-modulatory (NPNM) approaches such as brain stimulation, hypnosis, biofeedback, and neurofeedback (Tan et al., 2010). A number of these modalities have received empirical support in the Western literature. However, its generalizability and applicability to individuals with chronic pain outside of the United States is not known. A pilot study to assess the feasibility, acceptability, and relative efficacy of four NPNM modalities (self-hypnosis, cranial electrical simulation, HRV biofeedback, and cognitive behavioral therapy) for pain management was initiated in Singapore in 2012 and is still ongoing.

The data for this poster was obtained via the above described research project. This paper explored psychosocial correlates of chronic pain in the Singapore population, with a particular focus on sleep problems, pain catastrophizing, and negative and positive affect. Building on the findings of Buenaver et al. (2006), three mediation models were tested using a bootstrapping technique. Model 1 was a replication and examined the indirect effects of pain catastrophizing through sleep problems on pain outcomes (intensity and interference). Model 2 extended the previous research and assessed the indirect effect of negative and positive affect (conceptualized as a ratio) through sleep problems on pain outcomes. Model 3 linked the three variables and examined the indirect effect of pain catastrophizing through affect on sleep problems. For the study, 33 patients with chronic pain in the lower back or knees were recruited from the National University Hospital, a tertiary teaching hospital in Singapore. Pain interference was shown to be more strongly associated with other variables than pain intensity. Pain catastrophizing, sleep problems, and negative affect had significant correlations with other variables, while positive affect generally showed no significant associations. Model 1 was only supported for the indirect effects of pain catastrophizing (magnification) on pain interference. Model 3 was supported for pain catastrophizing and all its three components of rumination, magnification, and helplessness. The findings show the importance of considering psychosocial factors for chronic pain patients in Singapore, and that pain catastrophizing and negative affect should be addressed in the treatment of sleep problems for chronic pain patients.

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Looking for Alternative Screening Measures for Use on National Depression Screening Day

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National Depression Screening Day (NDSD) has been held since 1991 with approximately 500,000 individuals screened yearly. Many screening sites located at college counseling centers typically order a combination of screening tools. These four measures usually include: the Hands Depression Screening Tool (Bear et al., 2000); the Carroll-Davidson generalized anxiety screener (Carroll & Davidson, 2000); the Mood Questionnaire for bipolar disorder (Seetal et al., 2009); and the SPRINT-4 post-traumatic stress disorder screen (Soberay, Faragher, Barbash, Brookover, & Grimsley, 2013). The Outcome Questionnaire-

45.2 (OQ-45; Lambert et al., 2004) is considered the gold standard of outcome measures and has been widely studied, demonstrating solid reliability and validity.

In the current study of 285 students, the OQ-45 was used as a screening tool in addition to the NDSD tools. Preliminary results indicate that the Hands Depression Screening Tool correlates significantly (r = .81) with the OQ-45 total scores. The Carroll-Davidson generalized anxiety screener also correlates significantly (r = .74) with the OQ-45 total scores.

It is clear that the OQ-45 might be effectively used as a screening tool in college populations. NDSD screeners are also used at a cost to most counseling centers, whereas administering additional OQ-45's for screening purposes might save some operating cost and do as well at screening individuals wondering about their mental health.

Since the mid 1990s BYU CAPS has utilized a 45-item measure of psychological distress with clients, the Outcome Questionnaire-45 (OQ-45). The OQ-45 also takes about 7 to 10 minutes to complete. It is considered the gold standard of outcome measures and has been widely studied, demonstrating solid reliability and validity. The screening measures used in NDSD are not as widely studied. CAPS faculty and staff must train and re-train to use the Hands Screening tool each year. There is also financial cost associated with using the traditional NDSD screening tools. Anecdotally, CAPS clinicians have noticed that the screening tools may be too sensitive (a threat to validity), creating too many false positives for mental health conditions. Based on the results of the screenings, a high number of referrals to BYU CAPS are made which creates a backlog of students trying to be seen by CAPS clinicians immediately following NDSD. The current investigation looks to see if the OQ-45 could be used as a more reliable and valid screening tool. If the OQ-45 could be used as an NDSD screener, we could perhaps be more effective in giving at-risk students referrals to counseling, save money and time of CAPS clinicians, and help alleviate some of the clinical demand placed on CAPS immediately following NDSD.

The questions asked included: Do individuals who fall in the clinically significant range of the NDSD screening measures also fall in the clinical range on the OQ-45? If not, what are the differences? Are there any patterns to false positives (or false negatives)? Will the OQ-45 be an accurate and efficient screening measure to use during NDSD events?

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Effects of Neurofeedback Training in a Group of Adolescents in Conflict with the Law in Sonora Mexico

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Background and Description. Juvenile delinquency is a problem that affects any society. According with the U.S. Department of Justice, 1,470,000 juvenile arrests were registered in this country in 2011 (Puzzanchera, 2013), and in Mexico, the state of Sonora has the most adolescent population in internment (INEGI, 2012). According to some studies, antisocial and criminal behavior is associated with attention deficit, learning problems, and impulsivity (Barkley, 1997). One method that has been utilized is neurofeedback (NFB) training (Monastra et al., 2006); however, at the present time there is not much information about the effects of this procedure in adolescents in conflict with the law, nor information about the impact of the NFB in this population in Mexico.

Objective. The objective of this study is to determine changes in performance on the WISC-IV and the Stroop task (Golden, 2001), in a group of adolescents in conflict with the law, after the implementation of NFB protocols.

Methods. Participants consisted of 9 male adolescents. The group ranged in ages from 14 to 16 years. All participants were inmates of an internment center at the city of Hermosillo Sonora, Mexico. The training protocol selected consisted of the increase of beta waves, alpha waves and sensorimotor rhythm (SMR), in 48 sessions of 20 minutes each. In the sessions that increased alpha waves, the participants were relaxed and with their eyes closed. The WISC-IV and the Stroop task (Golden, 2001) were applied before and after the training protocol.

Results. Pre- to post-training scores were compared. Paired sample *t*-tests were performed to assess changes in pre- and post-training scores from the WISC-IV and Stroop task. Significant differences were found in the four indeces of the WISC-IV: Verbal Comprehension ($p < .001$); Perceptual reasoning ($p < .001$); working memory ($p < .01$) and processing time ($p < .01$) as well as in the CI index ($p < .001$). On the other hand, in the Stroop task significant differences were found in reading the words, naming the color, and naming the color instead of reading the words.

Conclusion. This is a preliminary study about the effects of NFB training in adolescents in conflict with the law in Mexico; however, more investigation and more subjects are necessary to generalize these results with this kind of population and to collaborate with the social reinsertion of these adolescents.

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Examining the Cognitive Benefits of a Phonemic Intelligence Program in Older Adults with Mild Cognitive Impairment

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Background and Description. The incidence and prevalence of Mild Cognitive Impairment (MCI) is difficult to estimate (Ward, Arrighi, Michels, & Cedarbaum, 2012). Yet many older adults express concerns to practitioners during office visits about memory issues. Current treatment of MCI focuses on diet, exercise, lifestyle changes, as well as social and intellectual stimulation (Cooper, Li, Lyketsos, & Livingston, 2013). Web-based memory exercise programs are available (Fortman, 2012) although older adults may lack Internet access and computer skills. The office visit is the opportunity for practitioners to engage older adults to discuss in-office interventions such as the complementary and alternative modalities (CAM) of neurofeedback and in-home interventions of Phonemic Intelligence exercise (PIE). PIE is a process of producing human sounds while positioning the hands on the head in a specific pattern to increase energy to targeted areas of the brain (Pillai). The purpose of this study is to explore the effectiveness of PIE for older adults who are experiencing alterations in memory.

Methods. Older adults who self-identify as experiencing memory changes are volunteers for the study. Exclusion criteria included persons taking seizure or anti-psychotic medications, a history of seizures, or active major expression. A quasi-experiential design is used with an anticipated sample of intervention $n = 50$ and control $n = 50$. A rolling enrollment during a 6-month period is being used. Subjects volunteer for an 8-week period in study. All subjects

complete the Geriatric Depression Scale, Memory Assessment Scales, and QEEG at the beginning and end of the 8-week period. The intervention group is taught PIE and instructed to practice PIE twice daily and record in a log.

Results. Data collection and active recruitment is in progress. *N* = 45 total participants with a low attrition rate.

Conclusion. QEEGs will be analyzed via z-scores and ANOVA pre- and post-intervention using NeuroGuide software. Areas of neuronal de-regulation will be reevaluated at the end of the study to assess whether the Phonemic Intelligence program improved functioning in the de-regulated areas of the brain. Pre- and post-MAS will be scored.

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Discrimination of EEG Gamma (36–50 Hz) Activity in Wernicke's Area While Listening to an Audio Book

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Previous studies have found that about three fourths of human subjects can discriminate EEG alpha activity (Frederick, 2012; Kamiya, 1968), but relatively few can do it consistently across 10 sessions. Since gamma activity has previously been shown to be involved in conscious awareness of sensory information (Engle & Singer, 2001), we hypothesized subjects could reliably discriminate gamma (36–50 Hz) activity in Wernicke's area (Brodmann Area 22; left hemisphere). We also hypothesized that listening to an audio book during the discrimination task might be a more naturalistically valid condition for attending to activity in this region, and result in better performance than a quiet eyes-closed condition. Eight participants completed a total of 35 (median 4) sessions. Each second, a 90-second baseline was recorded. Eyes were closed during the baseline and task conditions. During the task, low (< 40th percentile of baseline) and high gamma events (> 60th percentile) triggered a prompt that participants then responded to using a high or low key press, and received immediate feedback on their answer. For the first half of odd-numbered sessions, participants listened to an audio book. Two participants achieved criterion performance (binomial $p < .01$). Consistent with psychophysics, these participants were better able to discriminate very high and low signal intensities as compared to moderately high and low

intensities. In the four odd-numbered sessions during which participants reached criterion, performance improved in the second half of the session after the audio book had been turned off. This contradicted our hypothesis and suggested that the audio book might have served as more of a distraction than a relevant stimulus. However, a comparison to the even numbered sessions did not support this interpretation. In conclusion, only 25% of subjects able to discriminate gamma power in Wernicke's area within five sessions in this study. Future studies will assess whether providing standard neurofeedback training during half of each session may improve EEG state discrimination performance (as suggested by Cinciripini, 1984; Kotchoubey, Kubler, Strehl, Flor, & Birbaumer, 2002).

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One Successful Case of Neurofeedback Sessions for Social Phobia and Academic Performance

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Feelings of nervousness and anxiety in social settings always vexed Client A. Her past psychological trauma, which was inflicted upon her over an extended period of time, made her extremely difficult to feel at ease when she had to deal with interpersonal issues. Client A had been attempting various methods to overcome this difficulty, including counseling sessions or a medical leave. However, nothing seemed to have substantial efficacy on improving her perceived symptoms. In dire need to survive within a very competitive academic setting, she sought a therapy using neurofeedback technique. The therapy involved T3–T4 Delta, Pz Alpha, P4 Beta, and C3 Beta. After the first month of T3–T4 Delta (intended for a brain recovery) and P4 Beta (intended to improve reading other people's emotions) trainings, she discovered herself interacting freely with whomever came across her life. In the second month of therapy, C3 Beta training (intended to eliminate persistent daydreaming) was added. During this period, she felt that she was able to focus on the difficult lectures with such ease. Although her study hours decreased due to crammed schedule hunting for a job, the grades obtained in exams and assignment improved

compared to the previous semesters. In the third month, Pz Alpha training was added to boost her peak performance because job interviews were awaiting her. To her surprise, she was able to present herself and her competency without being self-conscious, let alone being neither nervous nor anxious. The job hunting went extremely successfully and she now has a great work-life balance with a strong sense of subjective wellbeing. Several implications could be delineated from this case study. The client showed some resistance toward traditional counseling sessions. The fact that counseling sessions involve prolonged vis-à-vis contact bore too much burden for Client A. Every time the counseling sessions were attempted, they were terminated prematurely. The alternative method of mental training, such as Vipassana meditation, was even detrimental to her wellbeing since it induced nightmares and other dreadful emotions. Taken together, neurofeedback was suitable for this case because 1) it does not involve prolonged vis-à-vis contact, and 2) it can be altered for mental training techniques (such as meditation) without tapping into the deep psyche.