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Aim and Scope

NeuroRegulation is a peer-reviewed journal providing an integrated, multidisciplinary perspective on clinically relevant research, treatment, and public policy for neurofeedback, neuroregulation, and neurotherapy. The journal reviews important findings in clinical neurotherapy, biofeedback, and electroencephalography for use in assessing baselines and outcomes of various procedures. The journal draws from expertise inside and outside of the International Society for Neurofeedback and Research to deliver material which integrates the diverse aspects of the field. Instructions for submissions and Author Guidelines can be found on the journal website (http://www.neuroregulation.org).

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Contents

NeuroRegulation

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Welcome to *NeuroRegulation* Volume 5, Issue 4. We appreciate your joining us for this issue.

In 2013 the International Society for Neurofeedback and Research (ISNR) board felt it extremely important to have an open-access journal, one where the research, reviews, and commentary in our field is not locked behind subscription pay-walls, but openly available to researchers, students, clinicians, and the general public alike. Due to copyright and financial constraints it was necessary to establish a new journal, rather than change access to the society's original journal. As such, the board voted to cease publication of the *Journal of Neurotherapy* and to stand up a new, open-access journal, *NeuroRegulation*. One very unique aspect of this open-access format, in an era where many such journals charge authors upwards of thousands of dollars to publish, is the decision for ISNR to sponsor the journal so as to not charge authors or institutions any fees to publish. More so, as a service to the neuroscience community, currently, membership in ISNR is not necessary to publish at no charge in our journal. It is hoped our readers understand the importance of that board's decision several years ago and the significant ongoing contribution ISNR makes to our field.

As we come to the end of our fifth year of publication and move into our sixth year, we pause to acknowledge our growth and accomplishments. In addition to being indexed in Elsevier's Embase and Scopus databases during 2017 and 2018, respectively, our exposure and readership is growing greatly. In identifying the most viewed publications for each year, over the last 5 years, the article with the most views is the study on the effects of chocolate on the qEEG and blood pressure by Montopoli et al. (2015), with 13,560 views and counting. The next most viewed paper, with 8,540 views to date, was an evaluation of the impacts of digital addiction by Peper and Harvey (2018). Both of these papers were picked up and promoted by Science Daily, which brought an additional spotlight and recognition for *NeuroRegulation*. Our third most viewed article, with 8,529 views so far, is a study where neurofeedback (NF) is combined with heart rate variability (HRV) training by White et al. (2017). From our 2014 publications, Esmail and Linden's (2014) review of NF with Parkinson's Disease has been viewed 3,626 times to date, and from 2016 the study from La Marca and O'Connor (2016) with results of NF improving reading achievement with Attention-Deficit/ Hyperactivity Disorder (ADHD) students has so far had 3,223 views. In looking forward to our next 5 years and beyond we anticipate many additional exceptional scholarly contributions.

In the current issue Connie McReynolds, Lelah Villalpando, and Cynthia Britt present data for NF improvements in ADHD symptoms in school-aged children. Next, Laura Barry and Gregory Nooney present data for passive Infrared Hemoencephalography (pIR HEG) in athletic performance. Then, Kirtley Thornton provides insight and perspective on placebo effects as they may be inferred to NF. Finally, select abstracts for the proceedings of the 2018 ISNR annual conference are included in this issue.

NeuroRegulation thanks the authors in this issue, as well as those before them over the years, for their valuable contributions to the scientific literature for NF and learning. We strive for high quality and interesting empirical topics. We encourage the members of ISNR and other biofeedback and neuroscience disciplines to consider publishing with us. It is important to stress that publication of case reports is always useful in furthering the advancement of an intervention for both clinical and normative functioning. We encourage researchers, clinicians, and students practicing NF to submit case studies! We thank you for reading *NeuroRegulation*!

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References

Esmail, S., & Linden, D. E. J. (2014). Neural networks and neurofeedback in Parkinson's disease. *NeuroRegulation, 1*(3-4), 240–272. http://dx.doi.org/10.15540/nr.1.3-4.240

La Marca, J. P., & O'Connor, R. E. (2016). Neurofeedback as an intervention to improve reading achievement in students with attention-deficit/hyperactivity disorder, inattentive subtype. *NeuroRegulation, 3*(2), 55–77. http://dx.doi.org/10.15540/nr.3.2.55

Montopoli, M., Stevens, L. C., Smith, C. J., Montopoli, G., Passino, S., Brown, S., … Wu, J. (2015). The acute electrocortical and blood pressure effects of chocolate. *NeuroRegulation, 2*(1), 3–28. http://dx.doi.org/10.15540/nr.2.1.3

Peper, E., & Harvey, R. (2018). Digital addiction: Increased loneliness, anxiety, and depression. *NeuroRegulation, 5*(1), 3–8. http://dx.doi.org/10.15540/nr.5.1.3

White, E. K., Groeneveld, K. M., Tittle, R. K., Bolhuis, N. A., Martin, R. E., Royer, T. G., & Fotuhi, M. (2017). Combined neurofeedback and heart rate variability training for individuals with symptoms of anxiety and depression: A retrospective study. *NeuroRegulation, 4*(1), 37–55. http://dx.doi.org/10.15540/nr.4.1.37

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NeuroRegulation

Using Neurofeedback to Improve ADHD Symptoms in School-Aged Children

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Abstract

The diagnosis and treatment of the behaviors associated with attention-deficit/hyperactivity disorder (ADHD) predominantly involves pharmacological interventions. Many children experience significant negative side effects (e.g., appetite suppression, insomnia, headaches, stomachaches, irritability, and impaired height) from the initial and continued use of stimulant medication. Consequently, many parents are motivated to consider alternative treatments for ADHD such as neurofeedback. This paper presents an archival review of the improvements in auditory and visual attention and response control after 40 sessions of artifact-corrected neurofeedback for 51 children ages 6 to 17 with ADHD. Initially, the majority of these clients were identified as having severe to extreme auditory and visual attention impairments. The IVA-2 CPT was administered prior to treatment and after 20 and 40 treatment sessions were completed. After 20 sessions of neurofeedback significant improvements of both auditory and visual attention and response control were found with small to large size effects. The clients continued to improve after an additional 20 sessions, with medium to large size effects after 40 sessions. At completion of treatment the mean of eight of the nine attention and response control scores fell within the "normal" range.

Keywords: attention-deficit/hyperactivity disorder (ADHD); auditory processing; visual processing; neurofeedback; Integrated Visual and Auditory (IVA2) Continuous Performance Test; nonpharmacological

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Introduction

According to Visser, Zablotsky, Holbrook, Danielson, and Bitsko (2015), about 11% of children, or some 6.4 million school-aged children (i.e., > 1 in 10), in the United States have been diagnosed with Attention-Deficit/Hyperactivity Disorder (ADHD). In fact, the percentage of children diagnosed with this disorder increased 42% between the years 2003 to 2012 (Visser et al., 2015). Child-learning experts believe that many more teens and children are being diagnosed with an attention-deficit disorder than is merited (e.g., Grohol, 2013). Contributing factors to the increased diagnosis of ADHD will be discussed, coupled with a discussion on using neurofeedback as an alternative treatment method for ADHD.

The purpose of this archival study was to evaluate the clinical effectiveness of neurofeedback treatment for children who presented with a variety of attentional and behavioral symptoms including anxiety, panic attacks, learning, concentration difficulties, sleep disorders, depression, and memory concerns. It was hypothesized that the Integrated Visual and Auditory Continuous Performance Test – Version 2 (IVA-2 CPT) global measures of visual and auditory attention

processing would show a significant improvement after both 20 and 40 sessions of treatment with the greatest degree of improvement expected after 40 treatment sessions.

Traditional Diagnostic Processes for ADHD

Psychiatrists, physicians, psychologists, social workers, and school counselors, along with community, parent, and children's support groups all exist to help children better manage their dysregulated behaviors. In addition, numerous types of pharmacological and behavioral interventions are commonly utilized toward correcting the undesirable behaviors. Yet, with all this support, children still experience negative outcomes from their inappropriate behaviors. Most importantly, academic improvement due to any of these interventions alone, or in combination, has not shown long-term results in that children continue to struggle with low self-esteem and perform poorly academically, some to the point of dropping out of school (Lee, Lahey, Owens, & Hinshaw, 2008; Owens, Hinshaw, Lee, & Lahey, 2009). In all, because they are not behaving appropriately, children with ADHD symptoms continue to demonstrate difficulty with developing appropriate family, school, and social interactions.

The two primary methods to diagnose ADHD are specified by either the American Psychiatric Association (APA) Diagnostic and Statistical Manual Fifth Edition (DSM-5; APA, 2013), or the International Statistical Classification of Diseases and Related Health Problems, 10th Revision, in the form of the ICD-10, a global coding system developed by the World Health Organization (WHO) for classifying diseases and clinical procedures. Other factors that may increase ADHD diagnoses include medical conditions that mimic ADHD, along with the influence of both national- and state-based educational policies. Such policies support the eligibility of ADHD under the criteria of Other Health Impaired for special education services within the school systems. A brief review of the DSM-5 diagnostic criteria for childhood ADHD identifies three primary diagnostic classifications, which include the specifiers of inattention, hyperactivity/impulsivity, or both (see Table 1).

The criteria for the diagnosis of ADHD, Predominately Inattentive Presentation requires the child must be 17 years of age or younger and exhibit six or more of the symptoms (see Table 1.A.) for at least 6 months. In addition, these symptoms must be inconsistent with the child's developmental level, have a negative effect on the child's social and academic activities, and occur often. The criteria for the diagnosis of ADHD, Predominately Hyperactive-Impulsive Presentation indicates that six or more of the listed symptoms (see Table 1.B.) must be present for at least 6 months, must be inconsistent with the child's developmental level, must have a negative effect on the child's social and academic activities, and must occur often. The diagnosis of ADHD Combined Presentation (see Table 1.C.) is given when the child meets both sets of criteria.

Table 1

DSM-5 General Diagnostic Criteria.

A. Inattention

1. Fails to pay close attention to details
2. Has trouble sustaining attention
3. Does not seem to listen when spoken to directly
4. Fails to follow through on instructions and fails to finish schoolwork or chores
5. Has trouble getting organized
6. Avoids or dislikes doing things that require sustained focus/thinking
7. Loses things frequently
8. Easily distracted by other things
9. Forgets things

B. Hyperactivity/Impulsivity

1. Fidgets with hands/feet or squirms in chair
2. Frequently leaves chair when seating is expected
3. Runs or climbs excessively
4. Trouble playing/engaging in activities quietly
5. Acts "on the go" and as if "driven by a motor"
6. Talks excessively
7. Often blurts out answers before questions are completed
8. Trouble waiting or taking turns
9. Interrupts or intrudes on what others are doing

C. Inattentive and Hyperactive/Impulsive (Combined Presentation)

Both criterion of inattention and hyperactivity-impulsivity are met for the past 6 months

Source: American Psychiatric Association, DSM-5, 2013

Table 2
ICD-10 Diagnostic Criteria.

- A behavior disorder in which the essential features are signs of developmentally inappropriate inattention, impulsivity, and hyperactivity.
- A behavior disorder originating in childhood in which the essential features are signs of developmentally inappropriate inattention, impulsivity, and hyperactivity. Although most individuals have symptoms of both inattention and hyperactivity-impulsivity, one or the other pattern may be predominant. The disorder is more frequent in males than females. Onset is in childhood. Symptoms often attenuate during late adolescence, although a minority experience the full complement of symptoms into mid-adulthood.
- A disorder characterized by a marked pattern of inattention and/or hyperactivity-impulsivity that is inconsistent with developmental level and clearly interferes with functioning in at least two settings (e.g., home, school). At least some of the symptoms must be present before the age of 7 years.

Source: https://www.icd10data.com/

Table 3
Additional ICD-10 Diagnostic Disorders Based on Behavioral and Developmental Problems.

- Specific reading disorder
- Other developmental disorders of scholastic skills
- Developmental disorder of scholastic skills, unspecified
- Conduct disorder confined to family context
- Conduct disorder, childhood-onset type
- Conduct disorder, adolescent-onset type
- Oppositional defiant disorder
- Other conduct disorders
- Conduct disorder, unspecified
- Other childhood emotional disorders
- Childhood emotional disorder, unspecified
- Other childhood disorders of social functioning
- Childhood disorder of social functioning, unspecified
- Unspecified behavioral and emotional disorders with onset usually occurring in childhood and adolescence
- Attention and concentration deficit

Source: https://www.icd10data.com/

The ADHD diagnostic criteria identified by the ICD-10, which includes consideration of the behavioral characteristics for a diagnosis of ADHD in children, are itemized in Table 2. The ICD-10 classification system provides general diagnostic category types including ADHD that is either predominantly Inattention or Hyperactive, along with ADHD that is either Combined or Unspecified. In addition, to further categorize a child's behavior, the ICD-10 provides 15 supplementary codes (see Table 3) in a section titled Behavioral and Developmental Disorders.

Medical Symptoms Mimicking ADHD

In supporting the potential of misdiagnosing ADHD, Saul (2014) identified several medical symptoms that have the capacity to imitate ADHD symptoms due to complaint features that mimic the diagnostic criteria for ADHD. These health-related conditions include difficulties with vision, sleep disturbances, substance abuse, mood disorders (e.g., bipolar, depression), hearing problems, learning disabilities, sensory-processing disorders, giftedness, seizure disorder, obsessive-compulsive disorder, Tourette's syndrome, Asperger's syndrome (i.e., an Autism spectrum disorder), neurochemical distractibility/impulsivity, schizophrenia, fetal alcohol syndrome, and Fragile X syndrome.

Educational Policy Influences on Diagnoses

Hinshaw and Scheffler (2014) identified factors associated with educational policies that have contributed to the number of school-aged children who are diagnosed with ADHD. They uncovered a relationship between the rates of ADHD diagnoses and the knowledge that many states changed how schools were budgeted in the 1990s. Specifically, operating funds for schools were distributed based on the number of students reaching set performance measures (e.g., graduation rates, achievement test scores). By the early 2000s, Hinshaw and Scheffler (2014) noted that federal dollars were subjected to the same type of budget management based mainly on student test performance. Together, both these actions at the state and federal levels were highly correlated with increases in the diagnosis of ADHD.

Hinshaw and Scheffler (2014) concluded that the resulting increases in ADHD diagnoses in school-aged children were caused by two parallel processes that motivated schools to accept children with ADHD as a means of increasing their student numbers. On the one hand, schools with high-performance standards attracted parents who wanted their children with ADHD to attend such schools for treatment

purposes (Hinshaw & Scheffler, 2014). Conversely, schools in certain states were permitted to remove children who were diagnosed with ADHD from their performance metrics, which improved the overall achievement ranking of the district (Hinshaw & Scheffler, 2014). Thus, based on the heightened drive for academic achievement and stricter school accountability, the diagnosis of ADHD rose steeply, particularly, for the neediest children (Hinshaw & Scheffler, 2014).

Traditional Treatment Approach for ADHD

Once a child has a diagnosis of ADHD, the next step is generally to determine the type of treatment needed to improve the child's behavior. Treatment approaches to reduce the behavioral symptoms of ADHD in children generally fall into one of two categories, either behavior modification and/or medication.

The results of behavior modification and/or medication interventions have positive outcomes for some children (e.g., Fabiano et al., 2009) and not for others (e.g., Sonuga-Barke et al., 2013). Specifically, in a meta-analysis of non-medication treatment modalities, Sonuga-Barke et al. (2013) did not find substantive positive treatment effects for behavioral interventions in addressing ADHD. However, Fabiano et al. (2009) uncovered support for behavioral interventions, although its long-term results were not evaluated. In a later meta-analysis, Daley et al. (2014) noted that behavioral interventions were associated with several positive benefits including bettering the empowerment of parents and reducing the conduct problems of children diagnosed with ADHD. However, positive outcomes in respect to a child's academic achievements and social skills resulting from behavioral interventions were not substantiated in this review.

For treatments based on the medication approach, several studies have identified concerns regarding the side effects of some of the typical prescription-drug regimens. Ellis (2016) discovered the possibility of bone loss (i.e., osteopenia) in children, who were taking methylphenidate (Ritalin), dexmethylphenidate (Focalin), dextroamphetamine (Dexedrine), atomoxetine (Strattera), and lisdexamfetamine (Vyvanse) medications. Nearly 25% of the children in the Ellis study displayed lower bone-mineral density in the femur, femoral neck, and lumbar spine when they were compared with children who had not taken any of these medications. Ellis also reported additional medication side effects in children consisting of gastrointestinal problems such as decreased appetite and an upset stomach, which could possibly worsen nutrition as well as diminish normal calcium intake. In other research, Brazier (2015) reported that the use of methylphenidate (Ritalin, Concerta, Medikinet, Equasym) resulted in a 60% higher risk of sleep problems, along with an almost 300% greater risk of decreased appetite. Poulton et al. (2013) reported height suppression of between 1 to 1.5 inches associated with long-term use of ADHD medication. The MTA study (2004) found that physical growth (height and weight) was diminished in children following 14 months of intensive medication treatment. Additionally, in a long-term follow-up study, Swanson et al. (2017) also found evidence of height suppression in young adults following consistent use of medication from childhood to adulthood. Moreover, in a study of 41 students with ADHD, Steiner, Sheldrick, Gotthelf, and Perrin (2011) found that medication did not have long-lasting effects and that fewer than 50% of the children remained consistent with their prescribed dosage over a 6-month time frame. Swanson et al. (2017) also noted that consistent use of medication from childhood to adulthood occurred in less than 10% of ADHD cases and that long-term use was not associated with a reduction in symptom severity in adulthood.

ADHD medication is prescribed to reduce the symptoms of ADHD but does not correct the underlying causes of the behaviors. Remarkably, children are being prescribed medication at younger ages, even as early as preschool age (DSM-5; APA, 2013). Dunlop and Newman (2016) concluded that the use of stimulant medication in children diagnosed with ADHD may be too simple of an approach to treat the complex factors underlying an individual's unregulated behavioral and emotional difficulties.

An incongruity associated with ADHD pharmacological interventions is the existence of a subset of children for whom medications provide little or no improvement in their behavioral functioning (Gleason, 2013; Prasad et al., 2013). Partial cause for a noneffective result may come from rushing some medications to the market place before achieving sufficient scientific documentation (Swanson & Volkow, 2009). For example, Swanson and Volkow noted that Adderall was reapproved in 1996 as a treatment for ADHD even though there were no controlled randomized clinical trials of its effects on children with ADHD. These authors further commented that despite initial indications of a long-term benefit over the first two treatment years, additional analyses after three treatment years were

unable to document any long-term relative advantages of the ongoing treatment with stimulants. In addition, a systematic review and meta-analysis conducted by Prasad et al. (2013) of 43 studies involving a pooled-subject total of 2,110 children revealed that drug treatment benefited a child's school performance by at most only 15%, with only a maximum of 14% of children viewed as being more on-task. The limited benefits of stimulant medication were identified by Gleason (2013) as an important reason to intensify the field's research efforts to identify alternative therapies.

With respect to the long-term benefits of medication treatments in ameliorating the behavioral symptoms of ADHD, Riddle et al. (2013) discovered that nearly 90% of the 186 children investigated continued to struggle with ADHD symptoms after 6 years of drug treatment. That is, the long-term use of ADHD medications did not result in reduced symptoms in the children who had taken medications (Riddle et al., 2013). Instead, they were found to have symptoms as severe as children who were medication-free (Riddle et al., 2013). A question that arises from these medication limitations is: Why do ADHD symptoms seem to persist following traditional treatment approaches?

The Impact of Inattention

"Limited processing capacity invariably implies a competition for attention…The term *inattention* usually implies that, at a given moment, the thing being attended to is either not what it was intended to be or not what adaptively it ought to be. If a single definition could be derived… it would refer…to the state of the individual through which learning takes place. It [attention] makes heavy demands upon the brain's processing capacity" (McCallum, 2015, p. 15–16).

Many theories of attention have been postulated over the decades, centuries, and millennia from Greek and Roman philosophers to modern day theorists. Attention has been linked to psychological processing, memory, learning, and perception (Norman, 1982). Processing of information is generally held to be part of the function of attention and memory. In order to understand incoming information, it is necessary to process the information to extract meaning (Norman, 1982). In other words, one must be able to pay attention to be able to respond appropriately. Children who cannot pay attention are thereby unable to respond appropriately to their environmental cues. This inability to attend results in a plethora of difficulties for the child that generates behavioral reactions such as disorganization, lack of follow-through, not listening when spoken to directly, daydreaming, forgetting things, frequently losing things, being easily distracted, lack of focus, or even oppositional defiant disorder, which are common descriptors for children diagnosed with ADHD.

Negative Effects of Processing Problems

Male students, in particular, can demonstrate restless behaviors or disorganization, or take on the role of the class clown (Woliver & Ibrahim, 2012), which is often preferred by them rather than feeling rejected because they are unable to learn. For females, their problems may manifest in a different manner and, as a result of less "acting out" behaviors, are not identified as frequently as their male peers as being impaired. Female students may be passed along through school even when they are progressively falling behind in their academic work. In the end, female students may experience greater consequences than their male counterparts, as evidenced by an increased incidence of attempted suicide and other forms of self-injurious behaviors (Chronis-Tuscano et al., 2010; Hinshaw et al., 2012). Regardless of gender, both genders may experience a negative trajectory that has been recognized and documented for children struggling with behaviors associated with ADHD (Lee et al., 2008; Owens et al., 2009). Negative outcomes include a decreased likelihood of completing high school, a higher incidence of involvement with the criminal justice system, and diminished employment outcomes (Breslau, Miller, Chung, & Schweitzer, 2011; Hinshaw & Ellison, 2016; Pingault et al., 2011).

Children who are unable to follow through on instructions and requests even with repeated instructions or who cannot organize tasks typically experience greater levels of frustration. Increased levels of frustration often result in negative behaviors leading to behavioral dysregulation, acting-out behaviors, and a diminished sense of self.

Behavior Is a Form of Communication

Children are not always able to effectively verbally communicate the difficulties they are experiencing. One of the communication tools of a child is their behavior. When behaviors are seen as problematic, parents and teachers seek to reduce or eliminate the unwanted behavior. Although there is evidence that behavioral interventions have positive outcomes for some children (Evans, Owens, & Bunford, 2014), the goal of behavioral interventions is to reduce or eliminate the unwanted behaviors. However, there

may be useful information that the child is communicating through these "problematic" behaviors that requires the understanding of parents and teachers—and may be the child's only means to receive the help that he or she needs.

By considering that problematic behaviors are a type of language to be decoded, a shift occurs in the perception of the observed "negative" behaviors. In considering that behavior is a form of communication, behaviors can provide information about what the child is struggling with in his or her life at home and at school. Shifting from attempting to reduce the negative behaviors using traditional medication or behavioral interventions to identifying the underlying cause of the behavioral symptoms creates an alternative approach to working with the child. Developing a greater understanding of the auditory and/or visual processing difficulties the child is experiencing provides a different context within which to apply a different intervention; namely, one that corrects the underlying auditory and visual processing difficulties. This shift has the potential to yield positive benefits for all involved—the child, the teacher, and the parents.

Auditory Processing Difficulties

Children who have auditory processing difficulties (APD) may engage in behaviors that mimic the behavioral symptoms of ADHD (Gyldenkærne, Dillon, Sharma, & Purdy, 2014). APD is not about hearing loss, typically identified through audiological assessments, but rather concerns how the brain is processing auditory stimuli. Distinguished from Central Auditory Processing Disorder (CAPD), Chermak and Musiek (1997) postulated that CAPD is an input disorder impeding selective and divided auditory attention, while ADHD is an output disorder causing sustained attention deficits across modalities.

Tomlin, Dillon, Sharma, and Rance (2015) identified the need for better assessment measures to ferret out APD and cognitive limitations in children when attempting to determine causality of APD. Furthermore, Sharma, Purdy, and Kelly (2009) concluded that assessing central auditory processing, language and reading disorders did not provide a full explanation of auditory processing difficulties. Some identified symptoms of APD are noted in Table 4.

Children with APD may have difficulty learning when they are being taught in a noisy classroom environment (Behavioural Neurotherapy Clinic, 2016; Moore, Ferguson, Edmondson-Jones, Ratib & Riley, 2010) and may be unable to follow along in a conversation or remember what is said to them when asked to perform multi-stepped tasks. Instructions must be repeated multiple times and still the child does not follow through with the requested tasks in home and school environments. Because of APD, children may define themselves as less intelligent and lose confidence in themselves. Children with APD may engage in disruptive behaviors (Woliver & Ibrahim, 2012), and some may find it better to be labeled a "problem child" (Swingle, 2015, p. 106) than incapable or, even worse, as "slow."

Table 4

Auditory Processing Difficulties.

- Difficulty hearing in noisy environments
- Difficulty following long conversations
- Problems with reading comprehension
- Trouble understanding verbal math problems
- Difficulty remembering spoken information (i.e., auditory memory deficits)
- Difficulty taking notes
- Difficulty maintaining focus on an activity if other sounds are present
- Easily distracted by other sounds in the environment
- Difficulty with organizational skills
- Difficulty following multi-step directions
- Difficulty in directing, sustaining, or dividing attention
- Difficulty with reading and/or spelling
- Difficulty processing nonverbal information
- Anxiety, which might lead to illnesses such as irritable bowel syndrome or panic attacks

Source: American Academy of Audiology, 2010

Visual Processing Difficulties

Visual processing difficulties (VPD) are not about nearsightedness or farsightedness, but rather speak to how a child's brain processes visual information (Epstein, 2015). Children who have VPD may have difficulties remaining attentive to visual tasks. Farrar, Call, and Maples (2001) discovered that children diagnosed with ADHD have problems with visual memory and spatial orientation. Hagen, Moore, Wickham, and Maples (2008) found that children who have trouble with visual skills have difficulty with attention, which interferes with executive brain functioning and mimics ADHD symptoms. Children with VPD may be easily distracted by too much visual stimulation.

Some of the identified VPD symptoms include those noted in Table 5. Children who have VPD may demonstrate difficulty in remembering information that has been shown to them. They also may struggle with remembering letters and numbers, as if they have a short- or long-term memory problem (Epstein, 2015).

Table 5
Visual Processing Difficulties.

- May exhibit difficulty with tasks that require copying (e.g., taking notes from a whiteboard)
- Written copies may be missing words
- Often cannot remember even basic facts about material read silently
- Complains of eye strain or frequently rubs eyes despite no presence of poor eye sight
- Below average reading or writing level coupled with high oral comprehension and verbal skills
- Math skills may be demonstrated below average, may ignore function signs, omit steps or confuse visually similar formulae
- Routinely fails to observe or recognize changes in bulletin-board displays, signs, or posted notices

Source: New Brunswick Department of Education, 1999

Identifying Auditory and Visual Processing Difficulties

The Integrated Visual and Auditory Continuous Performance Test – Version 2 (IVA-2; Sandford & Sandford, 2014) supports clinicians in their efforts to identify an individual's strengths and weaknesses in visual and auditory processing (Sandford & Sandford, 2014). Although the ability to discriminate between APD and ADHD has yet to be fully established in the treatment of ADHD, Gyldenkærne et al. (2014) uncovered some degree of correlation between APD and ADHD measures. However, "even though deficits in both APD and maintained attention co-occur in more children than would be expected from chance alone, the two conditions are separate and largely independent conditions, even though they may have similar symptoms" (Gyldenkærne et al., 2014, p. 676). Determining whether or not auditory and visual processing difficulties are a function of ADHD, or if ADHD has become a catchment category for auditory and visual processing difficulties, remains inconclusive.

Regardless of how auditory and visual processing difficulties are categorized, when a child cannot process what is being said to him or her regardless of the number of times the auditory and/or visual information is repeated, something is interfering with the child's ability to do so. Children generally want to succeed in school, and they want to have positive relationships with their parents, peers, and teachers. They want to have a better life and they want to do well. Yet many are unable to achieve these goals, despite their best efforts. Identifying and strengthening APD and VPD processes in children may lead to a reduction in problematic behaviors, yielding improvements both at home and at school. Based on our clinic work with children who have auditory and visual processing difficulties, often associated with an ADHD diagnosis, the results of this work are reported and discussed in the following sections.

Table 6 lists some of the behavioral symptoms identified with the IVA-2 assessment in children who have auditory/visual attentional difficulties.

Table 6
Symptoms of Auditory/Visual Processing Deficits Identified via IVA-2.

- Significant problems remaining alert (i.e., likely to tune out)
- Problems shifting sets (i.e., likely to drift off)
- Difficulty getting back on track when distracted by auditory or visual stimuli
- Deficits in auditory or visual working memory
- Difficulty in maintaining focus to auditory or visual stimuli
- Difficulty following directions accurately
- Misunderstanding verbal instructions
- Problems with self-esteem or self-confidence
- Erratic responses to auditory and/or visual stimuli (i.e., makes more errors when high demand to perform)
- Frequent lapses in visual or auditory attention
- Rushes through written work resulting in careless errors
- Attention problems related to slow mental processing
- Problems with response inhibition and impulse control tendencies reflecting carelessness, thoughtlessness, or overreactivity
- Problems regulating and directing actions when stressed or tired (i.e., gives up)
- Acting out, irritability, and negative verbalizations
- Impaired social interactions with peers

Table 6 (continued)
Symptoms of Auditory/Visual Processing Deficits Identified via IVA-2.

- Trouble with self-direction and completing necessary work
- Tendencies reflecting distractible, divergent or variable attention processing when given a repetitive, demanding, structured, nonentertaining task
- Difficulties learning new tasks in the school environment
- Slow mental processing speed
- Problems sustaining attention and responding in a consistent manner when asked questions verbally or given written tests
- Starts tasks then quickly runs out of steam; may be very slow in getting the work done that needs to be completed
- Impulsive, agitated, chaotic, overexcited, and turbulent
- Significant problems with self-control
- Difficulty listening, remembering, or following rules
- Agitated, confused, or excessively impulsive response pattern
- Internally distracted to the point there is difficulty concentrating and performing meaningful mental activities
- Significant trouble with test performance

Source: IVA-2, Sandford & Sandford, 2014

Methods

Participants

Neurofeedback treatment was provided for 51 children (*n* = 35 males, *n* = 16 females, ages 6 to 17) who completed a total of 40 half-hour sessions of neurofeedback treatment. The participants for this study were randomly drawn from an archival database of children who had previously received individualized neurofeedback training within a university-based clinic setting. Only those children who completed 40 neurofeedback treatment sessions and were identified by the IVA-2 comprehensive diagnostic algorithm to have manifested significant ADHD symptomatology were selected for this study.

Participants were brought in by a parent or guardian and informed consent was obtained prior to starting the treatment. Clinical neurofeedback services were provided to participants based on a sliding fee scale and since this was an archival study they were not compensated. This study was approved by the California State University San Bernardino Institutional Review Board.

Measurements

The IVA-2 CPT has been found to be a valid and reliable measure of both visual and auditory attention functioning for children and adults and provides global and primary measures of attentional functioning. The normative sample, with approximately equal numbers of males and females, included 1,700 individuals ages 6 to 96 (Maddux, 2010). Furthermore, the IVA-2 provides both global and primary measures of attentional functioning that assess auditory and visual attention processing. All IVA-2 scale scores have a mean of 100 and a standard deviation of 15 (Sandford & Sandford, 2014).

The IVA-2 global and standard measures of attention used in this study are the Auditory Attention Quotient (AAQ), Visual Attention Quotient (VAQ), Full Scale Attention Quotient (FAQ), Auditory Response Control Quotient (ARCQ), Visual Response Control Quotient (VRCQ), Full Scale Response Control Quotient (FRCQ), Sustained Auditory Attention Quotient (SAAQ), Sustained Visual Attention Quotient (SVAQ), and the Sustained Full Scale Attention Quotient (SFAQ; Sandford & Sandford, 2014; see Appendix for scale descriptions).

The Auditory Attention Quotient (AAQ) is a global measure of attention comprised of three primary visual and auditory scales: Vigilance, Speed, and Focus. Vigilance measures errors of omission, and Speed provides a measure of the response time in milliseconds to visual and auditory stimuli targets. Focus is a measure of the variability of response time to auditory test targets. The Visual Attention Quotient (VAQ) is based on the exact same scales as the AAQ but differs in that it assesses visual test responses to the same measures of attention. The FAQ is a global composite scale comprised of the AAQ and VAQ scales, which are used in equal weights (not an average) to determine the FAQ (Sandford & Sandford, 2014).

The Auditory Response Control Quotient (ARCQ) is a global measure comprised of three response control scales: Prudence, Consistency, and Stamina. Prudence measures impulsivity and response inhibition as evidenced by three different types of errors of commission. Consistency measures the general variability of response times ignoring outliers and is a measure of the ability to stay on task. Stamina compares the mean reaction times of correct

responses between the first and last half of the IVA-2 test and is used to identify an individual's problems related to fatigue in mental processing speed over time. The Visual Response Control Quotient (VRCQ) has the exact same component scales as ARCQ but differs in that it specifically assesses visual test responses. The FRCQ is a composite scale comprised of the ARCQ and VRCQ scales; it is the combined measure of the auditory and visual primary scales that assess impulsivity, consistency of response time and performance stamina during the test (Sandford & Sandford, 2014).

The Sustained Auditory Attention Quotient (SAAQ) provides a global measure of a person's ability to respond to auditory stimuli under low demand conditions accurately, quickly, and reliably, and it is combined with an assessment of the person's ability to sustain attention and be flexible under high demand conditions when auditory stimuli change frequently. It is comprised of the following primary scales: Acuity, Dependability, Elasticity, Reliability, Steadiness, and Swiftness. Acuity measures errors of omission under low demand conditions, Dependability reflects the variability of reaction times under low demand conditions, Elasticity reflects the ability to be flexible when faced with changing conditions, Reliability measures idiopathic errors of commission, Steadiness is a measure of accuracy under high demand conditions, and Swiftness measures response times under low demand conditions when the targets are rare. The Sustained Visual Attention Quotient (SVAQ) measures the exact same type of factors as SAAQ, but specifically for visual test responses. The Sustained Full Scale Attention Quotient (SFAQ) is the combined weighted global measure of the SAAQ and SVAQ global scales (Sandford & Sandford, 2014).

Test Procedures

Every participant was administered the IVA-2 CPT before beginning their first neurofeedback session. Testing was individually administered and scored in accordance with the specified test guidelines. Some individuals were not able to validly respond to either visual or auditory IVA-2 test stimuli due to their extreme deficits in attentional functioning. In these cases, their "invalid scores" for IVA-2 were scored as zero in accordance with the recommended test interpretive procedures (Sandford & Sandford, 2014). After the completion of both 20 and 40 neurofeedback sessions, the IVA-2 was readministered. Fifty-one participants completed 40 neurofeedback sessions. The IVA-2 data was analyzed comparing baseline test scores and the scores obtained after the 20th and 40th neurofeedback sessions were completed.

Neurofeedback Treatment Protocols

An individualized neurofeedback training plan was developed for each participant and modified as necessary. Treatment was provided on a one-to-one basis in a private room setting. Therapeutic goals focused on improving auditory and/or visual attentional functioning and reducing any identified behavioral-related symptoms of anxiety or fine motor hyperactivity. Training was completed using the SmartMind 3 artifact-corrected neurofeedback system with a two-channel EEG station (BrainTrain, Inc., North Chesterfield, Virginia) which continuously filters out both brief facial activity, as well as frequently occurring eye-blink and eye-movement artifacts in real time without interrupting the training program. As conducted in a McReynolds, Bell, and Lincourt (2017) study, neurofeedback exercises were provided in game-like format that utilized both visual and auditory reinforcement, as well as graphs and numerical scores to provide positive reinforcement. The first step in the training session was to collect participants' baseline EEG data to determine *Z*-score feedback goals for each participant. Based on each individual's performance, they were provided clinically relevant feedback and adjustments were made to the training protocol to optimize their performance. Sensors were attached and secured using 10-20 electrode paste and electrode rings after the site locations were prepared. Impedance was checked to meet the manufacturer's requirements prior to the beginning of training. All EEG data was automatically deartifacted and recorded by the SmartMind 3 software.

Results

The three main global scale scores that measured combined changes in auditory and visual general attention (FAQ), sustained attention (SFAQ), and response control (FRCQ) comprised the first step of evaluation. These three tests were correlated .54 and a Bonferroni correction was calculated taking into account the correlation using an alpha criterion of .05. The IVA-2 scores of these three scales were collected at baseline, and after 20 and 40 sessions were completed. This resulted in a determination that the *p* value test of significance criterion needed to be .03 for the nine paired *t*-tests that were completed. Given that it was expected based on past research studies that neurofeedback would result in positive changes in both auditory and visual attention, one-tail *t*-test values were used in assessing significance. Since

the normative mean quotient score of the IVA-2 test is 100 and its standard deviation is 15, any change or difference of 8 or more quotient score points (i.e., greater than one half of a standard deviation) was considered to be of clinical significance. In order to evaluate whether neurofeedback training improves auditory and visual attention, paired sample *t*-tests were computed comparing the changes for the IVA-2 FAQ, SFAQ, and FRCQ global scale scores by comparing the scores between the baseline, after 20 sessions, after 40 sessions, and between 20 and 40 sessions.

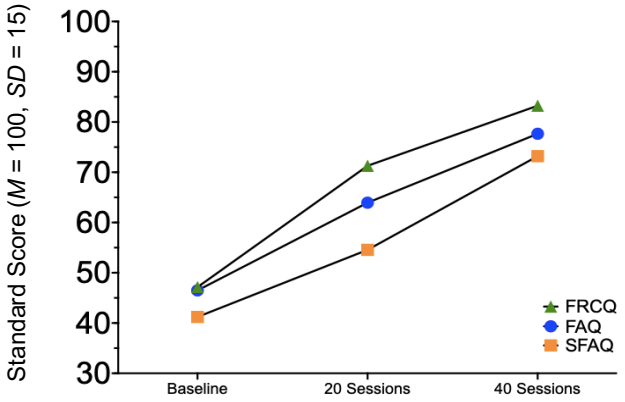

Figure 1. IVA-2 Global Combined Scale Score Changes During Training for FRCQ (Full-scale Response Control Quotient, 20 Sessions, $p < .001$, Cohen's $d = 0.61$; 20–40 Sessions, $p < .01$, Cohen's $d = 0.33$); FAQ (Full Scale Attention Quotient, 20 Sessions, $p < .001$, Cohen's $d = 0.45$; 20–40 Sessions $p < .01$, Cohen's $d = 0.37$); SFAQ (Sustained Full-scale Attention Quotient, 20 Sessions, $p < .01$, Cohen's $d = 0.34$; 20–40 Sessions, $p < .001$, Cohen's $d = 0.48$). See Tables 7, 8, 9.

As indicated in Figure 1 (see Tables 7, 8, 9), children significantly increased their Global Combined Scale Scores for FRCQ. Scores were found to be significantly higher after 20 sessions of treatment from a mean of 47 (Extremely Impaired) to 71 (Moderately to Severely Impaired), a 24-point increase, $t(50) = -4.59$, $p < .001$, Cohen's $d = 0.61$; FRCQ scores from 20 to 40 sessions of treatment significantly improved from a mean of 71 (Moderately to Severely Impaired) to 83 (Mildly Impaired), a 12-point increase, $t(50) = -2.79$, $p < .01$, Cohen's $d = 0.33$; overall, FRCQ scores between baseline and 40 sessions reflected a significant improvement from a mean of 47 (Extremely Impaired) to 83 (Mildly Impaired), a 36-point improvement, $t(50) = -6.22$, $p < .001$, Cohen's $d = 0.93$.

FAQ scores were found to be significantly higher after 20 sessions of treatment from a mean of 47 (Extremely Impaired) to 64 (Severely Impaired), a 17-point increase, $t(50) = -3.48$, $p < .001$, Cohen's $d = 0.45$; FAQ scores from 20 to 40 sessions improved from a mean of 64 (Severely Impaired) to 78 (Mildly to Moderately Impaired), a 14-point increase, $t(50) = -3.25$, $p < .01$, Cohen's $d = 0.37$; overall, FAQ scores between baseline and 40 sessions reflected a significant improvement from a mean of 47 (Severely Impaired) to 78 (Mildly to Moderately Impaired), $t(50) = -5.35$, $p < .001$, Cohen's $d = 0.82$, a 31-point improvement.

SFAQ scores were found to be significantly higher after 20 sessions of treatment from a mean of 41 (Extremely Impaired) to 55 (Extremely Impaired), a 14-point increase, $t(50) = -2.51$, $p < .01$, Cohen's $d = 0.34$; SFAQ scores from 20 to 40 sessions of treatment significantly improved from a mean of 55 (Extremely Impaired) to 73 (Moderately Impaired), an 18-point increase, $t(50) = -4.11$, $p < .001$, Cohen's $d = 0.48$; overall, SFAQ scores between baseline and 40 sessions reflected a significant improvement from a mean of 41 (Extremely Impaired) to 73 (Moderately Impaired), a 32-point improvement, $t(50) = -5.43$, $p < .001$, Cohen's $d = 0.85$ (see Table 7 for baseline to 20, Table 8 for 20 to 40, and Table 9 for Baseline to 40).

Table 7

Paired t-tests comparing changes in the mean IVA-2 main global measures of attention and response control scale scores between baseline and after completion of 20 neurofeedback sessions.

IVA-2 Attention Scales	Baseline	20 Sessions	Q Score Change	Pooled *SD*	Sig.	Cohen's *d*
FAQ	47	64	17	39.09	.001	0.45
SFAQ	41	55	14	39.15	.01	0.34
FRCQ	47	71	24	39.83	.001	0.61

Table 8
Paired t-tests comparing changes in the mean IVA-2 main global measures of attention and response control scale scores between 20 sessions and after completion of 40 neurofeedback sessions.

IVA-2 Attention Scales	20 Sessions	40 Sessions	Q Score Change	Pooled *SD*	Sig.	Cohen's *d*
FAQ	64	78	14	36.64	.01	0.37
SFAQ	55	73	18	39.00	.001	0.48
FRCQ	71	83	12	36.13	.01	0.33

Table 9
Paired t-tests comparing changes in the mean IVA-2 main global measures of attention and response control scale scores between baseline and after completion of 40 neurofeedback sessions.

IVA-2 Attention Scales	Baseline	40 Sessions	Q Score Change	Pooled *SD*	Sig.	Cohen's *d*
FAQ	47	78	31	37.90	.001	0.82
SFAQ	41	73	32	37.62	.001	0.85
FRCQ	47	83	36	38.67	.001	0.93

In Figure 1, the continued improvement in global attention and response control from 20 to 40 sessions that was significant can be viewed across the FRCQ and SFAQ global measures. Scores on the FRCQ were found to be significantly higher after 20 sessions of treatment with a 14-point increase and significantly higher with an 18-point increase after 40 sessions. The SFAQ global scale scores were found to be significantly higher after 20 sessions of treatment with a 14-point increase and significantly higher with an 18-point increase after 40 sessions. The FAQ test scores significantly increased 17 points from baseline to 20 sessions; however, unlike the FRCQ and the SFAQ, the FAQ showed a 14-point change from 20 to 40 sessions.

The FAQ, SFAQ, and FRCQ are combined global scales based on each of their two respective auditory and visual global scales. General attention, sustained attention and response control global scale scores improved at 20 sessions with small to medium size effects, and all three global measures continued to significantly improve after an additional 20 sessions of training, resulting in neurofeedback treatment significantly improving attention and self-control after additional training with large size effects (i.e., > 2 *SD*s).

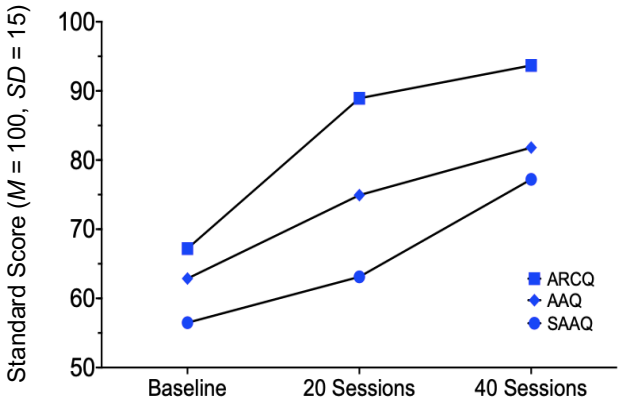

Figure 2. IVA-2 Auditory Scale Score Changes During Training for ARCQ (Auditory Response Control Quotient, 20 Sessions, $p < .001$, Cohen's $d = 0.73$; 20–40 Sessions, $p > .10$, Cohen's $d = 0.21$); AAQ (Auditory Attention Quotient, 20 Sessions, $p < .01$, Cohen's $d = 0.37$; 20–40 Sessions, $p < .05$, Cohen's $d = 0.23$); SAAQ (Sustained Auditory Attention Quotient, 20 Sessions, $p > .10$, Cohen's $d = 0.18$; 20–40 Sessions, $p < .01$, Cohen's $d = 0.40$). See Tables 10, 11, 12.

As indicated in Figure 2, ARCQ scores were found to be significantly higher after 20 sessions of treatment from a mean of 67 (Severely Impaired) to 89 (Slightly Impaired), a 22-point increase, $t(50) = -4.5$, $p < .001$, Cohen's $d = 0.73$; ARCQ scores from 20 to 40 sessions of treatment normalized from a mean of 89 (Slightly Impaired) to 94 (Average), a 5-point

increase, $t(50) = -1.18$, $p > .10$, Cohen's $d = 0.21$; overall, ARCQ scores between baseline and 40 sessions reflected a significant improvement from a mean of 67 (Severely Impaired) to 94 (Average), a 27-point improvement, $t(50) = -4.63$, $p < .001$, Cohen's $d = 0.81$.

AAQ scores were also found to be significantly higher after 20 sessions of treatment from a mean of 63 (Severely Impaired) to 75 (Moderately Impaired), a 12-point increase, $t(50) = -2.45$, $p < .01$, Cohen's $d = 0.37$; AAQ scores from 20 to 40 sessions of treatment improved from a mean of 75 (Moderately Impaired) to 82 (Mildly Impaired), a 7-point increase, $t(50) = -1.75$, $p < .05$, Cohen's $d = 0.23$; overall, AAQ scores between baseline and 40 sessions reflected a significant improvement from a mean of 63 (Severely Impaired) to 82 (Mildly Impaired), a 19-point improvement, $t(50) = -3.68$, $p < .001$, Cohen's $d = 0.57$.

SAAQ scores were found to be improved after 20 sessions of treatment from a mean of 56 (Extremely Impaired) to 63 (Severely Impaired), a 7-point increase, $t(50) = -1.21$, $p > .10$, Cohen's $d = 0.18$; SAAQ scores from 20 to 40 sessions of treatment significantly improved from a mean of 63 (Severely Impaired) to 77 (Mildly to Moderately Impaired), a 14-point increase, $t(50) = -2.66$, $p < .01$, Cohen's $d = 0.40$; overall, SAAQ scores between baseline and 40 sessions reflected a significant improvement from a mean of 56 (Extremely Impaired) to 77 (Mildly to Moderately Impaired), a 21-point improvement, $t(50) = -3.59$, $p < .001$, Cohen's $d = 0.57$ (see Tables 10, 11, 12).

Table 10

Paired t-tests comparing changes in the mean IVA-2 main global measures of auditory attention and response control scale scores between baseline and after completion of 20 neurofeedback sessions.

IVA-2 Attention Scales	Baseline	20 Sessions	Q Score Change	Pooled *SD*	Sig.	Cohen's *d*
AAQ	63	75	12	32.81	.01	0.37
SAAQ	56	63	7	36.89	.10	0.18
ARCQ	67	89	22	29.74	.001	0.73

Table 11

Paired t-tests comparing changes in the mean IVA-2 main global measures of auditory attention and response control scale scores between 20 sessions and after completion of 40 neurofeedback sessions.

IVA-2 Attention Scales	20 Sessions	40 Sessions	Q Score Change	Pooled *SD*	Sig.	Cohen's *d*
AAQ	75	82	7	29.40	.05	0.23
SAAQ	63	77	14	35.39	.01	0.40
ARCQ	89	94	5	23.01	.10	0.21

Table 12

Paired t-tests comparing changes in the mean IVA-2 main global measures of auditory attention and response control scale scores between baseline and after completion of 40 neurofeedback sessions.

IVA-2 Attention Scales	Baseline	40 Sessions	Q Score Change	Pooled *SD*	Sig.	Cohen's *d*
AAQ	63	82	19	32.99	.001	0.57
SAAQ	56	77	21	36.08	.001	0.57
ARCQ	67	94	27	32.67	.001	0.81

In Figure 2, the continued improvement in auditory attention and auditory response control from 20 to 40 sessions that was significant can be viewed across all three global auditory measures. The ARCQ test scores significantly increased 22 points after 20 sessions and increased 5 points after 40 sessions, reflecting that significant change occurred from baseline to 20 sessions. Scores on the AAQ were found to be significantly higher from baseline to 20 sessions of treatment with a 12-point increase and a 7-point increase after 40 sessions, reflecting significant change occurred on the AAQ global scale at 20 sessions. The SAAQ global scale scores increased 7 points after 20 sessions of treatment and was significantly higher with a 14-point increase after 40 sessions, reflecting that significant change occurred following 40 sessions.

Auditory attention (AAQ) global scale (Vigilance, Focus, and Speed) were in the Severely Impaired range at baseline and normalized to the Mildly Impaired range at 40 sessions. ARCQ (Prudence, Consistency, and Stamina) scores were in the Severely Impaired range at baseline and normalized to the Average range following 40 sessions of treatment. SAAQ (Inattention) scores were in the Extremely Impaired range at baseline and were in the Mildly to Moderately Impaired range following 40 sessions of treatment.

As indicated in Figure 3, VRCQ scores were found to be significantly higher after 20 sessions of treatment from a mean of 54 (Extremely Impaired) to 72 (Moderately Impaired), an 18-point increase, $t(50) = -3.20$, $p < .01$, Cohen's $d = 0.45$; VRCQ scores from 20 to 40 sessions of treatment significantly improved from a mean of 72 (Moderately Impaired) to 81 (Mildly Impaired), a 9-point increase, $t(50) = -2.11$, $p < .02$, Cohen's $d = 0.26$; overall, VRCQ scores between baseline and 40 sessions reflected a significant improvement from a mean of 54 (Extremely Impaired) to 81 (Mildly Impaired), a 27-point improvement, $t(50) = -4.35$, $p < .001$, Cohen's $d = 0.69$.

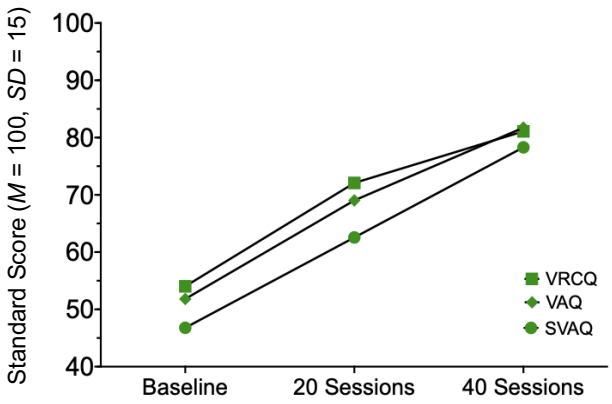

Figure 3. IVA-2 Visual Scale Score Changes During Training for VRCQ (Visual Response Control Quotient, 20 Sessions, $p < .01$, Cohen's $d = 0.45$; 20–40 Sessions, $p < .02$, Cohen's $d = 0.26$); VAQ (Visual Attention Quotient, 20 Sessions, $p < .01$, Cohen's $d = 0.43$; 20–40 Sessions, $p < .01$, Cohen's $d = 0.35$); SVAQ (Sustained Visual Attention Quotient, 20 Sessions, $p < .01$, Cohen's $d = 0.39$; 20–40 Sessions, $p < .001$, Cohen's $d = 0.41$). See Tables 13, 14, 15.

VAQ scores were found to be significantly higher after 20 sessions of treatment from a mean of 52 (Extremely Impaired) to 69 (Moderately to Severely Impaired), a 17-point increase, $t(50) = -3.03$, $p < .01$, Cohen's $d = 0.43$; VAQ scores from 20 to 40 sessions of treatment significantly improved from a mean of 69 (Moderately to Severely Impaired) to 82 (Mildly Impaired), a 13-point increase, $t(50) = -2.83$, $p < .01$, Cohen's $d = 0.35$; overall, VAQ scores between baseline and 40 sessions reflected a significant improvement from a mean of 52 (Extremely Impaired) to 82 (Mildly Impaired), a 30-point improvement, $t(50) = -4.64$, $p < .001$, Cohen's $d = 0.77$.

SVAQ scores were found to be significantly higher after 20 sessions of treatment from a mean of 47 (Extremely Impaired) to 63 (Severely Impaired), a 16-point increase, $t(50) = -2.53$, $p < .01$, Cohen's $d = 0.39$; SVAQ scores from 20 to 40 sessions of treatment significantly improved from a mean of 63 (Severely Impaired) to 78 (Mildly to Moderately Impaired), a 13-point increase, $t(50) = -3.33$, $p < .001$, Cohen's $d = 0.41$; overall, SVAQ scores between baseline and 40 sessions reflected a significant improvement from a mean of 47 (Extremely Impaired) to 78 (Mildly to Moderately Impaired), a 31-point improvement, $t(50) = -4.76$, $p < .001$, Cohen's $d = 0.82$ (see Tables 13, 14, 15).

Table 13
Paired t-tests comparing changes in the mean IVA-2 main global measures of visual attention and response control scale scores between baseline and after completion of 20 neurofeedback sessions.

IVA-2 Attention Scales	Baseline	20 Sessions	Q Score Change	Pooled *SD*	Sig.	Cohen's *d*
VAQ	52	69	17	39.88	.01	0.43
SVAQ	47	63	16	40.82	.01	0.39
VRCQ	54	72	18	40.13	.01	0.45

Table 14
Paired t-tests comparing changes in the mean IVA-2 main global measures of visual attention and response control scale scores between 20 sessions and after completion of 40 neurofeedback sessions.

IVA-2 Attention Scales	20 Sessions	40 Sessions	Q Score Change	Pooled *SD*	Sig.	Cohen's *d*
VAQ	69	82	13	36.84	.01	0.35
SVAQ	63	78	15	38.77	.001	0.41
VRCQ	72	81	9	35.33	.02	0.26

Table 15
Paired t-tests comparing changes in the mean IVA-2 main global measures of visual attention and response control scale scores between baseline and after completion of 40 neurofeedback sessions.

IVA-2 Attention Scales	Baseline	40 Sessions	Q Score Change	Pooled *SD*	Sig.	Cohen's *d*
VAQ	52	82	30	38.79	.001	0.77
SVAQ	47	78	31	38.51	.001	0.82
VRCQ	54	81	27	39.03	.001	0.69

In Figure 3, the continued improvement in auditory attention from 20 to 40 sessions that was significant can be viewed across all three global visual attention and response control measures. The VRCQ global scale scores increased 18 points from baseline to 20 sessions of treatment and increased 9 points after 40 sessions reflecting that significant change occurred after 20 and 40 sessions. Scores on the VAQ were found to be significantly higher from baseline to 20 sessions of treatment with a 17-point increase followed by a 13-point increase after 40 sessions reflecting significant change occurred on the visual global scales of attention after 20 and 40 sessions. The SVAQ test scores significantly increased 16 points after 20 sessions and increased 13 points after 40 sessions reflecting significant change occurred at 20 and 40 sessions.

Visual attention (VAQ) global scale (Vigilance, Focus, and Speed) were in the Extremely Impaired range at baseline and normalized to the Mildly Impaired range at 40 sessions. VRCQ (Prudence, Consistency, and Stamina) scores were in the Extremely Impaired range at baseline and normalized to the Mildly Impaired range following 40 sessions of treatment. SVAQ (Inattention) scores were in the Extremely Impaired at baseline and were in the Mildly to Moderately Impaired range following 40 sessions of treatment. Thus, these test results support the hypothesis that artifact-corrected neurofeedback training led to a significant improvement in global measures of both auditory and visual attention.

Discussion

Neurofeedback therapy, or EEG biofeedback, has been widely used for more than 40 years. During this time, it has gained recognition as an acceptable approach for treating conditions ranging from ADHD to anxiety, depression, sleep disorders, and learning disabilities (Hammond, 2011). For children identified as having impaired attention, once auditory and/or visual areas of weakness have been strengthened, many of the disruptive behavioral symptoms diminish (Ghaziri et al., 2013; Zhonggui, Shuhua, & Haiqing, 2005). This study specifically identified that artifact-corrected neurofeedback, which works by filtering out the contamination that continually results from naturally occurring EMG artifacts such as eye blinks, eye movements, and facial activity significantly improved both auditory and visual attention in impaired children.

As a group, these children initially presented as significantly impaired. After 40 half-hour treatment sessions, both their auditory and visual attention abilities improved and most IVA-2 scale score means were found to fall within the "normal" range (i.e., defined as a standard score of 77 or higher, 1.5 standard deviations; Sandford & Sandford, 2014) and all scales had effect sizes in the medium to large range demonstrating the clinical efficacy of this neurofeedback therapy. A standard score change of 8 points or more is clinically significant. With the additional 20 sessions, children gained 8+ points, and in some cases, a full *SD* of 15+ points. All scale score means improved from baseline to 20 sessions ranging from a 12-point to 24-point increase, except for SAAQ, which showed a 7-point increase.
The results of this archival study reveal that impaired children (N = 51) in this study needed 40 sessions to reach a normalized score. Improvements were noted across all global and standard scales, improvements were comprehensive and included both auditory and visual modalities. The improvements included response control (i.e., vigilance, consistency and stamina) in addition to both general attention (i.e., accuracy, consistency and speed) and sustained attention.

There were differences based on medium vs. large size effects for SVAQ versus SAAQ and ARCQ versus VRCQ. The SVAQ standard scales showed large effect size improvements (baseline to 40 sessions) and the SAAQ showed medium effect size improvements (baseline to 40 sessions). ARCQ showed large effect size improvements from the severely impaired range to only being slightly impaired (baseline to 40 sessions) and the VRCQ showed medium effect size improvements (baseline to 40 sessions). In respect to clinical improvement, greater changes were found on average for the visual attention scales (VAQ and SVAQ) in comparison to the auditory attention scales (AAQ and SAAQ) with these visual scale scores improving by .5 *SD* more than the auditory attention scales.

While this study utilized archival data and there was no control group to evaluate for possible test practice effects, the IVA-2 is an objective measure of attention which controls for practice effects in both its simplistic design (i.e., the test rule is to click if you see or hear the number one) and in its pretest instruction phase, which includes specific opportunities for individuals to practice the test before taking it. The reliability study in the test manual found that, on retesting, subjects did not significantly change by more than 3 to 4 points in either direction (Sandford & Sandford, 2014). Thus, any group increases in IVA-2 quotient scores greater than 3 to 4 points can be validly interpreted as a result of an active treatment and not due to practice effects. In this study, it was found that 40 half-hour neurofeedback sessions led specifically to the significant enhancement of auditory and visual attention as evidenced by the greater effect sizes observed and the significant increase in both auditory and visual attention scales from baseline to 20 sessions and from 20 to 40 sessions.

Thus, the hypothesis of this study that neurofeedback would significantly improve both auditory and visual attention was confirmed, and effect sizes were large for the enhancement on the three comprehensive IVA-2 global measures of general attention, sustained attention and response control (FRCQ, FAQ, and SFAQ, respectively). The findings of this archival study support that neurofeedback offers the potential as an alternative, nonpharmacological treatment approach that is clinically efficacious without any significant side effects.

Lubar (1995) found that the benefits of neurofeedback can potentially be long lasting. In a 10-year follow-up study, Lubar reported that about 80% of clients treated with neurofeedback substantially improved their symptoms of ADD and ADHD, and that the changes were maintained. Cannon and Lubar (2011) later reported that neurofeedback training may generate a global integration effect that progresses on a continuum yielding ongoing improvements in the areas of working memory, higher order executive functioning, affective processing, attention, and cognition. In

general, neurofeedback is becoming more widely accepted by many health care professionals and warrants institutional and governmental support for new research specifically with children who have ADHD symptomatology based on the results of this study and numerous others.

This research finding further substantiates the value and benefit of utilizing this new artifact corrected type of neurofeedback in the treatment of children with symptoms of ADHD and warrants further research as a neurofeedback intervention. In this study, initially children (N = 51) were found to be experiencing significant levels of attention impairment. After receiving neurofeedback treatment without any supportive counseling or coaching, all of them improved across the nine global and standard score measures of auditory and visual processing. Thus, the benefits of artifact corrected type neurofeedback demonstrated potential to help children improve their attentional functioning, which is consistent with the findings of La Marca et al. (2018) and consistent with the findings of improved attentional functioning of McReynolds et al. (2017).

In interpreting the results of this study, certain limitations were considered. A primary drawback was its archival nature. Follow-up evaluations at 6 months and 1 year to determine the long-term effects of neurofeedback treatment are also components recommended for consideration for future studies in this field.

While this archival research was not designed to evaluate learning effects, the evaluation and demonstration of individuals' learned control of brainwave activity is an important issue which needs to be addressed in future research. Thus, research specifically designed to measure learning effects respective to the targeted EEG frequencies trained is recommended. However, in order to evaluate learning effects this type of study would require that all participants in the study receive training that used the same standardized treatment protocol for each person. Any clinical modifications to meet a person's specific needs would not be permitted.

Based on 8 years of work with children using EEG biofeedback, children in our clinic have reported they are "able to pay attention even when they don't want to…" and that they are able to "choose whether to focus on what their friends are saying in the classroom or to listen to the teacher." Children in our clinic have gone from earning poor grades to passing grades, some excelling to the level of being on the honor roll for the first time in their academic lives. Based on child and parent self-report, the resulting enhanced attentional auditory and visual processing has led to improvements in academic work, decreased behavioral interventions, and improved family dynamics.

Future Directions

School systems use levels of interventions to support school-aged children's behavior, social, emotional, and academic readiness. Traditional forms of school-based interventions consist of approaches (i.e., Positive Behavioral Interventions and Supports; PBIS) to promote and improve social, emotional, and academic outcomes for all students. Behavioral interventions (i.e., Applied Behavioral Analysis; ABA) implement classical forms of conditioning to alter behaviors or introduce an alternative appropriate behavior.

Additional forms of support targeting specific areas of student social and emotional development are provided by mental health providers within the school setting through individual or group therapy. The goals of these interventions are to enhance the skills and competencies of school-aged children to be successful in school. Each of these interventions involves using visual or auditory instructions designed to enhance learning. The consistent limitation of each of these traditional school-based interventions is that children must be able to process the intervention information. As discussed earlier, if a child is unable to process auditory or visual information even the best planned interventions may fail.

In the future, coupling neurofeedback interventions with school-based support may provide a more effective and individualized form of intervention. Furthermore, neurofeedback intervention in schools may provide educators with additional tools and knowledge to individualize student support. La Marca and O'Connor (2016) found that by using neurofeedback children were able to improve their reading comprehension and demonstrated more focused attention in the classroom.

Using the IVA-2 as an initial assessment tool will provide school interventionists with specific information on the student's auditory and visual processing strengths and weaknesses. With more specific information gleaned from the IVA-2, interventions can be developed targeting specific areas of weakness and used to modify the learning environment while the student is participating in a neurofeedback training program. In addition, the

neurofeedback assessment and treatment process would serve as an intervention providing additional documentation in determining whether or not there is a need for a special education assessment.

Development of a pilot project in the school setting is necessary to support students by identifying auditory and visual processing difficulties. Offering individualized neurofeedback training in the school setting would generate useful information regarding alternative interventions that may reduce referrals to Special Education. In other words, if neurofeedback and the IVA-2 assessment can identify children who have auditory and visual processing difficulties and effectively reduce these impairments, school districts may find a reduction in the need for Special Education services for this group of children. A reduction in the use of Special Education services would benefit the school district as well as benefiting the children if they are not in actual need of these specialized services.

Neurofeedback provides the opportunity for students to retrain their self-regulation abilities associated with social-emotional wellness (Huang-Storms, Bodenhamer-Davis, Davis, & Dunn, 2007), a process that is not typically accomplished with pharmaceutical interventions. Current estimates of annual out-of-pocket costs associated with traditional ADHD treatment approaches (i.e., medication and therapist visits) averaged $2,125 per child in 2012 (Doshi et al., 2012), which is approximately equal to 20 hours of neurofeedback training. When coupled with the average annual cost to society of $5,007 per child (Robb et al., 2011), the financial cost to schools, to families, and to society suggest that alternative treatments for ADHD are necessary to help reduce the large financial impact of ADHD (Doshi et al., 2012). The use of neurofeedback is a viable alternative that addresses the underlying associated problems of auditory and visual processing at the core of many of the symptoms of ADHD. Given the ongoing annual costs of traditional approaches to ADHD, neurofeedback becomes a viable and cost-reducing nonpharmaceutical alternative; even more so given the potential for long-term, ongoing benefits gained following neurofeedback treatment (Cannon & Lubar, 2011; Lubar, 1995).

The enhancement of social-emotional well-being has the capacity to improve a student's academic performance (Durlak, Weissberg, Dymnicki, Taylor, & Schellinger, 2011; La Marca & O'Connor, 2016). Additionally, by including neurofeedback as a form of support for school-aged children educators would have access to an alternative intervention. As a nonpharmacological intervention, neurofeedback provides a mechanism that teaches children how to function better by training their brain. Therefore, neurofeedback as a form of intervention in the school systems should be considered in future research directions.

Conclusion

The results of this study supported the hypothesis that neurofeedback would significantly improve both auditory and visual attention of children with symptoms of ADHD. The children's improvement in their auditory and visual attention scores revealed they achieved clinically significant improvements after 40 half-hour treatment sessions. Artifact corrected neurofeedback proved to be a clinically efficacious intervention that helps normalize the significant attentional impairments symptomatic of ADHD in children ages 6 to 17.

References

American Academy of Audiology. (2010). Clinical practice guidelines. *Diagnosis, treatment and management of children and adults with central auditory processing disorder*. Retrieved May 2016 from https://audiology-web.s3.amazonaws.com/migrated/CAPD Guidelines 8-2010.pdf_539952af956c79.73897613.pdf

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). Washington, DC: Author. Retrieved from http://dx.doi.org/10.1176/appi.books.9780890425596

Behavioural Neurotherapy Clinic. (n.d.). Central Auditory Processing Disorder (CAPD). Retrieved May 1, 2016, from http://www.adhd.com.au/CAPD.htm

Brazier, Y. (2015, November 25). "ADHD medication: Is it a good idea?" *Medical News Today*. Retrieved May 2016 from https://www.medicalnewstoday.com/articles/303090.php

Breslau, J., Miller, E., Chung, W.-J. J., & Schweitzer, J. B. (2011). Childhood and adolescent onset psychiatric disorders, substance use, and failure to graduate high school on time. *Journal of Psychiatry Research, 45*(3), 295–301. http://dx.doi.org/10.1016/j.jpsychires.2010.06.014

Cannon, R., & Lubar, J. (2011). Long-term effects of neurofeedback training in anterior cingulate cortex: A short follow-up report, *Journal of Neurotherapy, 15*(2), 130–150. http://dx.doi.org/10.1080/10874208.2011.570688

Chermak, G. D., & Musiek, F. E. (1997). *Central auditory processing disorders: New perspectives*. San Diego, CA: Singular Publishing Group, Inc.

Chronis-Tuscano, A., Molina, B. S. G., Pelham, W. E., Applegate, B., Dahlke, A., Overmeyer, M., & Lahey, B. B. (2010). Very early predictors of adolescent depression and suicide attempts in children with attention-deficit/hyperactivity disorder. *Archives in General Psychiatry, 67*(10), 1044–1051. http://dx.doi.org/10.1001/archgenpsychiatry.2010.127

Daley, D., van der Oord, S., Ferrin, M., Danckaerts, M., Doepfner, M., Cortese, S., & Sonuga-Barke, E. J. S. (2014). Behavioral interventions in attention-deficit/hyperactivity disorder: A meta-analysis of randomized controlled trials across multiple outcome domains. *Journal of the American Academy of Child*

& Adolescent Psychiatry, 53(8), 835–847.e5. http://dx.doi.org /10.1016/j.jaac.2014.05.013

Doshi, J. A., Hodgkins, P., Kahle, J., Sikirica, V., Cangelosi, M. J., Setyawan, J., … Neumann, P. J. (2012). Economic impact of childhood and adult attention-deficit/hyperactivity disorder in the United States. *Journal of the American Academy of Child & Adolescent Psychiatry, 51*(10), 990–1002.e2. http://dx.doi.org/10.1016/j.jaac.2012.07.008

Dunlop, A. J., & Newman, L. K. (2016). ADHD and psychostimulants—Overdiagnosis and overprescription. *The Medical Journal of Australia*, 204(4), 139. http://dx.doi.org /10.5694/mja15.01387

Durlak, J. A., Weissberg, R. P., Dymnicki, A. B., Taylor, R. D., & Schellinger, K. B. (2011). The impact of enhancing students' social and emotional learning: A meta-analysis of school-based universal interventions. *Child Development, 82*(1), 405–432. http://dx.doi.org/10.1111/j.1467-8624.2010.01564.x

Ellis, M. (2016, March 3). "ADHD medication and low bone density: Are kids at risk?" *Medical News Today*. Retrieved May 30, 2016, from https://www.medicalnewstoday.com /articles/307389.php

Epstein, V. (2015, June 17). "Visual processing disorder: Is this what your child has?" Kars4Kids Smarter Parenting. Retrieved May 2016 from http://www.kars4kids.org/blog /visual-processing-disorder-is-this-what-your-child-has

Evans, S. W., Owens, J. S., & Bunford, N. (2014). Evidence-based psychosocial treatments for children and adolescents with attention-deficit/hyperactivity disorder. *Journal of Clinical Child & Adolescent Psychology, 43*(4), 527–551. http://dx.doi.org/10.1080/15374416.2013.850700

Fabiano, G. A., Pelham, W. E., Coles, E. K., Gnagy, E. M., Chronis-Tuscano, A., & O'Connor, B. C. (2009). A meta-analysis of behavioral treatments for attention-deficit/hyperactivity disorder. *Clinical Psychology Review, 29*(2), 129–140. http://dx.doi.org/10.1016/j.cpr.2008.11.001

Farrar, R., Call, M., & Maples, W. C. (2001). A comparison of the visual symptoms between ADD/ADHD and normal children. *Optometry, 72*(7), 441–451.

Ghaziri, J., Tucholka, A., Larue, V., Blanchette-Sylvestre, M., Reyburn, G., Gilbert, G., … Beauregard, M. (2013). Neurofeedback training induces changes in white and gray matter. *Clinical EEG and Neuroscience, 44*(4), 265–272. http://dx.doi.org/10.1177/1550059413476031

Gleason, M. M. (2013). Finding the tools for effective early intervention for preschool attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child & Adolescent Psychiatry, 52*(3), 228–230. http://dx.doi.org /10.1016/j.jaac.2012.12.008

Grohol, J. M., (2013). Is ADHD overdiagnosed? Yes & no. Retrieved May 2016 from http://psychcentral.com/blog /archives/2013/11/21/is-adhd-overdiagnosed-yes-no/

Gyldenkærne, P., Dillon, H., Sharma, M., & Purdy, S. C. (2014). Attend to this: The relationship between auditory processing disorders and attention deficits. *Journal of the American Academy of Audiology, 25*(7), 676–687. http://dx.doi.org /10.3766/jaaa.25.7.6

Hagen, H., Moore, K., Wickham, G., & Maples, W. C. (2008). Effect on the EYEPORT system of visual function in ADHD children: A pilot study. *Journal of Behavioral Optometry, 19*(2), 37–41.

Hammond, D.C. (2011). What is neurofeedback: an update. *Journal of Neurotherapy, 15*(4), 305–336, http://dx.doi.org /10.1080/10874208.2011.62309

Hinshaw, S. P., & Ellison, K. (2016). *ADHD: What everyone needs to know*. New York, NY: Oxford University Press.

Hinshaw, S. P., Owens, E. B., Zalecki, C., Huggins, S. P., Montenegro-Nevado, A. J., Schrodek, E., & Swanson, E. N. (2012). Prospective follow-up of girls with attention-deficit/hyperactivity disorder into early adulthood: Continuing impairment includes elevated risk for suicide attempts and self-injury. *Journal of Consulting and Clinical Psychology, 80*(6), 1041–1051. http://dx.doi.org/10.1037/a0029451

Hinshaw, S. P., & Scheffler, R. M. (2014). *The ADHD explosion: Myths, medication, money, and today's push for performance*. New York, NY: Oxford University Press.

Huang-Storms, L., Bodenhamer-Davis, E., Davis, R., & Dunn, J. (2007). QEEG-guided neurofeedback for children with histories of abuse and neglect: Neurodevelopmental rationale and pilot study. *Journal of Neurotherapy, 10*(4), 3–16. http://dx.doi.org/10.1300/J184v10n04_02

International Statistical Classification of Diseases and Related Health Problems 10th Revision (ICD-10) Diagnosis Codes F90: Attention-deficit hyperactivity disorders. Retrieved April 2016 from https://www.icd10data.com/ICD10CM/Codes/F01-F99/F90-F98/F90-

La Marca, J. P., Cruz, D., Fandino, J., Cacciaguerra, F. R., Fresco, J. J., & Guerra, A. T. (2018). *Journal of Neural Transmission, 125*(7), 1087–1097. http://dx.doi.org/10.1007 /s00702-018-1877-1

La Marca, J. P., & O'Connor, R. E. (2016). Neurofeedback as an Intervention to improve reading achievement in students with attention-deficit/hyperactivity disorder, inattentive subtype. *NeuroRegulation, 3*(2), 55–77. http://dx.doi.org/10.15540 /nr.3.2.55

Lee, S. S., Lahey, B. B., Owens, E. B., & Hinshaw, S. P. (2008). Few preschool boys and girls with ADHD are well-adjusted during adolescence. *Journal of Abnormal Child Psychology, 36*(3), 373–383. http://dx.doi.org/10.1007/s10802-007-9184-6

Lubar, J. F. (1995). Neurofeedback for the management of attention-deficit/hyperactivity disorders. In M. S. Schwartz (Ed.), *Biofeedback: A practitioner's guide* (pp. 493–522). New York, NY: Guilford Press.

Maddux, C. D. (2010). [Review of the IVA+Plus: Integrated Visual and Auditory Continuous Performance Test]. In R. A. Spies, J. F. Carlson, & K. F. Geisinger (Eds.), *The eighteenth mental measurements yearbook* (pp. 434–437). Lincoln, NE: Buros Institute of Mental Measurements.

McCallum, W. C. (2015). Attention. *Encyclopædia Britannica*. Encyclopædia Britannica, Inc. Retrieved from http://www.britannica.com/science/attention/The-neurophysiology-of-attention

McReynolds, C. J., Bell, J., & Lincourt, T. M. (2017). Neurofeedback: A noninvasive treatment for symptoms of posttraumatic stress disorder in veterans. *NeuroRegulation, 4*(3–4), 114–124. http://dx.doi.org/10.15540/nr.4.3-4.114

Moore, D. R., Ferguson, M. A., Edmondson-Jones, A. M., Ratib, S., & Riley, A. (2010). *Nature of auditory processing disorder in children. Pediatrics, 126*(2), e382–e390. http://dx.doi.org/10.1542 /peds.2009-2826

MTA Cooperative Group. (2004). National Institute of Mental Health multimodal treatment study of ADHD follow-up: Changes in effectiveness and growth after the end of treatment. *Pediatrics, 113*(4), 762–769.

New Brunswick Department of Education. (1999). Resource for the identification and teaching of students with specific learning disability. Retrieved from https://www2.gnb.ca /content/dam/gnb/Departments/ed/pdf/K12/ResourceForIdentificationTeachingStudentsSpecificLearningDisability.pdf

Norman, D. A. (1982). *Memory and attention: An introduction to human information processing* (2nd ed.). New York, NY: Wiley.

Owens, E. B., Hinshaw, S. P., Lee, S. S. & Lahey, B. B. (2009). Few girls with childhood attention-deficit/hyperactivity disorder show positive adjustment during adolescence. *Journal of Clinical Child & Adolescent Psychology, 38*(1), 132–143. http://dx.doi.org/10.1080/15374410802575313

Pingault, J.-B., Tremblay, R. E., Vitaro, F., Carbonneau, R., Genolini, C., Falissard, B., & Côté, S. M. (2011). Childhood trajectories of inattention and hyperactivity and prediction of educational attainment in early adulthood: A 16-year

longitudinal population-based study. The *American Journal of Psychiatry, 168*(11), 1164–1170. http://dx.doi.org/10.1176/appi.ajp.2011.10121732

Poulton, A. S., Melzer, E., Tait, P. ., Garnett, S. P., Cowell, C. T., Baur, L. A., & Clarke, S. (2013). Growth and pubertal development in adolescent boys on stimulant medication for attention deficit hyperactivity disorder. *The Medical Journal of Australia, 198*(1), 29–32. http://dx.doi.org/10.5694/mja12.10931

Prasad, V., Brogan, E., Mulvaney, C., Grainge, M., Stanton, W., & Sayal, K. (2013). How effective are drug treatments for children with ADHD at improving on-task behaviour and academic achievement in the school classroom? A systematic review and meta-analysis. *European Child & Adolescent Psychiatry, 22*(4), 203–216. http://dx.doi.org /10.1007/s00787-012-0346-x

Riddle, M. A., Yershova, K., Lazzaretto, D., Paykina, N., Yenokyan, G., Greenhill, L., … Posner, K. (2013). The preschool attention-deficit/hyperactivity disorder treatment study (PATS) 6-year follow-up. *Journal of the American Academy of Child & Adolescent Psychiatry, 52*(3), 264–278.e2. http://dx.doi.org/10.1016/j.jaac.2012.12.007

Robb, J. A., Sibley, M. H., Pelham, W. E., Foster, E. M., Molina, B. S. G., Gnagy, E. M., & Kuriyan, A. B. (2011). The estimated annual cost of ADHD to the U.S. education system. *School Mental Health, 3*(3), 169–177. http://dx.doi.org/10.1007/s12310-011-9057-6

Sandford, J. A., & Sandford, S. E. (2014). *IVA-2: Integrated Visual and Auditory Continuous Performance Test Manual*. North Chesterfield, VA: Brain Train, Inc.

Saul, R. (2014). *ADHD Does Not Exist*. New York, NY: HarperCollins Publishers.

Sharma, M., Purdy, S. C., & Kelly, A. S. (2009). Comorbidity of auditory processing, language, and reading disorders. *Journal of Speech, Language, and Hearing Research, 52*, 706–722. http://dx.doi.org/10.1044/1092-4388(2008/07-0226)

Sonuga-Barke, E. J. S., Brandeis, D., Cortese, S., Daley, D., Ferrin, M., Holtmann, M., … European ADHD Guidelines Group. (2013). Nonpharmacological interventions for ADHD: Systematic review and meta-analyses of randomized controlled trials of dietary and psychological treatments. *The American Journal of Psychiatry, 170*(3), 275–289. http://dx.doi.org/10.1176/appi.ajp.2012.12070991

Steiner, N. J., Sheldrick, R. C., Gotthelf, D., & Perrin, E. C. (2011). Computer-based attention training in the schools for children with attention deficit/hyperactivity disorder: A preliminary trial. *Clinical Pediatrics, 50*(7), 615–622. http://dx.doi.org/10.1177/0009922810397887

Swanson, J. M., Arnold, L. E., Molina, B. S. G., Sibley, M. H., Hechtman, L. T., Hinshaw, S. P., … for the MTA Cooperative Group. (2017). Young adult outcomes in the follow-up of the multimodal treatment study of attention-deficit/hyperactivity disorder: Symptom persistence, source discrepancy, and height suppression. *The Journal of Child Psychology and Psychiatry, 58*(6), 663–678. http://dx.doi.org/10.1111/jcpp.12684

Swanson, J. M., & Volkow, N. D. (2009). Psychopharmacology: Concepts and opinions about the use of stimulant medications. *The Journal of Child Psychology and Psychiatry, 50*(1–2), 180–193. http://dx.doi.org/10.1111/j.1469-7610.2008.02062.x

Swingle, P. G. (2015). *When the ADHD diagnosis is wrong: Understanding other factors that affect attention in children*. Santa Barbara, CA: Praeger.

Tomlin, D., Dillon, H., Sharma, M., & Rance, G. (2015). The impact of auditory processing and cognitive abilities in children. *Ear and Hearing, 36*(5), 527–542. http://dx.doi.org/10.1097/AUD.0000000000000172

Visser, S. N., Zablotsky, B., Holbrook, J. R., Danielson, M. L., & Bitsko, R. H. (2015). Diagnostic experiences of children with attention-deficit/hyperactivity disorder. *National Health Statistics Reports; 81*, 1–7. Hyattsville, MD: National Center for Health Statistics.

Woliver, R., & Ibrahim, M. (2012). *Auditory processing disorder: The hidden disability*. Long Island Press. Retrieved May 2016 from http://newideas.net/auditory-processing-disorder

Zhonggui, X., Shuhua, S., & Haiqing, X. (2005). A controlled study of the effectiveness of EEG biofeedback training on children with attention deficit hyperactivity disorder. *Journal of Huazhong University of Science and Technology, 25*(3), 368–370. http://dx.doi.org/10.1007/BF02828171

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Appendix

Description of IVA-2 Global and Standard Composite Scores

The IVA-2 provides nine global composite quotient scores to help gain an in-depth understanding of the variety of ways ADHD-type problems may manifest (Sandford & Sandford, 2014).

IVA-2 Measures	Description of Measures
AAQ (Auditory Attention Quotient)	Based on equal measures of auditory Vigilance, Focus, and Speed
ARCQ (Auditory Response Control Quotient)	Derived from auditory Prudence, Consistency, and Stamina scales
FAQ (Full Scale Attention Quotient)	Based on six primary visual and auditory scales each based on equal measures of visual and auditory Vigilance, Focus, and Speed
FRCQ (Full Scale Response Control Quotient)	Based on six primary visual and auditory scales each and equal weights (not an average) of ARCQ and VRCQ scales
SAAQ (Sustained Auditory Attention Quotient)	Provides a global measure of a person's ability to respond to auditory stimuli under low demand conditions
SFAQ (Sustained Full Scale Attention Quotient)	Combined global measure of the SAAQ and SVAQ global scales
SVAQ (Sustained Visual Attention Quotient)	Provides a global measure of a person's ability to respond to visual stimuli under low demand conditions
VAQ (Visual Attention Quotient)	Based on equal measures of visual Vigilance, Focus, and Speed
VRCQ (Visual Response Control Quotient)	Derived from visual Prudence, Consistency, and Stamina scales

IVA-2 Scales	Description of Scales
Attention Primary Scales	
Vigilance	Measure of inattention as evidenced by two different types of errors of omission
Focus	Reflects the total variability of mental processing speed for all correct responses
Speed	Reflects the average reaction time for all correct responses throughout the test and helps to identify attention-processing problems related to slow discriminatory mental processing
Response Control Primary Scales	
Prudence	Measure of impulsivity and response inhibition as evidenced by three different types of errors of commission
Consistency	Measures the general reliability and variability of response times and is used to help measure the ability to stay on task
Stamina	Compares the mean reaction times of correct responses during the first 100 trials to the last 100 trials; this score is used to identify problems related to sustaining attention and effort over time

NeuroRegulation

The Effect of Passive Infrared Hemoencephalography on Athlete Performance

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Abstract

This single case study explores the effects of a specific form of biofeedback on sports enhancement. Three college athletes from three different sports (baseball, volleyball, and basketball) were each subjected to five weekly sessions of passive infrared hemoencephalography (pIR HEG) from a licensed psychotherapist who had been trained in this form of biofeedback. Sports data were collected prior to the session, during the sessions, and after the sessions. In addition, card sorting and thermal imaging were done by the therapist during each of the five brain-training sessions. The results were mixed. The baseball and volleyball players demonstrated modest gains in their specific sports measures and in the card-sorting process, whereas the basketball player's measures were flat. The thermal imaging was also inconclusive. However, two out of three subjects reported subjective improvements in focus and concentration on the field and in their daily lives. In addition, two of the subjects reported improvements in their rate and intensity of headaches, which was not a specific goal of the treatment, but one which is routinely seen from pIR HEG treatment. There are significant limitations to this study that make it impossible to generalize. Further studies with longer treatment times and larger numbers of subjects are recommended.

Keywords: sports enhancement; hemoencephalography (HEG); EEG biofeedback; neurofeedback (NF); passive infrared hemoencephalography (pIR HEG)

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The human brain is only 2% of one's body mass (Wang et al., 2014). The study of the brain is difficult for many reasons. One main reason is that we cannot manipulate someone's brain. The brain is so intriguing, and everyone thinks very differently, but we cannot cut open a healthy human to conduct research. That leaves all of the research up to people who have had a head trauma, stroke, mood disorders, headaches, and neurodegenerative disorders (Wang et al., 2014). From laboratory studies, researchers have found that almost all of the brain's processes are sensitive to temperature (Wang et al., 2014). To study the brain of brain-injured participants we need to be aware of the brain temperature to keep them safe and effective for the trial (Wang et al., 2014). If researchers fail to do so, the functional activity and energy efficiency of the brain will go down remarkably for the participant (Wang et al., 2014).

Biofeedback

Wouldn't it be useful to have instant feedback regarding aches and pains that go on in our brains without having to cut open one's head? Dr. Antoine Remond discovered that electroencephalography (EEG) helped to identify those with attention-deficit disorder (ADD) or people with minimal brain dysfunction (Siever, 2008). Hershel Toomim's research led him to develop instruments to measure brain temperature and galvanic skin response (now known as electrodermal response; Siever, 2008). In 1984, Toomim and Chuck Davis created the world's

first wireless biofeedback system (Siever, 2008). Biofeedback is a procedure that provides a person feedback over a bodily function that allows that individual to develop more control over that function to treat the medical problem (Siever, 2008). Toomim, however, found a further connection between cerebral blood flow and EEG (Siever, 2008), which led him to build the first ever blood oxygenation biofeedback device, which he named nIR hemoencephalography (HEG; Siever, 2008).

EEG Biofeedback

EEG biofeedback (hereafter referred to as neurofeedback or NF) training specifically looks at brain waves and how a participant receives instant results (Hammond, 2007). This type of training is very specific to the temporal, occipital, and parietal regions of the brain. Depending on where the electrodes are placed determines which skill the training is focusing on: balance, concentration, anxiety, etc. (Hammond, 2007). A study looked at performance enhancement in golfers using EEG profiles (Arns, Kleinnijenhuis, Fallahpour, & Breteler, 2008). They specifically looked at the central-temporal parietal areas and found that if the left-temporal alpha increased, the athlete's performance decreased, but if the right-temporal alpha increased, the athlete's performance increased (Arns et al., 2008). The results showed that each athlete performed differently even though they were completing the same task (Arns et al., 2008). Another study looked at performance in dancers by using EEG slow wave heart rate coherence training (Thompson, Steffert, Ros, Leach, & Gruzelier, 2008). Results showed that EEG slow wave training increased confidence, well-being, energy, respiratory control, and pitch (Thompson et al., 2008). Thompson and colleagues (2008) found the same results in singers and musicians. EEG slow wave training is great for relaxation therapy, but EEG fast wave training involves more visuomotor activities (Thompson et al., 2008). Another study conducted by Vernon in NF training found that, if experimenters made one hemisphere more active, then the other hemisphere becomes dominant (2005). By reducing the verbalizations in the left-temporal lobe, it increases the visual-spatial process in the right-temporal lobe, which raises the question: can shifting more positivity or negativity on a specific lobe improve performance (Vernon, 2005)? Vernon (2005) looked at pre-elite archers and found that the use of NF does enhance performance.

Hemoencephalography (HEG)

There are two approaches to HEG which is another form of biofeedback training but pertains more to the activation of the prefrontal cortex and its functions. Toomim et al. (2004) developed near infrared spectrophotometry (nIR HEG) and Carmen (2004) developed passive infrared hemoencephalography (pIR HEG).

One of Toomim's studies used "top down" training, which allows for the brain to shift from the damaged areas and activate different areas for problem solving. They used a spectrophotometer to access blood flow in the prefrontal cortex (Toomim et al., 2004). Toomim and colleagues (2004) specifically looked at attention, impulsivity, reaction time, and reaction time variability. Results showed that after nIR HEG training there was an increase in blood flow in the brain corresponding to the training sites (Toomim, 2004). The study was limited to specific brain dysfunctions due to the area of the brain researchers wanted to explore (Toomim, 2004).

Headaches and migraines have also been studied and treated with HEG. A migraine is a lingering persistent pain causing the individual to suffer (Walker & Lyle, 2016). In the United States 18% of women and 6% of men receive migraines daily (Stokes & Lappin, 2010). Each person's migraine can be different, but they are a frequent phenomenon that can be studied and treated. There is no cure for migraines but one common form of treatment for migraines is peripheral skin temperature biofeedback, blood volume pulse, and electromyography feedback (Stokes & Lappin, 2010). In a previous study conducted by Stokes and Lappin (2010), participants were interviewed for 30 min for every 10 sessions they completed. Participants were asked questions and were tested using different types of biofeedback treatments: thermal biofeedback devices such as the HEG machine and the hand warming units, blood volume pulse feedback, and electromyography feedback (Stokes & Lappin, 2010). The point of this study was that participants learned to control their headaches by simply warming their foreheads or raising their hand because the hand warmers were too hot (Stokes & Lappin, 2010). Stokes and Lappin (2010) found that even if none of the biofeedback treatments helped to reduce headaches, it did help participants to be able to control their headaches or even prevent them.

Another study conducted by Carmen utilized pIR HEG. Carmen (2004) had participants pick out a movie of their choice and wear a headset monitor to measure waste heat caused by increased blood flow to the prefrontal cortex. Once the participant got emotionally involved in the movie, their limbic system was activated and their prefrontal cortex went partially off-line. The headset monitor device picked up the decreased heat in the prefrontal cortex due to the reduced blood flow, and the computer paused the movie. The participant was then instructed to focus and relax at the same time. By doing so, the participant's limbic activation was reduced and the prefrontal cortex activation was increased which also increased the blood flow in that area and, once it reached a preset level, the movie began to play again. As the participants were able to handle longer pause times without getting fatigued, their brain learned to spontaneously strengthen the functioning of the prefrontal cortex and inhibit the limbic system more efficiently. This resulted in improvements in migraine headache frequency and intensity (Carmen, 2004). Results showed that this was a very useful and long-lasting way to treat migraines, but it would take at least six sessions to really get the training to stick (Carmen, 2004).

Athletes often get headaches due to the stress of their sport. There has been quite a bit of research conducted on athletes and concussions. A study conducted by Keightley and colleagues (2014) studied young children with concussions and their working memory. Working memory is tied into the prefrontal cortex because information only stays in our working memory for a few seconds. The prefrontal cortex deals with impulse decisions and personality. To be successful, athletes must be able to react quickly and efficiently to rapidly changing stimuli while maintaining a relaxed focus. Since pIR HEG is designed to improve prefrontal cortex efficiency, we became interested in whether this treatment would result in performance improvement for athletes.

Sports Enhancement

NF has been proven to improve sports performance by maximizing optimal brain function (Ross, 2015). Specifically, NF can improve an athlete's attention, focus, and emotional control, slow cognitive decline, improve sleep, and help to restore brain function after a traumatic injury (Ross, 2015). NF in general has made sports psychology very cutting edge and up to date. With more advanced technology we can target individual sports and explore specific effects on the individual athlete (Hammond, 2007).

Athletes must master the skills of stepping back and analyzing multiple sources of input in a short period of time and of having enough focus and concentration to act quickly and definitively. These skills primarily involve prefrontal cortex brain activities. In this study we focused on pIR HEG as developed by Jeffrey Carmen (2004), which targets the prefrontal cortex and its variety of functions, such as impulse control, organization of emotional reactions, personality, prioritizing, competing, and complex planning (McKinley, O'Loughlin, Pennefather-O'Brien, & Harris, 2015). Since pIR HEG improves prefrontal cortex efficiency, we are hypothesizing that this treatment will result in an improvement in these athletes functioning in their particular sport.

Method

Participants

Participants included one male undergraduate student athlete from each of the following teams: basketball, volleyball, and baseball at Briar Cliff University (n = 3). The average age of participants was 21 years old. The study was approved by the Internal Review Board (IRB) of Briar Cliff University where the first author was a student in the Psychology department.

Setting

The training took place in the second author's office. The remaining pieces of the experiment took place in practice (gym, field, or course, depending on the sport) and at different locations based on competition.

Materials

Materials included an ordinary deck of cards, a DVD movie (of the participant's choice), the EZPIR system (Jeff Carmen; Manlius, NY), and an infrared camera. The EZPIR system has two major components: the headset with a heat sensor connected to a computer through a hardware encoder and BioEra software (Proatech LLC, www.proatech.com). The camera utilized was the Seek Compact Pro made by Seek Thermal. This instrument was designed to connect to an iPhone and utilizes the internal computer power of the iPhone in order to process the images, thus producing a false color image that distinguishes between different levels of heat. The pIR HEG headband heat sensor picks up waste heat which is distributed through the forehead and thus indirectly measures increased blood flow to the prefrontal cortex.

Procedure

The experimenter recruited participants by asking permission from the coach and asking the athletes to participate in the study. The researcher told the athletes that the study would be measuring their athletic performance while they completed pIR HEG training.

Each participant signed the informed consent form before training began. Individually each participant completed training once a week. Training included sorting a deck of cards, by suit and then numerical order, while being timed. The participant was instructed to complete the task as fast as possible. The researcher explained how sorting, impulse control, and speed correlated to the prefrontal cortex. Then the researcher took a picture of the subject's face with special attention to the forehead utilizing the Seek Compact Pro. The researcher then explained what the picture meant; the darker (cooler) parts could mean fatigue or slight depression and the lighter (warmer) parts are how engaged your prefrontal cortex is at that moment. The goal was that by the end of the training the whole prefrontal cortex would be warm and evenly distributed across the forehead.

Next, the participant was instructed to choose a movie that they could become emotionally invested in. After the movie was selected the participant was guided to put on a headband with the heat sensor. The researcher helped to place the detector just above the eyebrows but not too far in the hairline and ensured that no hair was underneath the sensor on the forehead. The participant was then instructed to watch the movie and wait for further instruction. The computer software is programmed to pause the movie when the subject becomes emotionally activated, causing a less active prefrontal cortex blood flow. Once the movie paused, a bar graph would appear on the left side of the screen. After the bar appeared on the screen the participant was instructed to relax their body and concentrate on the bar graph. If the bar did not move up or continuously went down, the researcher instructed the participant to relax and concentrate specifically on the neck and shoulder muscles while continuing to focus on the bar graph. When the subject was successful in this endeavor, there would be increased blood flow in the prefrontal cortex; the sensor would pick up the increased waste heat; and the bar on the graph would rise. Once the bar reached the yellow line at the top of the graph, the movie would resume, which was a tangible reward for the subject. If at any point the participant felt tired or exhausted, they were instructed to tell the researcher immediately. After approximately 30 minutes of watching the movie, the researcher took another picture using the Seek Compact Pro. Then the researcher explained what the picture meant to the participant. The participant then scheduled another meeting for training.

Data Analysis

A multiple-baseline design was used to show the effects of treatment of performance in athletes. Due to the fact there were a variety of sports in this study, we could not begin each treatment at the same time. To demonstrate a treatment effect, we collected data during practice and not solely at competition. We collected three practice baseline points and one game point (depending on the team's games schedule) before starting treatment. Data was then collected once a week in practice, treatment, and then games in between treatment.

Results

Baseball

The results for the baseball player's stats were broken down into balls and strikes over a series of 20 pitches. During baseline he averaged 13 strikes and 7 balls in practice, as seen in Figure 1. He did not pitch in any games during baseline. Once treatment began, he averaged 14 strikes and 6 balls in practice and 15 strikes and 5 balls in games. Figure 1 shows the rise a steady trend of the practice strikes decreasing and the practice balls increasing. His card-sorting time did decrease steadily throughout treatment indicating a significant trend. Figure 2 shows the before and after of the infrared images.

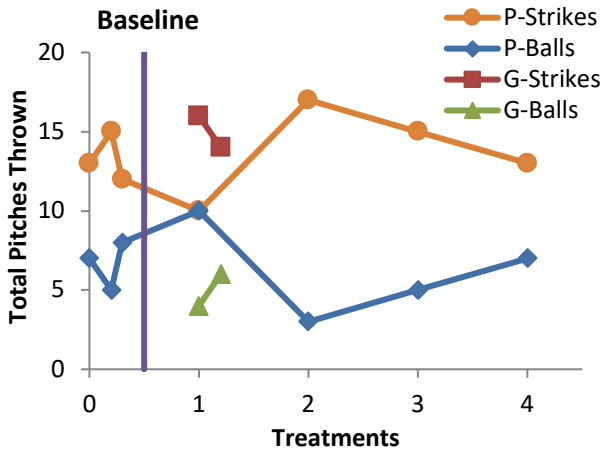

Figure 1. Baseball participant results during practice (P) and games (G) before and after treatments.

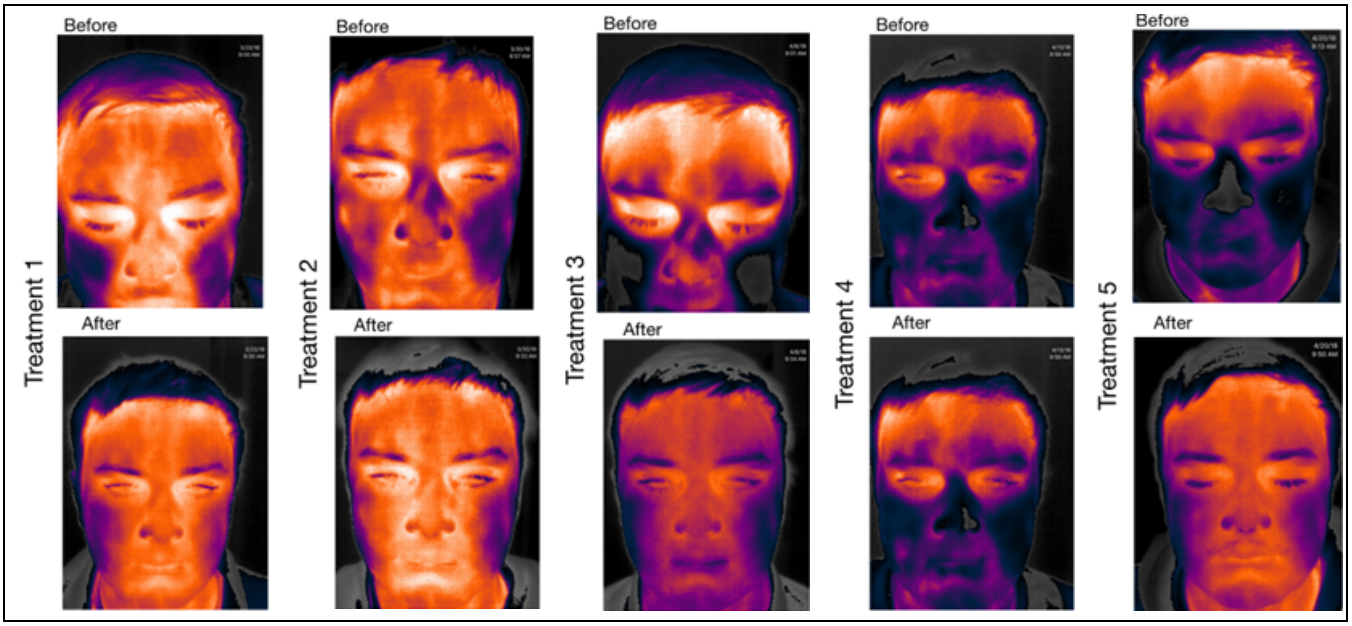

Figure 2. Baseball participant infrared images before and after treatments.

Volleyball

The results for the volleyball player's stats were broken down by passing rating, which is an average of all the passes he completed based on a 3-point rating scale. During baseline he averaged a 1.7 passing rate in practice and a 2.7 passing rate in a game, shown in Figure 3. Once treatment began, he averaged a 1.8 passing rate in practice and a 2.4 passing rate in a game. Figure 3 shows the consistency his passing became throughout treatment. His card-sorting time did steadily decrease causing a significant trend, meaning he steadily did improve throughout treatment; reasons will be discussed further in the Discussion. Figure 4 shows the before and after of the infrared images.

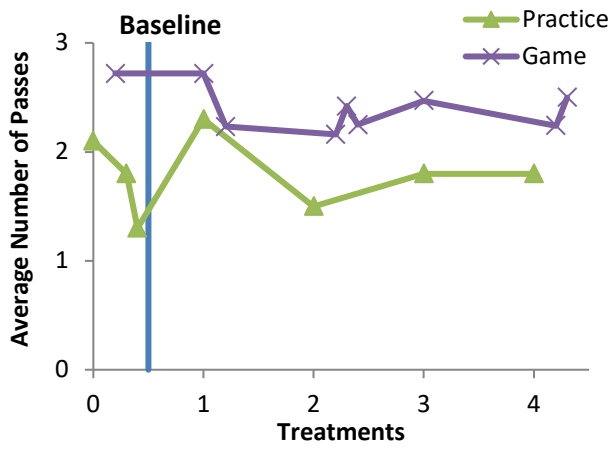

Figure 3. Volleyball participant results during practice (P) and games (G) before and after treatments.

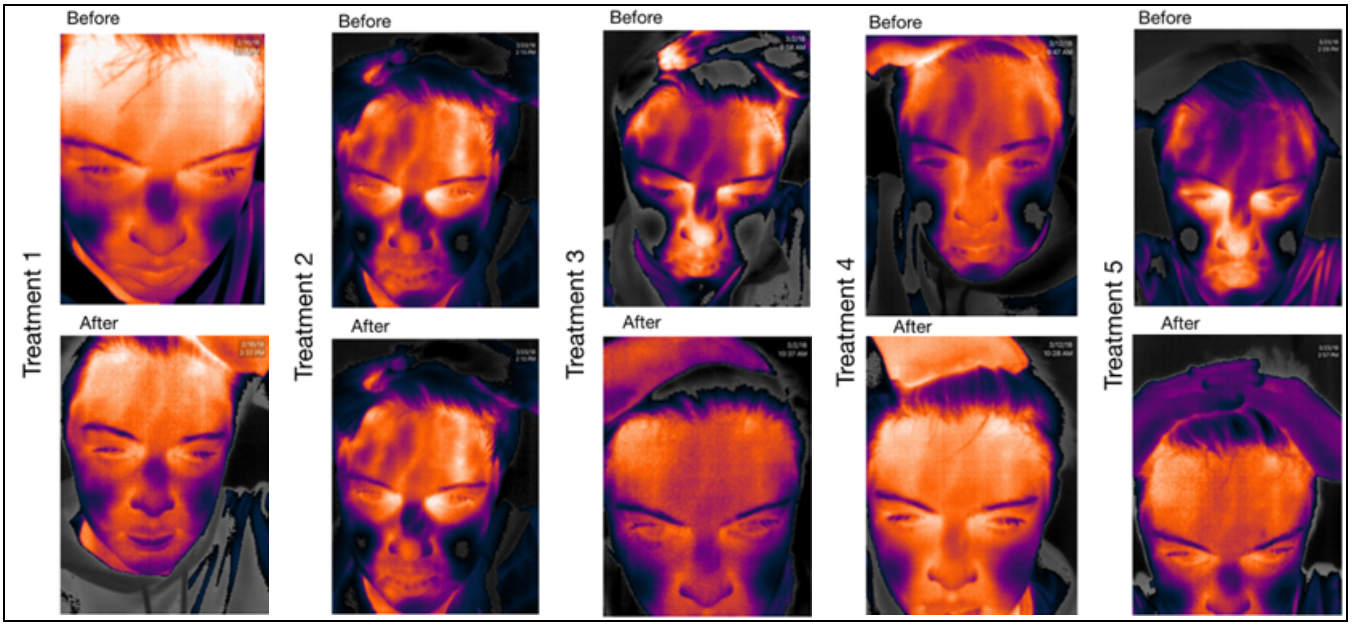

Figure 4. Volleyball participant infrared images before and after treatments.

Basketball

The results for the basketball player's stats were broken down by average free throws shot. During baseline he made 75.5% of shots in practice and 0% during the game, shown in Figure 5. The average percentage of shots made once treatment began was also 75% and average game percentage remained at 0. There was no change in percentage of shots made during games until two games posttreatment. His card-sorting time did decrease throughout treatment but not consistently enough to make a trend. Consequently, due to the lack of change in his statistics, treatment was not shown to be beneficial to this athlete in his game play. Figure 6 shows the before and after of the infrared images.

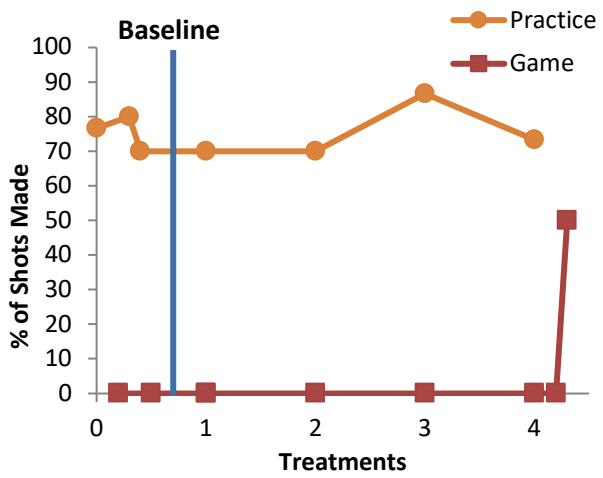

Figure 5. Basketball participant results during practice (P) and games (G) before and after treatments.

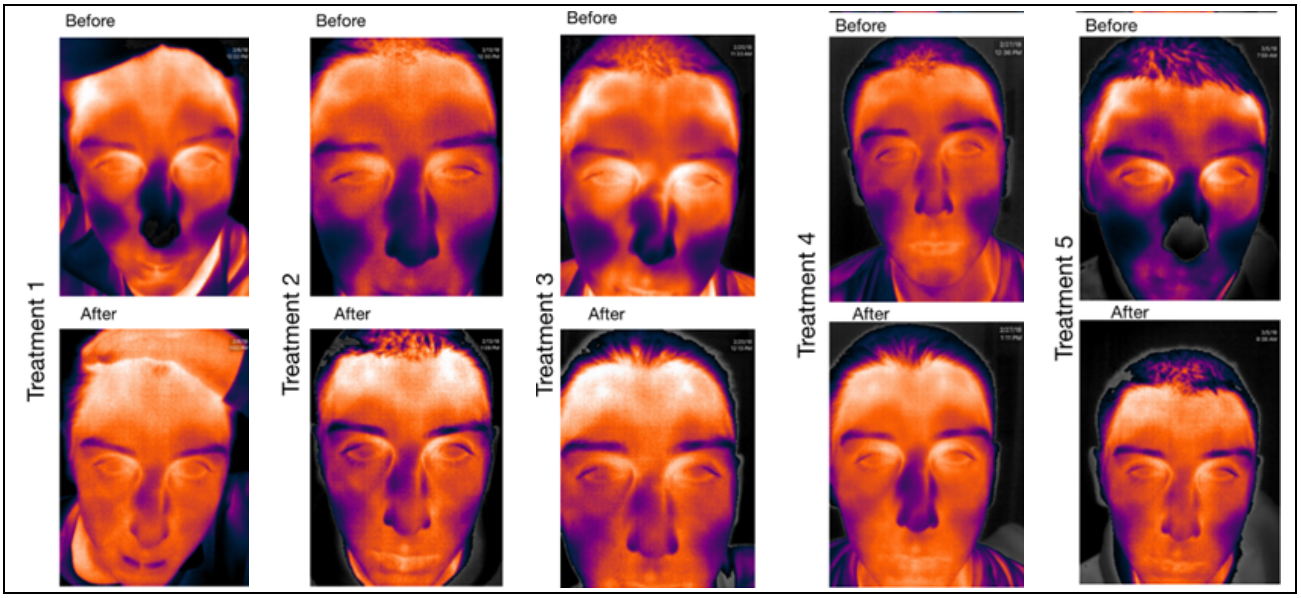

Figure 6. Basketball participant infrared images before and after treatments.

Social Validity

All three participants reported treatment had positively affected the way they see their game. The basketball player reported more concentration on how he shot the ball. The baseball player exclusively reported seeing an improvement in pitching strikes instead of balls. The volleyball player described an improved ability to "see the whole picture" during games and recalled a positive change in one specific game. All three did recall the mechanics of their sport improved. Along with their game play the baseball and volleyball player were able to concentrate on things outside of their game; e.g., homework, being more attentive in class, or demonstrating more initiative. Also, both the volleyball and baseball player reported to have had consistent migraines since grade school. Since beginning treatment, they have not had a migraine.

Discussion

In the current study we hypothesized that pIR HEG treatment will result in an improvement in these athletes' functioning in their particular sport. Our hypothesis was not fully supported. Participants were all chosen based on their playing time consistency, which allowed us to continuously collect data. Unfortunately, there is a lot of variability within a sport that did not allow us to generalize the sport overall. We did a nonconcurrent multiple baseline due to initial start date of the sport. Fortunately, we were able to have at least points of baseline before beginning treatment.

Secondly, we saw positive decreasing trends in the card-sorting task with the volleyball and baseball player. Both participants reported having consistent migraines since grade school and have reported they have decreased the frequency of them since beginning treatment. Additionally, they both reported to have carried the positive effects of this treatment into their everyday lives.

Unfortunately, there were quite a few limitations involved in this study and we are unable to generalize the results. There were consistent environment changes (e.g., weather, stress, gyms, etc.) that we could not control. Also, the length of study was a huge limitation. With the length of the semester and the initial starting times of each sport, we were forced to limit the study to 5 weeks. If the study would have been at least 8 weeks or longer we might have seen a stronger result. In addition, the measurement and interpretation of the infrared images was problematic (see Figures 2, 4, and 6). We needed a measurement that made it valid and reliable. The relatively inexpensive infrared camera used was capable of creating a false color spectrum, but it had limitations in being able to provide consistent images that could be compared.

We would recommend that future studies be conducted with longer periods of time, a larger

number of subjects, and increased number of pIR HEG sessions, ideally at least 10 to 12 weekly sessions. We would also recommend that a more expensive, sensitive infrared camera be used that would provide for a more reliable comparison of images.

If future studies would focus on just one sport, it would make it more likely that some generalization of that specific sport could be drawn. For example, a study could conduct treatment on several different basketball players playing different positions. Since each position has unique challenges and different stress levels, the results could more readily generalize to the sport of basketball.

Due to the diverse functionality of the prefrontal cortex of the brain, we believe that there are many creative ways in which further studies of pIR HEG's effects on sports enhancement would be useful and would encourage others to design such studies.

References

Arns, M., Kleinnijenhuis, M., Fallahpour, K., & Breteler, R. (2008). Golf performance enhancement and real-life neurofeedback training using personalized event-locked EEG profiles. *Journal of Neurotherapy, 11*(4), 11–18. http://dx.doi.org/10.1080/10874200802149656

Carmen, J. A. (2004). Passive infrared hemoencephalography: Four years and 100 migraines. *Journal of Neurotherapy, 8*(3), 23–51. http://dx.doi.org/10.1300/J184v08n03_03

Hammond, D. C. (2007). Neurofeedback for the enhancement of athletic performance and physical balance. *The Journal of the American Board of Sports Psychology, 1-2007*, 1–9.

Keightley, M. L., Saluja, S. R., Chen, J.-K., Gagnon, I., Leonard, G., Petrides, M., & Ptito, A. (2014). A functional magnetic resonance imaging study of working memory in youth after sports-related concussion: Is it still working? *Journal of Neurotrauma, 31*(5), 437–451. http://dx.doi.org/10.1089/neu.2013.3052

McKinley, M. P., O'Loughlin, V. D., Pennefather-O'Brien, E. E., & Harris, R. T. (2015). *Human anatomy* (4th ed.). New York, NY: McGraw-Hill Education International.

Ross, J. (2015, June 4). "5 ways neurofeedback improves sports performance." *Advanced Neurotherapy*. Retrieved from https://www.advancedneurotherapy.com/blog/2015/06/04/sports-performance-neurofeedback

Siever, D. (2008). History of biofeedback and neurofeedback. *Biofeedback, 36(2)*, 74-81.

Stokes, D. A., & Lappin, M. S. (2010). Neurofeedback and biofeedback with 37 migraineurs: A clinical outcome study. *Behavioral and Brain Functions, 6*(9), 1–10. http://dx.doi.org/10.1186/1744-9081-6-9

Thompson, T., Steffert, T., Ros. T., Leach, J., & Gruzelier, J. (2008). EEG applications for sport and performance. *Methods, 45*(4), 279–288 http://dx.doi.org/10.1016 /j.ymeth.2008.07.006

Toomim, H., Mize, W., Kwong, P. C., Toomim, M., Marsh, R., Kozlowski, G. P., … Rémond, A. (2004). Intentional increase of cerebral blood oxygenation using hemoencephalography (HEG): An efficient brain exercise therapy. *Journal of Neurotherapy: Investigations in Neuromodulation, Neurofeedback and Applied Neuroscience, 8*(3), 5–21. http://dx.doi.org/10.1300/J184v08n03_02

Vernon, D. J. (2005). Can neurofeedback training enhance performance? An evaluation of the evidence with implications for future research. *Applied Psychophysiology and Biofeedback, 30,* 347–364. http://dx.doi.org/10.1007/s10484-005-8421-4

Walker, A. K., & Lyle, R. R. (2016). Passive infrared hemoencephalography (pIR HEG) for the treatment of migraine without aura. *NeuroRegulation, 3*(2), 78–91. http://dx.doi.org/10.15540/nr.3.2.78

Wang, H., Wang, B., Normoyle, K. P., Jackson, K., Spitler, K., Sharrock, M. F., … Du, R. (2014). Brain temperature and its fundamental properties: A review for clinical neuroscientists. *Frontiers in Neuroscience, 8*, 307. http://dx.doi.org/10.3389/fnins.2014.00307

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NeuroRegulation

Perspectives on Placebo: The Psychology of Neurofeedback

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Abstract

The application of operant conditioning with EEG variables to produce changes in behavior has been gaining increasing interest in research and application areas. However, the methodology has come under scrutiny and criticism for its potential placebo effects. This article will examine those issues from the traditional methodologies of demonstrating effectiveness (control group, sham treatments) as well as examine the possible biochemical and electrophysiological effects of a placebo response. Specifically, the role of endorphins and dopamine and their relationship to the alpha and beta frequency in the placebo response will be examined. The research addressing the diffusion tensor imaging (DTI) and functional magnetic resonance imaging (fMRI) correlates of the intervention will be examined.

Keywords: neurotherapy; EEG biofeedback; placebo; alpha; endorphins; dopamine

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Definitions of Placebo and Effects

The common thread of varying definitions is that a placebo has (a) a positive effect on the patient's perception and self-report; and (b) does not have any physical beneficial effects on the illness for which it is prescribed.

Thibault and Raz (2017) conclude that "EEG-nf works, but it likely relies heavily on placebo phenomena" (p. 683). They further argue that "mental health professionals stand to benefit from studying the ubiquitous placebo influences that likely drive these treatment outcomes" (p. 679). "EEG-nf studies largely neglect investigating treatment mechanisms that rely on participant motivation, belief in the treatment administered, interacting with a practitioner, level of positive feedback, and sense of control of their brain signal" (p. 684). This paper will investigate the logic and evidence regarding the potential placebo effect in the neurotherapy (NT) situation.

In order to demonstrate a placebo effect, a research project must meet the following criteria document: (a) the expectation of the subject or study characteristics which encourage such an expectation; (b) the concomitant biological correlates (neurochemical, EEG, fMRI, DTI correlates of the expectation); and (c) the lack of a relationship between the biological correlates of the placebo and the biological correlates of the illness in question. To document that the effect is a real, substantive effect, (d) the results must distinguish between the effects of the intervention which have no measurable physical effect on the problem in question (a placebo) and those interventions which have a direct measurable physical effect on the problem.

It is clear that there is a real physiological effect from a placebo. The mechanism is not the pill or the treatment but rather the subject's perception that there is a value (positive/negative) in the pill or treatment. It is this belief that creates a change in the physiology of the brain and thus underlies the

perceived changes in symptoms. It is the goal of this paper to understand the relationship between the perception of change and the underlying electrophysiology and biochemistry of that change. We will address what is known/unknown about this relationship. If NT is a placebo response, then it is necessary to document the physiology of placebo response and its relationship to reported changes obtained with NT.

There is some inherent ambiguity built into the question of whether NT is a placebo in the traditional sense. It should be noted that the placebo argument originated in drug research addressing physical conditions, thus a physical intervention (pill, etc.) addressing a physical condition. NT is not a pill addressing a physical condition but an operant conditioning method to change the EEG signal which is related to behavior (such as attention). In both situations, however, the concern is whether the subject's subjective response is influencing the results which are tied to the relevant behavior issue (attention, etc.) and how much. In the case of attention-deficit disorder (ADD), if the "illness" is excessive theta activity and deficient beta and a placebo or NT changes the theta and beta values and relevant behavior, then the question is: Has a "cure" occurred? It is not assumed that the theta/beta issues are the sole physical issue in the attention-deficit/hyperactivity disorder (ADHD) subject.

This paper will examine research on the physical/physiological effects related to the placebo response (endorphins, dopamine, EEG signal) and determine if it is credible to account for the NT effects as a placebo.

Placebos work on patient's perceptions

Hróbjartsson and Gøtzsche (2010) reviewed the literature on placebos for 60 clinical conditions and concluded that there was a placebo effect in the clinical conditions of pain, nausea, asthma, and phobia but no effect in smoking, dementia, depression, obesity, hypertension, insomnia, and anxiety. Thus, only 7% of the conditions studied had a documented placebo effect. The perception of pain condition appears to be most responsive to the placebo effect (approximately 30 to 60 percent of people report a positive effect; Cherry, 2018).

In addition, not all subjects respond to the placebo effect. Beecher (1955) reviewed 26 studies and found on average 32% of the patients responded to placebo. In addition, it appears that some subjects are not responsive to the placebo effect due to genetics. "Predisposition to respond to placebo treatment may be in part a stable heritable trait" (Hall, Loscalzo, & Kaptchuk, 2015, p. 20).

Rossi (1986) concluded that "placebo is about 55–60% as effective as active medications irrespective of the potency of these active medications." Rossi also mentions that "in a study of morphine, there was a 50% pain reduction in 75% of the patients treated. The placebo group had a 50% pain reduction in 36% of the patients" (WRF, n.d.)

Kaptchuk et al. (2010) studied two groups of irritable bowel syndrome (IBS) patients. One group did not receive any treatment, while the other group was provided with pills labeled placebo and were told they were "fake, inert drugs" and that the "placebo pills, something like sugar pills, have been shown in rigorous clinical testing to produce significant mind-body self-healing processes'' (Kaptchuk et al., 2010). The group receiving the fake placebo drugs reported twice as much symptom relief as the no treatment group and comparable to the best real medicine for IBS. According to Feinberg (2013), Kaptchuk's interest is "not if, but how, placebo effects work," as Kaptchuk argues that "sham treatment won't shrink tumors or cure viruses" (p. 36).

Wechsler, Kelley, and Kaptchuk (2011) studied 40 asthma patients with four different interventions. They found that only the real treatment showed results. However, there was no difference between the patient's subjective response to the real versus sham treatments. The patient's subjective response was not consistent with the objective measures. Thus, the placebo does not cure the problem but does result in a patient's perception of change.

The argument that NT is solely a placebo effect for all of the participants who have been involved in the treatment must be understood in the context that the placebo effect has only been documented in 7% of the conditions, by 32% of the patients, is determined, in part, by genetics and is about half as effective as morphine (in terms of patient response rates). Thus, the "ubiquitous placebo influences" are not found everywhere.

Placebo's Physical Effects on the Body and Brain

The placebo's physical effects on activity in brain structures has been documented in Parkinson disease (Benedetti et al., 2004; de la Fuente-Fernández et al., 2001), pain (Eippert et al., 2009;

Petrovic, Kalso, Petersson, & Ingvar, 2002; Wager et al., 2004; Zubieta et al., 2005), depression (Leuchter, Cook, Witte, Morgan, & Abrams, 2002), and anxiety (Furmark et al., 2008; Petrovic et al., 2005). The anxiety and depression results are in contradiction to the Hróbjartsson and Gøtzsche Cochrane review. The hypothesis resulting from these studies suggest a prefrontal control mechanism that effects activity and neurotransmitters. A full understanding of the structural, functional, biochemical, and electrophysiological effects is required.

"Researchers have found that placebo treatments—interventions with no active drug ingredients—can stimulate real physiological responses, from changes in heart rate and blood pressure to chemical activity in the brain, in cases involving pain, depression, anxiety, fatigue, and even some symptoms of Parkinson's" (Feinberg, 2013).

Endorphins

Levine, Gordon, Jones, and Fields (1978) first reported that blocking the release of endorphins (chemically) by opioid antagonist naloxone would stop the placebo effect. Endorphins are the brain's natural pain relievers and are similar to morphine and opiates.

Benedetti et al. (2004) concluded that "there is ample evidence that expectancy-based placebo effects are mediated by endogenous opioids." Levine et al. (1978) noted that naloxone blocks the brain's ability to soak up endorphins. Volavka, James, Reker, and Cho (1979) showed that "naloxone elicited a significant slowing of the average alpha frequency" (p. 1267). This slowing effect would most likely be manifested in the lowering of the peak frequency of alpha. Thus, the alpha frequency appears to be involved in the placebo/endorphin response. The interrelationships between different measures of the alpha frequency (magnivolts, relative power, peak frequency) pose a complex problem, which is beyond the scope of this paper.

Lipman et al. (1990) reported that, in chronic pain subjects, the placebo responders had higher concentrations of endorphins in the cerebrospinal fluid than the placebo non-responders. Wager, Scott, and Zubieta (2007) concluded that placebo effects involve opioid activation in opioid receptor rich regions including "periaqueductal gray and nearby dorsal raphe and nucleus cuneiformis, amygdala, orbitofrontal cortex, insula, rostral anterior cingulate, and lateral prefrontal cortex" (p. 11056). Thus, the placebo effects involve a similar activity or results suggest that endogenous opioid release in core affective "brain regions is an integral part of the mechanism whereby expectancies regulate affective and nociceptive circuits" (p. 11056).

However, it has also been reported that the placebo effect can occur without the involvement of opioids and naloxone can only partially inhibit the placebo analgesia effect in some situations (Amanzio & Benedetti, 1999; Gracely, Dubner, Wolskee, & Deeter, 1983; Grevert, Albert, & Goldstein, 1983).

The relationship between endorphins and the EEG has not been studied directly. However, there is indirect evidence that the endorphins increase the level of alpha magnitudes. Mimasa et al. (1996) "found that the larger the changes in beta-endorphin following exercise, the higher the appearance rate of the alpha wave in EEG. There was a positive and significant correlation (r = .563, p < .05) between the increase in alpha wave component and that of the plasma beta-endorphin." It is unclear whether the increase was a magnitude or relative power increase. Thornton (2016) reported a positive correlation (r = .66, p < .05) between magnitude and relative power measures of alpha. However, Crabbe and Dishman (2004) were able to document that exercise results in increased alpha magnitudes. Pfefferbaum et al. (1979) found that "beta-endorphin and morphine produced similar increases in alpha power within 5 to 15 minutes after injection."

Peniston and Kulkosky (1989) compared a nonalcoholic control group, a traditionally treated alcoholic control group, and alcoholics receiving brainwave training (BWT). The BWT group showed significant increases in percentages of EEG record in alpha and theta rhythms (the focus of the intervention), increased alpha rhythm amplitudes, and improved outcome compared to the other two groups.

However, the alcoholics receiving abstinence, group psychotherapy, or antidepressants showed a significant elevation in serum beta-endorphin levels at the conclusion of the experiment. "This neuropeptide is an index of stress and a stimulant of caloric (e.g., ethanol) intake" (Peniston & Kulkosky, 1989, p.271). "Beta-endorphins play a role in certain behavioral patterns (stress, alcoholism), in obesity, diabetes, and psychiatric diseases" (Peniston & Kulkosky, 1989). Dalayeun, Norès, and Bergal (1993) application of brainwave treatment, a relaxation therapy, appears to counteract the increase in circulating beta-endorphin levels seen in the control

group of alcoholics. This partially explained result stands in contrast to the expected increases in alpha associated with increases in endorphin levels.

It appears that the placebo effect involves endorphins and alpha magnitudes, relative power and peak frequency. Thus, when a subject believes an intervention is going to be successful, the endorphins increase and the magnitudes of alpha increase, as well as probably other frequencies. It remains unclear whether the increase in alpha magnitudes is a result of the placebo effect or NT. Even an increase in the alpha and theta magnitudes during a session or treatment period is not an argument against a placebo effect as the argument could be made that the placebo's effect increases during the session gains or as more sessions are employed.

Dopamine

De la Fuente-Fernández et al. (2001) found that placebo-induced expectation of motor improvement activates endogenous dopamine in the striatum of Parkinsonian patients. Additional studies have pointed to dopamine involvement in the placebo response in pain conditions (Scott et al., 2008; Zubieta et al., 2005; Zubieta & Stohler, 2009).

Hall et al. (2012) stated that "Catechol-O-methyltransferase (COMT), an important enzyme in dopamine catabolism, plays a key role in processes associated with the placebo effect such as reward, pain, memory, and learning." As "COMT activity decreased, theoretically making more dopamine available in the prefrontal cortex, placebo responses increased in a linear fashion…associated with a positive outcome only in groups given a placebo (and not in the waitlist control group) is of particular importance, as it indicates that it is a predictor of the placebo effect, not just improvement in general" (Hall et al., 2012).

Dopamine has been shown to be part of the ADHD condition. An increase in psychostimulants should result in increases in dopamine levels and increases in EEG arousal frequencies (alpha and beta) and decreases in lower frequencies (delta and theta). "Methylphenidate: Ritalin produces a decrease in delta and theta, with a more pronounced posterior alpha increase and an increase in low beta, with effects delayed up to 6 hours, compared to the rapid effects of the amphetamines" (Gunkleman, 2009, p. 4). "Medication resulted in normalization of theta power, but, after medication, increased relative beta was also apparent in the female ADHD group" (Clarke, Barry, McCarthy, Selikowitz, & Johnstone, 2007). This result was also obtained in adults Bresnahan, Barry, Clarke, and Johnstone (2006), who reported that following medication, there was a "significant reduction in slow wave activity in the ADHD adult group to levels similar to those in the control group."

However, these findings have not been without contrary evidence. Lubar, Swartwood, Swartwood, and Timmermann (1996) failed to show increases in qEEG indicators of cortical arousal with methylphenidate. Other researchers (Barkley, 1998; Ernst et al., 1994; Matochik et al., 1994) have failed to demonstrate the neurophysiological effects of Ritalin at the cortical level.

There is solid research in the efficacy of operant conditioning (Staddon & Cerutti, 2003). The research has documented that NT can increase beta magnitudes and decrease theta magnitudes as well as increase alpha (Lubar et al., 1996; Sherlin, Arns, Lubar, & Sokhadze, 2010). The effect can be maintained up to 2 years (Gani, Birbaumer, & Strehl, 2008; Leins et al., 2007) and is maintained after cessation of medication (Monastra, Monastra, & George, 2002). Endorphins and dopamine have short-term effects. Foley et al. (1979) reported the half-life of beta-endorphin was 37 to 93 minutes. "The half-life of dopamine effect is 2 min" (ADHB, 2018). It is conceptually challenging to understand how it is possible that the placebo effect could be manifested 2 years later.

In conclusion, activation of an endorphin response appears tied to the alpha frequency, while dopamine's effects are predominantly in the beta frequency. NT and medications appear to break apart the naturally positive relationships between the magnitudes of the theta, alpha, and beta frequencies.

Neurotherapy and Structural/Functional Changes on fMRI, MRI, and DTI

Lévesque, Beauregard, and Mensour (2006) studied twenty ADHD unmedicated children who were divided between the experimental group (EXP; N = 15) and control (CON; N = 5) and baseline measure were obtained on two cognitive measures (Digit Span, Integrated Visual and Auditory [IVA] Continuous Performance Test [CPT]) and two questionnaire variables (Conners Parent Rating Scale, inattention and hyperactivity component). Following 40 sessions of NT, inhibiting theta microvolts (4–7 Hz) and rewarding beta microvolts (12–15 Hz, 15–18 Hz) at Cz, there were significant improvements in the EXP

group on the two cognitive and two questionnaire variables. In addition, a repeat fMRI scan was conducted while they (EXP and CON) performed a Counting Stroop task. The initial evaluation indicated significant increases in both groups in the left superior parietal location (Brodmann area 7). Postintervention fMRI scan revealed that the EXP group had increased activation of BA 24b-c and 32, which involve the anterior cingulate cortex, known to be involved in attentional issues (Bush et al., 1999). The CON group "did not receive an attentional training lasting the same time duration than the NT received by EXP subjects," which the authors suggested needs to be further evaluated (Lévesque et al., (2006). Thus, the NT intervention showed positive behavioral attentional and relevant fMRI results, with some qualifications regarding the CON group intervention.

In this study, the posttreatment Counting Stroop task requested the subjects to indicate the number of words presented (neutral condition) and then provided with an interference condition, during which the words *one*, *two*, *three*, and *four* were presented. The analysis compared the interference condition to the neutral condition. For both the EXP and CON groups, there was a significant locus of activation in the left superior parietal lobule (BA 7; fMRI) at both time periods. For the EXP group, at time 2, there were significant activations in the right BA 32 and left the caudate nucleus. Banich et al. (2000) reported that the anterior cingulate cortex is employed in selecting an appropriate response and allocate attentional resources. BA 32 is located in the anterior cingulate cortex. The caudate nucleus is involved in the inhibitory control of action (Nestler, Hyman, & Malenka, (2009). Thus, NT interventions appear to have an additional and relevant effect on brain structures that were not present for the CON group. Criteria #4, presented in this article, argues that if a treatment results in relevant change then it is real and not a placebo result. The NT intervention appears to have met that criteria, apart from the CON group intervention qualification.

Ghaziri et al. (2013) examined white matter (WM) and grey matter volume (GMV) in a sample of 30 participants under EXP, sham, and CON group conditions. They employed NT rewarding 15–18 Hz at F4 and P4 for 40 sessions (30 min each). Pre- and posttreatment data were available for the Integrated Visual and Auditory (IVA) test. The EXP and sham treatment showed significant increases on the IVA, while the sham group showed significant increases in the visual attention measure. The CON showed no significant improvements on either measure. The areas of interest for the structural magnetic resonance imaging (MRI) study and the fractional anisotropy (FA) measure of DTI involved the cingulum bundle (CB), superior longitudinal fasciculus (SLF), inferior longitudinal fasciculus (ILF), and the splenium of the corpus callosum (SCC).

For the EXP group, there were significant increases in fractional anisotropy (FA) in the right and left CB, right anterior corona radiata and SCC as was as left SLF and ILF. "These WM pathways are known to be associated with sustained attention" (Ghaziri et al., 2013, p. 269).

This result satisfies criteria (d) mentioned at the beginning of this paper which states "must distinguish between the effects of the intervention which have no measurable physical effect on the problem in question (a placebo) and those interventions have a direct measurable physical effect on the problem." In this case, the EXP group had a direct measurable physical effect on the problem of attention, as the affected "WM pathways are known to be associated with sustained attention." Thus, the intervention must be viewed not as a placebo but a valid, relevant intervention on the "cause" of the problem—WM pathways.

The visual attention performance correlated with the FA measures in the left SLF and left ALIC (left anterior limb of the internal capsule). In the sham and CON conditions, there were no significant FA increases at posttesting.

For the EXP group, the GMV measures showed increases in Brodmann areas 9, 20, 19, 6, 47, 22 and 7 while the sham group showed increases in BA 10, 6, and 18. Thus, the EXP group activated BA areas 9, 19, 20, 22, and 47 that the sham group did not. The sham group received training "for approximately 20 hours, participants in the SHAM group had to undergo a perceptual-cognitive 'training', consisting of staring at the computer screen and staying focused with respect to the animation displayed on that screen. The members of this group also received hours of personal coaching to pay attention visually" (Ghaziri et al., 2013, p. 269). This coaching, perhaps, explains their improvement on the visual attention IVA. Thus, the sham group did not result in increased communication patterns (increases in FA values) but did result in increased GMV in frontal areas (BA areas 10 and 6) while the EXP group increased frontal GMV in BA 9 and 47. The study was the "first empirical demonstration that NT can lead to microstructural changes in white and gray matter" (Ghaziri et al.,

2013). This study had the sham group engaged in training which addressed the problem in the Lévesque et al. (2006) study with the control group.

The lack of significant FA increases in the sham condition presents a serious challenge to the NT as placebo argument. This result indicates that the NT intervention is resulting in a substantive change in the physical functioning (FA) of the brain while the sham intervention does not. This is evident in both the communication measure (FA) and different GMV increases, reflecting that the NT approach is fundamentally changing. The presence of a sham condition argues definitively against a placebo effect.

Gevensleben, Holl, Albrecht, Schlamp, et al. (2009) and Gevensleben, Holl, Albrecht, Vogel, et al. (2009) studied 94 ADHD children with a randomization approach involving multicenters and a sham condition (a computerized attention skills training). The NT intervention involved decreasing theta (4–8 Hz) and increasing beta (13–20 Hz). The post-qEEG data showed reduced theta power, demonstrating efficacy and specificity. Gevensleben et al. (2010) conducted a 6-month follow-up on the 2009 study and found that the improvements in the NT group were comparable to the effects at the end of the training period (effect size of .71), employing parent rating scales. Thus, the maintenance of effect at the 6-month time period is problematic for a placebo explanation in addition to the reduction of theta magnitudes which is not concurrent with a reduction in beta magnitudes, the naturally occurring pattern.

Clinical Conditions

We will examine the different clinical conditions to determine if the NT effects can justifiably be called a placebo effect.

ADD

The ADD condition has been the subject of many investigations with modern medical imaging technology. Initial research focused on the theta/beta values. Eyes-closed, resting EEG data indicated that higher relative power of theta and reduced relative power of alpha and beta, as well as elevated theta/alpha and theta/beta ratios being associated with ADD/ADHD (Barry, Clarke, & Johnstone, 2003). There are other studies supporting this pattern (Chabot & Serfontein, 1996; Matsuura et al.,1993). Several EEG research reports have shown that beta activity is related to sustained attention (Arruda, Zhang, Amoss, Coburn, & Aue, 2009; Molteni, Bianchi, Butti, Reni, & Zucca, 2007), thus relevant to the ADHD's problem in attention.

NT approaches (combined theta/beta training with the training of slow cortical potentials, SCPs) obtained a reduction of theta activity (Gevensleben et al., 2010). Improvements on the EEG measures was associated with improvements on an ADHD rating scale. Similarly, Monastra et al. (2002) reported a decrease of the theta/beta quotient in a group of children with ADHD with an initially enhanced theta/beta quotient. The clinical value of the theta/beta ratio resides in its high correlation (r = .99) and age-related changes in the ADHD behavioral symptomatology (Snyder & Hall, 2006).

More recently, the research has focused on deficits in white matter tracts (Hamilton et al., 2008; Konrad et al., 2012; Niogi, Mukherjee, Ghajar, & McCandliss, 2010; Pavuluri et al., 2009; Qiu et al., 2011). Niogi, Mukherjee, Ghajar, and McCandliss (2010) conducted a DTI study (healthy participants) and showed a positive correlation between FA values in the anterior limb of the internal capsule and performance on a sustained attention task.

Kong et al. (2006) has reported DTI research on ADHD which reported significantly lower FA values in the left hemisphere involving the frontoparietal networks (SLF, ILF, and CB), or are implicated in interhemispheric processing within parietal areas (SCC). Van Ewijk, Heslenfeld, Zwiers, Buitelaar, and Oosterlaan (2012) conducted a meta review of the DTI area and ADHD and concluded that "alterations in white matter integrity were found in widespread areas, most consistently in right anterior corona radiata, right forceps minor, bilateral internal capsule, and left cerebellum."

Thus, the ADHD pattern is one of elevated theta, reduced beta activity, and decreased FA values. The decreased FA values would imply lowered coherence and phase values. Treatment approaches have indicated the ability of NT to decrease theta and increase beta levels. Specific interventions on coherence and phase values in the ADHD subject has not been published to the best of the author's knowledge.

If the placebo effect of alpha magnitude increase was occurring during the NT intervention, one would expect an increase in theta and beta1 magnitudes and thus no significant change in the theta/beta ratios. Yet the research (Gevensleben, Holl, Albrecht, Schlamp, et al., 2009; Monastra et al., 2002)

report decreases in theta magnitudes and changes in theta/beta ratios which employ magnitude values.

Drug abuse

Peniston and Kulkosky (1989) studied alcoholics receiving BWT and showed a gradual increase in alpha and theta brain "rhythms" across the 15 experimental sessions. It is assumed that *rhythms* means *amplitudes*. The reason for the intervention was the previous research which hypothesized a decreased alpha level in alcoholics (Gabrielli et al., 1982). Saletu, Anderer, Saletu-Zyhlarz, Arnold, and Pascual-Marqui (2002) documented with qEEG and LORETA mapping studies of detoxified alcohol-dependent patients, as compared with normal controls, higher values of absolute and relative beta power, and lower values in alpha and delta/theta power for the alcohol patients.

Sokhadze, Cannon, and Trudeau (2008) reviewed the research on substance use disorders and biofeedback and concluded that "alpha theta training—either alone for alcoholism or in combination with beta training for stimulant and mixed substance abuse and combined with residential treatment programs, is probably efficacious…. Based on the guidelines jointly established by the Association for Applied Psychophysiology and Biofeedback (AAPB) and the International Society for Neurofeedback and Research (ISNR)" (p. 1). The NT intervention used most frequently was the rewarding of alpha (8–13 Hz) and theta (4–8 Hz) in eyes-closed condition. The Scott, Kaiser, Othmer, and Sideroff (2005) modification involved initially rewarding C3-FPZ and C4-PZ SMR (12–15 Hz) and beta (15–18 Hz) while inhibiting theta (2–7 Hz) and high beta (22–30 Hz), and then followed up with the Peniston protocol (reward alpha and theta). Abstinence was obtained for 77% of the EXP subjects and 44% for the controls at 1 year after intervention. The controls were involved in the Minnesota Model 12-step-oriented program as well as additional time in treatment which equaled the EXP time in treatment.

There remains the possibility of a placebo effect with endorphins and dopamine affecting the alpha and beta frequencies. However, the endorphins and dopamine would be increasing alpha and beta. The authors did not report the changes in the frequencies. However, the results were better than most rehabilitation programs and the 1-year time reassessment is a long time to expect a placebo effect to last.

Depression

There are numerous studies that report decreases in depression on the Minnesota Multiphasic Personality Inventory (MMPI) and other measures following NT, typically involving alpha/theta protocols (Cheon, Koo, & Choi, 2016; Grin-Yatsenko et al., 2018; Hammond, 2005; Raymond, Varney, Parkinson, & Gruzelier, 2005; Wang et al., 2016). Scott et al. (2005) reported improvements on the MMPI which included the experimental group's changes and exhibited a significant improvement compared with the changes in the control subjects on the Hypochondriasis, Depression, Conversion Hysteria, Schizophrenia, and Social Introversion scales.

As with the other clinical conditions, the possible placebo effect of increased endorphins or dopamine related to increases in alpha and beta remains a possibility. Contrary to that possibility, Sokhadze and Daniels (2016) reported on changes in self-perception of positive emotional state following 12 sessions of NT involving increasing the prefrontal relative power of 35–45 Hz EEG band at Fpz (middle of forehead). The NT training resulted in a significant linear increase of the relative power of the 35–45 Hz gamma measure and increase in self-report of happiness scores. Follow-up at 3.9 months showed maintenance of gains in happiness measure as well as MicroCog and IVA+Plus neurocognitive tests. The foundation for the research was raised in three previous publications (Cowan, & Albers, 2009; Cowan & Sokhadze, 2010, 2011). In addition, there is no research that the author is aware of which ties increases in the gamma frequency to a placebo effect.

Anxiety

Walker (2009) studied 19 PTSD patients with alpha/theta NT and obtained significant reductions in anxiety, while the control group (N = 4) did not show any reductions in anxiety. A decline in alpha activity has been reported in anxiety disorders using the qEEG (Buchsbaum et al., 1985; Heller, Nitschke, Etienne, & Miller, 1997).

The qEEG abnormalities in 100 anxious patients were reported by Gurnee (2003). He described six qEEG subtypes—high beta, high alpha, low alpha, cingulate dysfunction, high mean frequency beta, and high mean frequency alpha. Interventions directed towards these problems were generally effective in reducing anxiety. The placebo effect (alpha increase) could be employed to account for some of these improvements. However, the explanation of a sole alpha placebo effect across these diverse EEG

conditions faces the same problem previously discussed of alpha increases related to general magnitude increases across the different frequencies.

Cognitive – Memory

Thornton and Carmody (2013) reported on significant improvements in cognitive function in a group of 79 participants, including normal individuals, traumatic brain-injured, and specific learning disabilities (children, adult). Table 1 presents the results of the 2013 article as well as two previous articles (Thornton & Carmody, 2005, 2008).

Table 1
Treatment Effects of NT.

	Auditory Memory SD Effect	Auditory % Effect	Reading Memory SD Effect	Reading % Effect
Normal (*n* = 12)	1.66	59%	1.29	101%
TBI (*N* = 36)	2.3 (*N* = 36)		1.85 (*N* = 13)	143%
Adult SLD (*N* = 17)	1.42	86%	1.71	219%
Child SDL (*n* = 14)	1.28	74%	1.38	225%
Total (average; *N* = 79)	**1.67**	**73%**	**1.56**	**172%**

The following definitions of the qEEG variables are as follows:

- **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
- **M:** Absolute Magnitude*:* the average absolute magnitude (as defined in microvolts) of a band over the entire epoch (one second)
- **PA:** Peak Amplitude: the peak amplitude of a band during an epoch (defined in microvolts)
- **PF:** Peak Frequency: the peak frequency of a band during an epoch (defined in frequency)

Connectivity Measures

- **Spectral Correlation Coefficient (SCC)**: spectral morphology comparison correlation between two channels using the formula $(\sum|X||Y|)^2 / (\sum|X|^2 \sum|Y|^2)$ expressed in percent, where X and Y represent the Fourier series of the two channels and $\sum$ represents the summation within a band's frequency range.
- **Phase:** peak amplitude phase difference between two channels using the formula $100(1-|\theta_1-\theta_2|/\pi)$. A value of 100 percent represents zero degrees out of phase and a value of zero percent represents 180 degrees out of phase.

The bandwidths were grouped according to the following divisions: delta (0–4 Hz), theta (4–8 Hz), alpha (8–13 Hz), beta1 (13–32 Hz), beta2 (32–64 Hz).

An analysis of the changes in the relative power values of the different frequencies showed that the relative power of beta2 (32–64 Hz) was the only frequency that showed significant improvements when analyzed using confidence intervals. The relative power of beta2 was a main focus of the interventions. Thus, a placebo's effect on the relative power value of beta2 seems unlikely, given the previous discussion, as the placebo response is focused on the alpha and lower beta frequencies.

The correlation between % improvement in relative power of beta2 was most evident in the TBI group (+.46) and adult specific learning disability (+.77) in the auditory memory condition.

The conclusion for the coherence values was that "the average raw value of the Spectral Correlation Coefficient (SCC) change for alpha was 6.1 points (2.09 *SD*), for SCC beta1 (13–32 Hz) 6.53 points (1.81 *SD*), and for beta2 (32–64 Hz) 7.5 points (1.77 *SD*)."

Thus, the NT interventions were able to obtain significant increases in the relative power of beta2 and coherence values from alpha to beta2, across all subjects.

As previously noted, the placebo's effect is in the alpha or beta frequency and not the gamma frequency. In addition, alpha values are generally negatively related to the gamma frequency values (Thornton, 2016). Thus, it appears implausible that the placebo effect could account for these changes as there is no evidence to support the effect of placebos on relative power of beta2 (32–64 Hz) or any changes in the coherence and phase values of the beta1 of beta2 frequencies, which are critical to successful cognitive functioning (Thornton, 2016).

Discussion / Conclusions

Thibault and Raz (2017) make the argument that "placebo effects dominate EEG-nf outcomes. Whereas most neurofeedback experts acquiesce to this insight" (p. 684). The support for this statement resides in following quotes from experts:

> It would be naïve to believe that neurofeedback offers an adequate and sufficient treatment for any disorder (Joel Lubar, personal communication, 2016).…"It would be foolish to conclude that a foundation of knowledge has been realized enabling textbooks to be written [on EEG- nf]" (Gruzelier, 2014, p. 178).…Niels Birbaumer proposed that the cumulative evidence in favor of EEG-nf is preliminary and we stand to benefit from more controlled evidence to confirm that genuine feedback is a necessary component to achieve positive treatment outcomes (personal communication, 2016). (p. 688)

These direct quotes do not mention placebo effects, so it is difficult to discern how the authors came to that conclusion.

Thibault and Raz (2017) further state that "in light of the comparable benefits of veritable-versus-sham feedback, conflicts of interest, and a weak theoretical underpinning, advocating for EEG-nf poses a conundrum…Sparse evidence supports the idea that humans can reliably modulate EEG-nf signals" (p. 684). This conclusion is in stark contrast to the Thornton and Carmody (2013) and Thornton (2006) research study of cognitive changes, which consistently documented the ability of the approach to change the EEG signals along a number of measures and across different subjects.

A "weak theoretical underpinning" is a grossly inaccurate way to characterize operant conditioning, a concept Skinner proposed in 1938 (Skinner, 1938). The research cited in this article does show the effectiveness of NT over sham treatment, thus providing research support for its effectiveness which effectively addresses criticisms. In addition, the NT as a placebo argument has considerable problems as:

1) It claims "ubiquitous placebo influences" of NT (Thibault & Raz, 2017). This statement is not consistent with research on placebo effects, which show only 7% overall effectiveness across different clinical conditions, only 32% of patients respond to placebos, a genetic predisposition, and a lower percentage of patient responsiveness than an effective drug (for pain).

2) The biochemical effect of the endorphins and dopamine levels of a placebo are not sufficient to explain the short-term and long-term effects, given their half-lives. However, this criticism requires qualification as some patients show the placebo effect long after the half-lives of the endorphins and dopamine have passed. A possible answer is that the patient is continuing to produce these neurotransmitters by continuing to affect the alpha frequency. If this is the case, then we are still left with the problem of the cause. Is the continuing alpha activity the result of a placebo effect or effective operant conditioning? Since both the biofeedback typical program addresses alpha and the biochemical effect appears to be in the alpha frequency, it appears that the data presently available is unable to directly address that question.

3) The relationship of the biochemical effects on the alpha and beta frequencies appear to possibly explain the NT results. However, the gamma responsivity is inconsistent with the biochemical hypothesis as there is no evidence of a biochemical effect on this frequency.

4) Occam's razor states that among competing hypotheses, the one with the fewest assumptions should be selected. NT as a placebo has considerable problems in its explanation of the research findings. It is simpler to assume and more consistent with the literature that operant conditioning is the effective operant rather than some inconsistent and intermittent placebo effect.

5) This article has presented empirical evidence that humans can have an effect on the EEG signal. The relationship between the qEEG variables and cognitive performance is a complex one. It is difficult to assert that the placebo's effect is on all/most of the variables that relate to performance. If we reward a qEEG variable that has an empirical relationship to performance (memory) and the variable and memory performance improve, it is logical to conclude that the

intervention is not a placebo. To assert that a placebo knows what qEEG variable relates to performance and increases the values of those specific variables (among the 2,000+ available) is a difficult position to defend.

In conclusion, the effect of a placebo or subject's expectation can be linked to biochemical and electrophysiological activity. However, the relationship is a complex one and the ability of a placebo to explain all the effects is not credible, given the evidence reviewed. The alternate explanation that operant conditioning is causing the changes in the EEG signals is a more plausible and simpler one.

References

Amanzio, M., & Benedetti, F. (1999). Neuropharmacological dissection of placebo analgesia: Expectation-activated opioid systems versus conditioning-activated specific subsystems. The *Journal of Neuroscience, 19(1)*, 484–494. http://dx.doi.org/10.1523/JNEUROSCI.19-01-00484.1999

Arruda, J. E., Zhang, H., Amoss, R. T., Coburn, K. L., & Aue, W. R. (2009). Rhythmic oscillations in quantitative EEG measured during a continuous performance task. *Applied Psychophysiology and Biofeedback, 34*(1), 7–16. http://dx.doi.org/10.1007/s10484-008-9071-0

Auckland District Health Board (ADHB). (2018). *Newborn Services Drug Protocol. Dopamine hydrochloride*. Retrieved from http://www.adhb.govt.nz/newborn/DrugProtocols /DopaminePharmacology.htm

Banich, M. T., Milham, M. P., Atchley, R., Cohen, N. J., Webb, A., Wszalek, T., … Magin, R. (2000). fMRI studies of Stroop tasks reveal unique roles of anterior and posterior brain systems in attentional selection. *Journal of Cognitive Neuroscience*, *12*(6), 988–1000. http://dx.doi.org/10.1162 /08989290051137521

Barkley, R. A. (1998). *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (2nd ed.). New York, NY: Guilford Press.

Barry, R. J., Clarke, A. R., & Johnstone, S. J. (2003). A review of electrophysiology in attention-deficit/hyperactivity disorder: I. Qualitative and quantitative electroencephalography. *Clinical Neurophysiology*, *114*(2), 171–183. http://dx.doi.org/10.1016 /S1388-2457(02)00362-0

Beecher, H. K. (1955). The powerful placebo. *Journal of the American Medical Association*, *159*(17), 1602–1606. http://dx.doi.org/10.1001/jama.1955.02960340022006

Benedetti, F., Colloca, L., Torre, E., Lanotte, M., Melcarne, A., Pesare, M., … Lopiano, L. (2004). Placebo-responsive Parkinson patients show decreased activity in single neurons of subthalamic nucleus. *Nature Neuroscience*, *7*(6), 587–588. http://dx.doi.org/10.1038/nn1250

Bresnahan, S. M., Barry, R. J., Clarke, A. R., & Johnstone, S. J. (2006). Quantitative EEG analysis in dexamphetamine-responsive adults with attention-deficit/hyperactivity disorder. *Psychiatry Research*, *141*(2), 151–159. http://dx.doi.org /10.1016 /j.psychres.2005.09.002

Buchsbaum, M. S., Hazlett, E., Sicotte, N., Stein, M., Wu, J., & Zetin, M. (1985). Topographic EEG changes with benzodiazepine administration in generalized anxiety disorder. *Biological Psychiatry*, *20(8)*, 832–842. http://dx.doi.org/10.1016/0006-3223(85)90208-2

Bush, G., Frazier, J. A., Rauch, S. L., Seidman, L. J., Whalen, P. J., Jenike, M. A., … Biederman, J. (1999). Anterior cingulate cortex dysfunction in attention-deficit/hyperactivity disorder revealed by fMRI and the Counting Stroop, *Biological Psychiatry*, *45*(12), 1542–1552.

Chabot, R. J., & Serfontein, G., (1996). Quantitative electroencephalographic profiles of children with attention deficit disorder. *Biological Psychiatry, 40(10)*, 951–963. http://dx.doi.org/10.1016/0006-3223(95)00576-5

Cheon, E.-J., Koo, B.-H., & Choi, J.-H. (2016). The efficacy of neurofeedback in patients with major depressive disorder: An open labeled prospective study. *Applied Psychophysiology and Biofeedback*, *41*(1), 103–110. http://dx.doi.org/10.1007 /s10484-015-9315-8

Cherry, K. (2018, November 14). How the Placebo Effect Works in Psychology. Retrieved from https://www.verywellmind.com /what-is-the-placebo-effect-2795466

Clarke, A. R., Barry, R. J., McCarthy, R., Selikowitz, M., & Johnstone, S. J. (2007). Effects of stimulant medications on the EEG of girls with attention-deficit/hyperactivity disorder. *Clinical Neurophysiology, 118*(12), 2700–2708. http://dx.doi.org/10.1016/j.clinph.2007.08.020

Cowan, J. D., & Albers, S. A. (2009). *Manual for the peak brain happiness trainer: Exited happiness and Neureka!* Protocols. Goshen, KY: Peak Achievement Training.

Cowan, J. D., & Sokhadze, E. (2011). Prefrontal gamma neurofeedback improves emotional state and cognitive function. *Applied Psychophysiology and Biofeedback*, *36*, S220.

Cowan, J., & Sokhadze, E. (2010, September 29–October 3). *Happiness specifically increases a clarified 40 Hz EEG rhythm used for neurofeedback*. Presented at the 18th International Society Neurofeedback Research Annual Conference, Denver, CO.

Crabbe, J. B., & Dishman, R. K. (2004). Brain electrocortical activity during and after exercise: A quantitative synthesis. *Psychophysiology, 41*(4), 563–574. http://dx.doi.org/10.1111 /j.1469-8986.2004.00176.x

Dalayeun, J. F., Norès, J. M., Bergal, S. (1993). Physiology of beta-endorphins. A close-up view and a review of the literature. *Biomedicine & Pharmacotherapy, 47*(8), 311–320. http://dx.doi.org/10.1016/0753-3322(93)90080-5

de la Fuente-Fernández, R., Ruth, T. J., Sossi, V., Schulzer, M., Calne, D. B., & Stoessl, A. J. (2001). Expectation and dopamine release: Mechanism of the placebo effect in Parkinson's disease. *Science*, *293*(5532), 1164–1166. http://dx.doi.org/10.1126/science.1060937

Eippert, F., Bingel, U., Schoell, E. D., Yacubian, J., Klinger, R., Lorenz, J., & Büchel, C. (2009). Activation of the opioidergic descending pain control system underlies placebo analgesia. *Neuron*, *63*(4), 533–543. http://dx.doi.org/10.1016 /j.neuron.2009.07.014

Ernst, M., Zametkin, A. J., Matochik, J. A., Liebenauer, L. Fitzgerald, G. A., & Cohen, R. M. (1994). Effects of intravenous dextroamphetamine on brain metabolism in adults with attention-deficit/hyperactivity disorder (ADHD): Preliminary findings. *Psychopharmacology Bulletin*, *30*(20), 219–225.

Feinberg, C. (2013, January–February). The placebo phenomenon. An ingenious researcher finds the real ingredients of "fake" medicine. *Harvard Magazine*, *115*(3), 36–39.

Foley, K. M., Kourides, I. A., Inturrisi, C. E., Kaiko, R. F., Zaroulis, C. G., Posner, J. B., … Li, C. H. (1979). β-Endorphin: Analgesic and hormonal effects in humans. *Proceedings of the National Academy of Sciences of the United States of America*, *76*(10), 5377–5381.

Furmark, T., Appel, L., Henningsson, S., Åhs, F., Faria, V., Linnman, C., … Fredrikson, M. (2008). A link between serotonin-related gene polymorphisms, amygdala activity, and placebo-induced relief from social anxiety. *The Journal of Neuroscience*, *28*(49), 13066–13074. http://dx.doi.org /10.1523/JNEUROSCI.2534-08.2008

Gabrielli, W. F., Mednick, S. A., Volavka, J., Pollock, V. E., Schulsinger, F. & Itil, T. M. (1982). Electroencephalograms in children of alcoholic fathers. *Psychophysiology*, *19*(4), 404-407.

Gani, C., Birbaumer, N., & Strehl, U. (2008). Long term effects after feedback of slow cortical potentials and of theta-beta-amplitudes in children with attention deficit/hyperactivity disorder (ADHD). *International Journal of Bioelectromagnetics*, *10*(4), 209–232.

Gevensleben, H., Holl, B., Albrecht, B., Schlamp, D., Kratz, O., Studer, P., … Heinrich, H. (2009). Distinct EEG effects related to neurofeedback training in children with ADHD: A randomized controlled trial. *International Journal of Psychophysiology, 74*(2), 149–157. http://dx.doi.org/10.1016 /j.ijpsycho.2009.08.005

Gevensleben, H., Holl, B., Albrecht, B., Schlamp, D., Kratz, O., Studer, P., … Heinrich, H. (2010). Neurofeedback training in children with ADHD: 6-month follow-up of a randomised controlled trial. *European Child & Adolescent Psychiatry, 19*(9), 715–724. http://dx.doi.org/10.1007 /s00787-010-0109-5

Gevensleben, H., Holl, B., Albrecht, B., Vogel, C., Schlamp, D., Kratz, O., … Heinrich, H. (2009). Is neurofeedback an efficacious treatment for ADHD? A randomized controlled clinical trial. *Journal of Child Psychology and Psychiatry*, *50(7)*, 780–789. http://dx.doi.org/10.1111/j.1469-7610.2008.02033.x

Ghaziri, J., Tucholka, A., Larue, V., Blanchette-Sylvestre, M., Reyburn, G., Gilbert, G., … Beauregard, M. (2013). Neurofeedback training induces changes in white and gray matter. *Clinical EEG and Neuroscience*, *44*(4), 265–272. http://dx.doi.org/10.1177/1550059413476031

Gracely, R. H., Dubner, R., Wolskee, P. J., & Deeter, W. R. (1983). Placebo and naloxone can alter post-surgical pain by separate mechanisms. *Nature*, *306*(5940), 264–265. http://dx.doi.org/10.1038/306264a0

Grevert, P., Albert, L. H., & Goldstein, A. (1983). Partial antagonism of placebo analgesia by naloxone. *Pain, 16(1)*, 129–143. http://dx.doi.org/10.1016/0304-3959(83)90203-8

Grin-Yatsenko, V. A., Othmer, S., Ponomarev, V. A., Evdokimov, S. A., Konoplev, Y. Y., & Kropotov, J. D. (2018) Infra-low frequency neurofeedback in depression: Three case studies. *NeuroRegulation*, *5*(1), 30–42. http://dx.doi.org /10.15540/nr.5.1.30

Gruzelier, J. H. (2014). EEG-neurofeedback for optimising performance. III: A review of methodological and theoretical considerations. *Neuroscience & Biobehavioral Reviews*, *44*, 159 –182. http://dx.doi.org/10.1016/j.neubiorev.2014.03.015

Gunkelman, J. (2009, December 23). Drug exposure and EEG/qEEG findings. Retrieved from http://brainm.com /software/pubs/Gunkelman Drug exposure and EEG.pdf

Gurnee, R. (2003). *QEEG subtypes of anxiety*. Presented at the International Society for Neurofeedback and Research 11th Annual Conference, Houston, TX.

Hall, K. T., Lembo. A. J., Kirsch. I., Ziogas, D. C., Douaiher, J., Jensen, K. B., … Kaptchuk, T. J. (2012). Catechol-O-methyltransferase val158met polymorphism predicts placebo effect in irritable bowel syndrome. *PLoS ONE*, *7*(10), e48135. http://dx.doi.org/10.1371/journal.pone.0048135

Hall, K. T., Loscalzo, J., & Kaptchuk, T. J. (2015). Genetics and the placebo effect: The placebome. *Trends in Molecular Medicine, 21*(5), 285–294. http://dx.doi.org/10.1016 /j.molmed.2015.02.009

Hamilton, L. S., Levitt, J. G., O'Neil, J., Alger, J. R., Luders, E., Phillips, O. R., ... Narr, K. L. (2008). Reduced white matter integrity in attention-deficit hyperactivity disorder. *Neuroreport*, *19*(17), 1705–1708. http://dx.doi.org/10.1097 /WNR.0b013e3283174415

Hammond, D. C. (2005). Neurofeedback with anxiety and affective disorders. *Child and Adolescent Psychiatric Clinics of North America*, *14*(1), 105–123. http://dx.doi.org/10.1016 /j.chc.2004.07.008

Heller, W., Nitschke, J. B., Etienne, M. A., & Miller, G. A. (1997). Patterns of regional brain activity differentiate types of anxiety. *Journal of Abnormal Psychology*, *106*(3), 376–385. http://dx.doi.org/10.1037/0021-843X.106.3.376

Hróbjartsson, A., & Gøtzsche, P. A. (2010). Placebo interventions for all clinical conditions. *Cochrane Database of Systematic Reviews*, *1.* http://dx.doi.org/10.1002 /14651858.CD003974.pub3

Kaptchuk, T. J., Friedlander, E., Kelley, J. M., Sanchez, M. N., Kokkotou, E., Singer, J. P., … Lembo, A. J. (2010). Placebos without deception: A randomized controlled trial in irritable bowel syndrome. *PLoS ONE*, 5(12), e15591. http://dx.doi.org /10.1371/journal.pone.0015591

Kong, J., Gollub, R. L., Rosman, I. S., Webb, J. M., Vangel, M. G., Kirsch, I., & Kaptchuk, T. J. (2006). Brain activity associated with expectancy-enhanced placebo analgesia as measured by functional magnetic resonance imaging. *The Journal of Neuroscience*, *26*(2), 381–388. http://dx.doi.org/10.1523 /JNEUROSCI.3556-05.2006

Konrad, A., Dielentheis, T. F., Masri, D. E., Dellani, P. R., Stoeter, P., Vucurevic, G., & Winterer, G. (2012). White matter abnormalities and their impact on attentional performance in adult attention-deficit/hyperactivity disorder. *European Archives of Psychiatry and Clinical Neuroscience, 262*(4), 351–360. http://dx.doi.org/10.1007/s00406-011-0251-1

Leins, U., Goth, G., Hinterberger, T., Klinger, C., Rumpf, N., & Strehl, U. (2007). Neurofeedback for children with ADHD: A comparison of SCP and theta-beta protocols. *Applied Psychophysiology and Biofeedback*, *32*(2), 73–88. http://dx.doi.org/10.1007/s10484-007-9031-0

Leuchter, A. F., Cook, I. A., Witte, E. A., Morgan, M., & Abrams, M. (2002). Changes in brain function of depressed subjects during treatment with placebo. *The American Journal of Psychiatry*, *159*(1), 122–129. http://dx.doi.org/10.1176 /appi.ajp.159.1.122

Lévesque, J., Beauregard, M., & Mensour, B. (2006). Effect of neurofeedback training on the neural substrates of selective attention in children with attention-deficit/hyperactivity disorder: A functional magnetic resonance imaging study. *Neuroscience Letters*, *394*(3), 216–221. http://dx.doi.org /10.1016/j.neulet.2005.10.100

Levine, J. D., Gordon, N. C., Jones, R. T., & Fields, H. L. (1978). The narcotic antagonist naloxone enhances clinical pain. *Nature*, *272*, 826–827.

Lipman, J. J., Miller, B. E., Mays, K. S., Miller, M. N., North, W. C., & Byrne, W. L. (1990). Peak B endorphin concentration in cerebrospinal fluid: reduced in chronic pain patients and increased during the placebo response. *Psychopharmacology*, *102*(1), 112–116. http://dx.doi.org /10.1007/BF02245754

Lubar, J. F., Swartwood, M. O., Swartwood, J. N., & Timmermann, D. L. (1996). Quantitative EEG and auditory event-related potentials in the evaluation of attention-deficit/hyperactivity disorder: Effects of methylphenidate and implications for neurofeedback training. [Monograph: Assessment of Attention-Deficit/Hyperactivity Disorders]. *Journal of Psychoeducational Assessment*, 143–204.

Matochik, J. A., Liebenauer, L. L., King, A. C., Szymanski, H. V., Cohen, R. M., & Zametkin, A. J. (1994). Cerebral glucose metabolism in adults with attention deficit hyperactivity disorder after chronic stimulant treatment. *American Journal of Psychiatry*, *151*(5), 658–664. http://dx.doi.org/10.1176 /ajp.151.5.658

Matsuura, M., Okubo, Y., Toru, M., Kojima, T., He, Y., Hou, Y., … Lee, C. K. (1993). A cross-national EEG study of children with emotional and behavioral problems: A WHO collaborative study in the Western Pacific Region. *Biological Psychiatry, 34*(1–2), 59–65. http://dx.doi.org/10.1016/0006-3223(93)90257-E

Mimasa, F., Hayashi, T., Shibata, M., Yoshitake, Y., Nishijima, Y., & Moritani, T. (1996). Movement of electroroencephalogram and plasm β-endorphin in the aerobic exercise. *Japanese Journal of Physical Fitness and Sports Medicine, 45*(5), 519–526. http://dx.doi.org/10.7600/jspfsm1949.45.519

Molteni, E., Bianchi, A. M., Butti, M., Reni, G., & Zucca, C., (2007). Analysis of the dynamical behaviour of the EEG rhythms during a test of sustained attention. *Conference Proceedings: Annual International Conference of the IEEE Engineering in Medicine and Biology Society*, 1298–1301. http://dx.doi.org/10.1109/IEMBS.2007.4352535

Monastra, V. J., Monastra, D. M., & George, S. (2002). The effects of stimulant therapy, EEG biofeedback, and parenting style on the primary symptoms of attention-deficit/hyperactivity disorder. *Applied Psychophysiology and Biofeedback*, *27*(4), 231–249. http://dx.doi.org/10.1023 /A:1021018700609

Nestler, E. J., Hyman, S. E., & Malenka, R. C., (2009). *Molecular Neuropharmacology: A Foundation for Clinical Neuroscience* (2nd ed., p. 321). New York, NY: McGraw-Hill Medical.

Niogi, S., Mukherjee, P., Ghajar, J., & McCandliss, B. D. (2010). Individual differences in distinct components of attention are linked to anatomical variations in distinct white matter tracts. *Frontiers in Neuroanatomy*, *4*, 2. http://dx.doi.org/10.3389 /neuro.05.002.2010

Pavuluri, M. N., Yang, S., Kamineni, K., Passarotti, A. M., Srinivasan, G., Harral, E. M., … Zhou, X. J. (2009). Diffusion tensor imaging study of white matter fiber tracts in pediatric bipolar disorder and attention-deficit/hyperactivity disorder. *Biological Psychiatry*, *65*(7), 586–593. http://dx.doi.org /10.1016/j.biopsych.2008.10.015

Peniston, E. G., & Kulkosky, P. J. (1989). Alpha-theta brainwave training and beta-endorphin levels in alcoholics. *Alcoholism, Clinical & Experimental Research*, *13*(2), 271–279. http://dx.doi.org/10.1111/j.1530-0277.1989.tb00325.x

Petrovic, P., Dietrich, T., Fransson, P., Andersson, J., Carlsson, K., & Ingvar, M. (2005). Placebo in emotional processing—induced expectations of anxiety relief activate a generalized modulatory network. *Neuron*, *46*(6), 957–969. http://dx.doi.org/10.1016/j.neuron.2005.05.023

Petrovic, P., Kalso, E., Petersson, K. M., & Ingvar, M. (2002). Placebo and opioid analgesia—imaging a shared neuronal network. *Science, 295*(5560), 1737–1740. http://dx.doi.org /10.1126/science.1067176

Pfefferbaum, A., Berger, P. A., Elliot, G. R., Tinklenberg, J. R., Kopell, B. S., Barchas, J. D., & Li, C. H. (1979). Human EEG response to beta-endorphin. *Psychiatry Research, 1*(1), 83–88. http://dx.doi.org/10.1016/0165-1781(79)90031-3

Qiu, M.-G., Ye, Z., Li, Q.-Y., Liu, G.-J., Xie, B., & Wang, J. (2011). Changes of brain structure and function in ADHD children. *Brain Topography*, *24*(3-4), 243–252. http://dx.doi.org /10.1007/s10548-010-0168-4

Raymond, J., Varney, C., Parkinson, L. A., & Gruzelier, J. H. (2005). The effects of alpha/theta neurofeedback on personality and mood. *Cognitive Brain Research*, *23*(2–3), 287–292. http://dx.doi.org/10.1016/j.cogbrainres.2004.10.023

Rossi, E. L. (1986). *The psychobiology of mind-body healing: New concepts of therapeutic hypnosis* (Rev. ed.). New York, NY: W. W. Norton & Company, Inc.

Saletu, B., Anderer, P., Saletu-Zyhlarz, G. M., Arnold, O., & Pascual-Marqui, R. D. (2002). Classification and evaluation of the pharmacodynamics of psychotropic drugs by single-lead pharmaco-EEG, EEG mapping and tomography (LORETA). *Methods and Findings in Experimental and Clinical Pharmacology*, *24*(Suppl. C), 97–120.

Scott, D. J., Stohler, C. S., Egnatuk, C. M., Wang, H., Koeppe, R. A., & Zubieta, J.-K. (2008). Placebo and nocebo effects are defined by opposite opioid and dopaminergic responses. *Archives of General Psychiatry*, *65*(2), 220–231. http://dx.doi.org/10.1001 /archgenpsychiatry.2007.34

Scott, W. C., Kaiser, D., Othmer, S., & Sideroff, S. I. (2005). Effects of an EEG biofeedback protocol on a mixed substance abusing population. *The American Journal of Drug and Alcohol Abuse, 31*(3), 455–469. http://dx.doi.org/10.1081 /ADA-200056807

Sherlin, L., Arns, M. Lubar, J., & Sokhadze, E. (2010). A position paper on neurofeedback for treatment of ADHD. *Journal of Neurotherapy*, *14*(2), 66–78. http://dx.doi.org/10.1080 /10874201003773880

Skinner, B. F. (1938). The behavior of organisms: An experimental analysis. New York, NY: Appleton-Century-Crofts, Inc.

Snyder, S. M., & Hall, J. R. (2006). A meta-analysis of quantitative EEG power associated with attention-deficit/hyperactivity disorder. *Journal of Clinical Neurophysiology*, *23*(5), 441–456. http://dx.doi.org/10.1097/01.wnp.0000221363.12503.78

Sokhadze, E. M., & Daniels, R. (2016). Effects of prefrontal 40 Hz-centered EEG band neurofeedback on emotional state and cognitive functions in adolescents. *Adolescent Psychiatry*, *6(2)*, 116–129. http://dx.doi.org/10.2174 /2210676606666161025115616

Sokhadze, T. M., Cannon, R. L., & Trudeau, D. L. (2008). EEG biofeedback as a treatment for substance use disorders: Review, rating of efficacy, and recommendations for further research. *Applied Psychophysiology and Biofeedback, 33(1)*, 1–28. http://dx.doi.org/10.1007/s10484-007-9047-5

Staddon, J. E. R., & Cerutti, D. T. (2003). Operant conditioning. *Annual Review of Psychology, 54*, 115–144. http://dx.doi.org/10.1146/annurev.psych.54.101601.145124

Thibault, R. T., & Raz, A. (2017). The psychology of neurofeedback: Clinical intervention even if applied placebo. *American Psychologist*, *72*(7), 679–688. http://dx.doi.org /10.1037/amp0000118

Thornton, K. E. (2006). *No child left behind goals (and more) are attainable with neurocognitive interventions* (Vol. 1). North Charleston, SC: Booksurge Press.

Thornton, K. (2016). *How the cognitive brain works: The quantitative EEG and cognition*. Clover, SC: Kirtley Thornton. Printed by Create Space.

Thornton, K. E., & Carmody, D. P. (2005). EEG biofeedback for reading disabilities and traumatic brain injuries. *Child and Adolescent Psychiatric Clinics of North America*, *14*(1), 137–162.

Thornton, K. E., & Carmody, D. P. (2008). Efficacy of traumatic brain injury rehabilitation: interventions of qEEG-guided biofeedback, computers, strategies, and medications. *Applied Psychophysiology and Biofeedback, 33*(2), 101–124. http://dx.doi.org/10.1007/s10484-008-9056-z

Thornton, K. E., & Carmody, D. P. (2013). The relation between memory improvement and qEEG changes in three clinical groups as a result of EEG biofeedback treatment. *Journal of Neurotherapy*, *17*(2), 116–132. http://dx.doi.org/10.1080 /10874208.2013.785183

van Ewijk, H., Heslenfeld, D. J., Zwiers, M. P., Buitelaar, J. K., & Oosterlaan, J. (2012). Diffusion tensor imaging in attention deficit/hyperactivity disorder: A systematic review and meta-analysis. *Neuroscience & Biobehavioral Reviews*, *36*(4), 1093–1106. http://dx.doi.org/10.1016/j.neubiorev.2012.01.003

Volavka, J., James, B., Reker, D., & Cho, D. (1979). Electroencephalographic and other effects of naloxone in normal men. *Life Sciences*, *25*(14), 1267–1272. http://dx.doi.org/10.1016/0024-3205(79)90471-5

Wager, T. D., Rilling, J. K., Smith, E. E., Sokolik, A., Casey, K. L., Davidson, R. J., … Cohen, J. D. (2004). Placebo-induced changes in fMRI in the anticipation and experience of pain. *Science*, *303*(5661), 1162–1167. http://dx.doi.org/10.1126 /science.1093065

Wager, T. D., Scott, D. J., & Zubieta, J. K. (2007). Placebo effects on human mu-opioid activity during pain. *Proceedings of the National Academy of Sciences of the United States of America,*

104(26), 11056–11061. http://dx.doi.org/10.1073/pnas.0702413104

Walker, J. E. (2009). Anxiety associated with post traumatic stress disorder—The role of quantitative electroencephalograph in diagnosis and in guiding neurofeedback training to remediate the anxiety. *Biofeedback*, *37*(2), 67–70. http://dx.doi.org/10.5298/1081-5937-37.2.67

Wang, S.-Y., Lin, I.-M., Peper, E., Chen, Y.-T., Yeh, Y.-C., … Chu, C. (2016). The efficacy of neurofeedback among patients with major depressive disorder: Preliminary study. *NeuroRegulation*, *3*(3), 127–134. http://dx.doi.org/10.15540/nr.3.3.127

Wechsler, M. E., Kelley, J. M., & Kaptchuk, T. J. (2011). Placebos and other interventions in asthma. *The New England Journal of Medicine*, *365*(15), 1447.

World Research Foundation (WRF) Staff. (n.d.) The power of mind and the promise of placebo. Retrieved from http://www.wrf.org/alternative-therapies/power-of-mind-placebo.php

Zubieta, J.-K., Bueller, J. A., Jackson, L. R., Scott, D. J., Xu, Y., Koeppe, R. A., … Stohler, C. S. (2005). Placebo effects mediated by endogenous opioid activity on mu-opioid receptors. *The Journal of Neuroscience*, *25*(34), 7754–7762. http://dx.doi.org/10.1523/JNEUROSCI.0439-05.2005

Zubieta, J.-K., & Stohler, C. S. (2009). Neurobiological mechanisms of placebo responses. *Annals of the New York Academy of Sciences*, *1156*(1), 198–210. http://dx.doi.org/10.1111/j.1749-6632.2009.04424.x

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KEYNOTE PRESENTATIONS

Assessment and Outpatient Treatment of Addiction Using Neurofeedback and a Functional Medicine Approach

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The most common type of addiction treatment in the United States today is based on the Minnesota Model. This style of treatment relies on 12 Steps principles and was developed over 60 years ago. Despite a tremendous amount of research and new evidence-based practices, very few treatment centers incorporate innovations like neurofeedback.

In this session, we will look at and review several cases studies from the Atlanta Healing Center that illustrate important advances in treatment of the chronic brain disease of addiction. We will focus on the importance of making the proper diagnosis; medication-assisted recovery; evaluating co-occurring psychiatric, cognitive, and pain conditions; assessing the hormonal and nutritional status of patients; and providing treatment modalities like neurofeedback. Education of the family and patient is important and connecting with recovery support is essential for the patient to have the best possible outcome in the management of this potentially life-threatening disease.

The Effects and Mechanisms of Mindfulness Meditation, Cognitive Therapy, and Mindfulness-based Cognitive Therapy for Chronic Low Back Pain

Melissa Day
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Chronic low back pain (CLBP) is a pervasive, costly, and highly disabling condition. Research has shown that CLBP is inadequately managed solely by biomedical approaches alone. Thus, current guidelines put forth by the Centers for Disease Control and Prevention in the U.S. recommend nonpharmacological therapy as the first line approach to chronic pain management. In this context, there is evidence that Cognitive Therapy (CT) and mindfulness meditation (MM) programs are beneficial for a range of CLBP-related outcomes. Although not previously tested for CLBP management, evidence in other pain populations suggests that Mindfulness-based Cognitive Therapy (MBCT)—which seamlessly integrates CT and MM techniques—might also be particularly well suited for improving pain, mood, and function.

An expanding body of research is investigating the potential neuromodulatory function of these psychosocial pain treatments. Although scarce research has examined brain state-related changes in the context of CT and MBCT for pain, within MM, several studies in pain samples have used electroencephalogram (EEG) at pre- and posttreatment to test the possible role of brain activity changes in association with improved pain-related outcomes. Results found MM was associated with power increases primarily in the alpha band, and this increase in alpha was suggested to play a key role in the effects of MM on pain. It is not known, however, if this potential neuromodulatory pathway is unique to MM as delivered as an isolated technique, or if it might also play a role in other similarly efficacious treatments, such as CT and MBCT.

In this session I will present data from a recently completed randomized controlled trial comparing MM versus CT versus MBCT within a CLBP sample. Treatment consisted of eight weekly, 2-hour group-delivered sessions. EEG brain state data was obtained at pre- and posttreatment, as was self-reported pain-related outcome measures of pain interference, pain intensity, physical function, and depression. I will present (1) the treatment-related changes in the self-reported outcomes, (2) an

analysis of change in brain activity across the three treatments, and (3) how potential changes in brain state are associated with changes in the self-reported outcomes.

Is Addiction a Brain Disease? And Does It Matter?

Marc Lewis
University of Toronto, Toronto, Ontario, Canada

Over the past 20 to 30 years, medical authorities have come to define and explain addiction as a brain disease. However, the domination of the disease model skews the science of addiction, diverts attention from key social-psychological factors, and results in potentially harmful trends in policy and clinical practice. In this talk I review the distortions and omissions of the classic brain disease model and point to problems in the treatment philosophy derived from it. I then outline an alternative model of addiction based on principles of learning and development. This model views addiction as an entrenched habit for regulating emotional needs, learned through the repeated pursuit of highly motivating but short-lived rewards. Developmental-learning models of addiction help explain individual differences in vulnerability (and recovery) based on early emotional difficulties and current psychological and social resources.

INVITED PRESENTATIONS

The Central Brain Mechanisms of Pain and the Neuromodulation Techniques for Addressing It

Dirk DeRidder
Dunedin School of Medicine, University of Otago, Dunedin, New Zealand

Although chronic pain is one of the most important medical problems facing society, there has been limited progress in the development of novel therapies for this condition. The key to more successful pain treatment is to understand the mechanisms that generate and maintain chronic pain. Anatomically there exist at least two ascending pain input pathways and one descending pain inhibitory pathway. One input pathway encodes the painfulness, whereas the other pathway encodes the suffering or emotional pain associated with the painful stimulus. The pain inhibitory pathway probably encodes the percentage of the time the pain is dominantly present during the day. The anatomical pathways can be visualized using functional MRI meta-analyses, and LORETA EEG further shows that chronic pain is an imbalance between the ascending and descending pain inhibitory pathways. This is indeed confirmed both by activity, functional and effective connectivity EEG analyses.

Nonpharmacological treatment for chronic pain using spinal cord stimulation normalizes this imbalance, supporting the concept that pain is truly a balance disorder between pain input and pain suppression in the brain, and causally related to this imbalance. Pain thus is not merely the result of more pain input via the spinal cord or brainstem.

This imbalance mechanism, also known as thalamocortical dysrhythmia, might be universal in view of the pathophysiological analogy between pain, tinnitus, Parkinson's disease, and major depression. Furthermore, thalamocortical dysrhythmia and reward deficiency syndrome (obesity, addiction, ADHD, and personality disorders) may be two sides of the same coin as suggested by EEG source analyzed conjunction analyses between thalamocortical dysrhythmia and reward deficiency syndromes. As such, this new conceptualization of pain, Parkinson, tinnitus, depression, addiction, ADHD, OCD, and personality disorders as imbalances in the brain paves the way for neuromodulation techniques such as transcranial electrical stimulation and infraslow neurofeedback to normalize this imbalance.

Integrating Mindfulness with Bio and Neurofeedback

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Bio and neurofeedback are powerful treatment modalities shown to be effective at alleviating numerous psychophysiological conditions. Biofeedback provides a way to work with challenging conditions in cases for which other interventions have been unsuccessful, such as chronic pain, anxiety, headaches, and trauma. At the same time, bio/neurofeedback treatment itself can stall, leaving the client and the therapist feeling frustrated and unsure of how to proceed. These challenges include situations when the client is highly anxious about his or her physiological symptoms, feels pressure to "do things right," becomes easily overwhelmed with emotional stimuli, or is simply too distracted to attend to the computer screen for more than a few minutes at a time. Oftentimes, these challenges are due to the clients' unhelpful efforts to control the

fundamentally uncontrollable aspects of their internal experience.

Mindfulness-based approach to bio/neurofeedback can help people experience change through mindful, nonjudgmental awareness and acceptance, providing the therapist and the client a way to work with what gets in the way of biofeedback success. In this talk participants will learn how to apply mindfulness-based skills to their biofeedback practice in order to help their clients reap the benefits of biofeedback without getting stuck in unproductive attempts to control their internal experience.

Update on the Work Towards CPT Codes and Third-Party Reimbursement

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ISNR, in partnership with AAPB and BCIA, have taken significant strides in the past few years toward pushing for insurance reimbursement. This presentation will provide a macro-level progress report on the CPT coding workgroup trying to modernize our codes, petitions for inclusion as a recognized organization for AMA activities for CPT coding and Relative Value Unit (RVU) determinations, and national-level efforts for insurance reimbursement.

STUDENT AWARD PRESENTATIONS – PLENARY SESSIONS

Tuning the Traumatized Brain: LORETA *Z*-score Neurofeedback and Heart Rate Variability Biofeedback for Chronic PTSD

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Introduction. Neuroimaging studies have identified numerous abnormalities within the default mode (DMN), salience (SN), and central executive (CEN) neural networks of those suffering from PTSD (Lanius, Frewen, Tursich, Jetly, & McKinnon, 2015). A systematic review of the literature revealed ten studies (n = 213) that examined neurofeedback as a method for altering these neural patterns and alleviating PTSD symptoms (Foster & Thatcher, 2015; Gapen et al., 2016; Huang-Storms, Bodenhamer-Davis, Davis, & Dunn, 2006; Kluetsch et al., 2014; Paret et al., 2014; Peniston & Kulkosky, 1991; Peniston, Marrinan, Deming, & Kulkosky, 1993; Pop-Jordanova & Zorcec, 2004; Smith, 2008; van der Kolk et al., 2016). These studies demonstrated mostly medium to large improvements following a variety of NF training modalities. Low-resolution electromagnetic tomography analysis (LORETA) *z*-score neurofeedback (LZNF) is a newer modality that is believed to produce more targeted and efficient outcomes than traditional modalities; however, it has not been adequately examined for the treatment of PTSD. This study is the first to examine the effectiveness of this modality using a controlled, experimental design.

Method. The purpose of this research study was to examine the effects of LZNF training, as compared to heart rate variability biofeedback (HRVB) training, on PTSD symptoms, autonomic regulation, and brainwave activation patterns in adults with chronic PTSD. Twenty-four participants were alternately assigned to receive 15 sessions of either LZNF (n = 12) or HRVB (n = 12) training. HRVB was chosen as an active control condition due to the ability to closely match many conditions to the LZNF group (i.e., time with trainer, resting state, body-computer interface, similar audio/visual feedback, etc.) while providing an ethical alternative for this sensitive population. Psychophysiological measurements (i.e., 19-channel EEG and HRV) were recorded before, during, and after a single session of training as well as before and after 15 training sessions. Psychosocial questionnaires were completed during the pre- and postintervention assessments.

Results. The data for this study is still being analyzed and thus results are not yet available; however, visual examination of the data and symptom reports suggest the results will be positive. The full results will be analyzed and ready to present before this ISNR conference. Paired and independent samples t-tests and Cohen's d effect sizes are being utilized to examine both within- and between-group changes after 1 and 15 sessions. Pre–post changes will be analyzed for mean LORETA current source density (CSD) z-scores of three neural networks (i.e., DMN, SN, and CEN); HRV metrics (i.e., standard deviation of NN intervals, root mean square of the successive difference, low-frequency power); and total scores on the PTSD Checklist for DMS-V and Beck Anxiety Inventory. I have hypothesized that LZNF will produce greater changes in LORETA CSD z-scores and PTSD symptoms, while HRVB will produce greater changes in HRV metrics.

Conclusion. This study is expected to provide important, preliminary data regarding the effectiveness of both LZNF and HRVB training on

PTSD symptoms and HRV, as well as their differential effects on each of the neural networks suspected to underlie PTSD symptomology.

References

Foster, D. S., & Thatcher, R. W. (2015). Surface and LORETA neurofeedback in the treatment of post-traumatic stress disorder and mild traumatic brain injury. In R. W. Thatcher & D. S. Foster (Eds.), Z score neurofeedback: Clinical applications (pp. 59–92). San Diego, CA: Academic Press.

Gapen, M., van der Kolk, B. A., Hamlin, E., Hirshberg, L., Suvak, M., & Spinazzola, J. (2016). A pilot study of neurofeedback for chronic PTSD. *Applied Psychophysiology and Biofeedback, 41*(3), 251–261. http://dx.doi.org/10.1007 /s10484-015-9326-5

Huang-Storms, L., Bodenhamer-Davis, E., Davis, R., & Dunn, J. (2006). QEEG-guided neurofeedback for children with histories of abuse and neglect: Neurodevelopmental rationale and pilot study. *Journal of Neurotherapy, 10*(4), 3–16. http://dx.doi.org/10.1300/J184v10n04_02

Kluetsch, R. C., Ros, T., Théberge, J., Frewen, P. A., Calhoun, V. D., Schmahl, C., … Lanius, R. A. (2014). Plastic modulation of PTSD resting-state networks and subjective wellbeing by EEG neurofeedback. *Acta Psychiatrica Scandinavica, 130*(2), 123–136. http://dx.doi.org/10.1111/acps.12229

Lanius, R. A., Frewen, P. A., Tursich, M., Jetly, R., & McKinnon, M. C. (2015). Restoring large-scale brain networks in PTSD and related disorders: A proposal for neuroscientifically-informed treatment interventions. *European Journal of Psychotraumatology, 6*(1). http://dx.doi.org/10.3402 /ejpt.v6.27313

Paret, C., Kluetsch, R., Ruf, M., Demirakca, T., Hoesterey, S., Ende, G., & Schmahl, C. (2014). Down-regulation of amygdala activation with real-time fMRI neurofeedback in a healthy female sample. *Frontiers in Behavioral Neuroscience, 8*, 299. http://dx.doi.org/10.3389/fnbeh.2014.00299

Peniston, E. G., & Kulkosky, P. J. (1991). Alpha-theta brainwave neurofeedback therapy for Vietnam veterans with combat-related post-traumatic stress disorder. *Medical Psychotherapy, 4*, 47–60.

Peniston, E. G., Marrinan, D. A., Deming, W. A., & Kulkosky, P. J. (1993). EEG alpha-theta brainwave synchronization in Vietnam theater veterans with combat-related post-traumatic stress disorder and alcohol abuse. *Advances in Medical Psychotherapy, 6*, 37–50.

Pop-Jordanova, N., & Zorcec, T. (2004). Child trauma, attachment and biofeedback mitigation. *Prilozi / Makedonska Akademija Na Naukite I Umetnostite, Oddelenie Za Biološki I Medicinski Nauki = Contributions / Macedonian Academy of Sciences and Arts, Section of Biological and Medical Sciences, 25*(1–2), 103–114.

Ros, T., Baars, B. J., Lanius, R. A., & Vuilleumier, P. (2014). Tuning pathological brain oscillations with neurofeedback: A systems neuroscience framework. *Frontiers in Human Neuroscience, 8*, 1008. http://dx.doi.org/10.3389 /fnhum.2014.01008

Smith, W. D. (2008). The effect of neurofeedback training on PTSD symptoms of depression and attention problems among military veterans. Capella University. Retrieved from http://gradworks.umi.com/33/15/3315214.html

van der Kolk, B. A., Hodgdon, H., Gapen, M., Musicaro, R., Suvak, M. K., Hamlin, E., & Spinazzola, J. (2016). A randomized controlled study of neurofeedback for chronic PTSD. PLoS ONE, 11(12), e0166752. http://dx.doi.org /10.1371/journal.pone.0166752

Personalized EEG-Neurofeedback as a Treatment for ADHD

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Several neurophysiological subtypes based on electroencephalographic (EEG) biomarkers have been identified in attention-deficit/hyperactivity disorder (ADHD; Johnstone, Gunkelman, & Lunt, 2005). However, most studies investigating the efficacy of neurofeedback (NFB) as a treatment for ADHD use uniform treatment protocols that are not taking into account individual EEG biomarkers (Arns, de Ridder, Strehl, Breteler, & Coenen, 2009). A recent pilot study suggests that personalizing NFB protocols to individual EEG biomarkers of ADHD might lead to increased specificity and efficacy of treatment (Arns, Drinkenburg, & Kenemans, 2012). Hence, the objective of this presentation is to investigate the effects of personalized EEG-NFB as a treatment for ADHD. It will provide an overview of personalized EEG-NFB protocols for ADHD and introduce results from a pilot project that aimed to integrate a neurofeedback clinic as part of the services offering of the Office for Students with Disabilities in a Canadian college. A hundred and eight college students with a diagnosis of ADHD received free personalized EEG-NFB twice a week over a period of 4 months. Half of the participants was randomly assigned to the experimental condition. The other half was put on a waiting list to serve as a control group and received treatment later. Resting-state EEG signals were recorded to evaluate overall brain activity pre- and posttraining, and to determine individual EEG-biomarkers for selection of personalized treatment protocol. ADHD behavioral symptoms were assessed pre- and posttraining using the Conners' Adult ADHD Rating Scale (CAARS-S:L), the Integrated Visual and Auditory Continuous Performance Test (IVA-2) and assessment of executive functions. A significant change was observed in subjects trained in EEG-NFB, both in brain activation patterns and at the behavioral level. More specifically, normalization of targeted resting brain waves was observed in the experimental group. Results from this pilot project demonstrate the feasibility of personalizing NFB protocols to individual EEG biomarkers of ADHD and the efficacy of NFB as a treatment for ADHD. On a broader level, this presentation will allow for a better understanding of the impact of neurofeedback training on neural and behavioral correlates of ADHD.

References

Arns, M., de Ridder, S., Strehl, U., Breteler, M., & Coenen, A. (2009). Efficacy of neurofeedback treatment in ADHD: The effects on inattention, impulsivity and hyperactivity: A meta-analysis. *Clinical EEG Neuroscience, 40*(3), 180–189. http://dx.doi.org/10.1177/155005940904000311

Arns, M., Drinkenburg, W., & Kenemans, J. L. (2012). The effects of qEEG-informed neurofeedback in ADHD: An open-label pilot study. *Applied Psychophysiology and Biofeedback, 37*(3), 171–180. http://dx.doi.org/10.1007 /s10484-012-9191-4

Johnstone, J., Gunkelman, J., & Lunt, J. (2005). Clinical database development: Characterization of EEG phenotypes. *Clinical EEG and Neuroscience, 36*(2), 99–107. http://dx.doi.org /10.1177/155005940503600209

The Nonlinear Brain: Investigating Neural Entrainment Using Missing Pulse Rhythms

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Introduction. Many rhythm perception experiments employ simple isochronous rhythms, in which synchronous neural or behavioral responses are observed (Bauer, Kreutz, & Herrmann, 2015; Repp, 2005a, 2005b). However, responses at the stimulus frequency do not allow one to distinguish whether synchrony occurs as a response to a common input or as the result of an emergent population oscillation that entrains at a particular frequency. It is possible to create a rhythm with no spectral energy at the pulse frequency by manipulating the number of events that occur anti-phase (180°) versus in-phase (0°) with the basic rhythmic cycle. Dynamical analysis predicts neural oscillation will emerge at such a "missing" pulse frequency (Large, 2010). Previous studies have shown that subjects tap along to complex rhythms at the missing pulse frequency (Large, Herrera, & Velasco, 2015)—a finding that supports the prediction, and responses at missing pulse frequencies have been seen in auditory brain areas using magnetoencephalography (MEG; Tal et al., 2017).

Aims. This study aimed to investigate whether the sensorimotor system, as measured by 32-channel cortical EEG, would entrain to a complex rhythm at the pulse frequency even when the complex rhythm contained no spectral power at that frequency.

Methods. The experiment utilized four different rhythms of varying complexity (one simple, two complex, and one random rhythm) created from 100 ms tones with a 200 Hz fundamental frequency (F0). Offline the EEG was decomposed into the cortical-steady state response (SS-EP) and the subcortical frequency following response (FFR). Fast Fourier Transform (FFT) of the Hilbert envelope showed energy at the repetition frequency (2 Hz) for the simple rhythm, but no spectral energy at the missing pulse frequency (2 Hz) for the complex rhythms. EEG responses to these stimuli were examined for evidence of neural oscillations and power modulations at the missing pulse frequency predicted by dynamical analysis.

Results. We report evidence of responses in the EEG to the pulse frequency of missing pulse rhythms. We also note a differing topography of power at the pulse frequency across the scalp for the complex rhythms versus the simple and random rhythms.

Conclusions. These data support the theory that rhythmic synchrony occurs as the result of an emergent population oscillation that entrains at this particular frequency. Additional analyses examined whether the FFR to the F0 is modulated by whether the stimuli are perceived as being on-beats or off-beats in the rhythmic context.

References

Bauer, A.-K. R., Kreutz, G., & Herrmann, C. S. (2015). Individual musical tempo preference correlates with EEG beta rhythm. *Psychophysiology, 52*(4), 600–604. http://dx.doi.org/10.1111 /psyp.12375

Large, E. W. (2010). Neurodynamics of music. In M. R. Jones, R. R. Fay, & A. N. Popper (Eds.), *Springer Handbook of Auditory Research, Volume 36: Music perception* (Vol. 36, pp. 201–231). Springer Science+Business Media, LLC. Retrieved from http://link.springer.com/10.1007/978-1-4419-6114-3_7

Large, E. W., Herrera, J. A., & Velasco, M. J. (2015). Neural networks for beat perception in musical rhythm. *Frontiers in Systems Neuroscience, 9*, 159. http://dx.doi.org/10.3389 /fnsys.2015.00159

Repp, B. H. (2005a). Rate limits of on-beat and off-beat tapping with simple auditory rhythms: 2. The roles of different kinds of accent. *Music Perception, 23*(2), 165–188.

Repp, B. H. (2005b). Sensorimotor synchronization: A review of the tapping literature. *Psychonomic Bulletin & Review, 12*(6), 969–992. http://dx.doi.org/10.3758/BF03206433

Tal, I., Large, E. W., Rabinovitch, E., Wei, Y., Schroeder, C. E., Poeppel, D., & Golumbic, E. Z. (2017). Neural entrainment to the beat: The "missing-pulse" phenomenon. *The Journal of Neuroscience, 37*(26), 6331–6341. http://dx.doi.org /10.1523/JNEUROSCI.2500-16.2017

STUDENT AWARD PRESENTATIONS – POSTERS

Noninvasive Cranial Nerve Stimulation for Human Cognitive Performance Enhancement

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

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

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

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

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

References

Berry, S. M., Broglio, K., Bunker, M., Jayewardene, A., Olin, B., & Rush, A. J. (2013). A patient-level meta-analysis of studies evaluating vagus nerve stimulation therapy for treatment-resistant depression. *Medical Devices: Evidence and Research, 6*, 17–35. http://dx.doi.org/10.2147/MDER.S41017

DeGiorgio, C. M., Soss, J., Cook, I. A., Markovic, D., Gornbein, J., Murray, D., … Heck, C. N. (2013). Randomized controlled trial of trigeminal nerve stimulation for drug-resistant epilepsy. *Neurology, 80*(9), 786–791. http://dx.doi.org/10.1212/WNL.0b013e318285c11a

Sara, S. J. (2009). The locus coeruleus and noradrenergic modulation of cognition. *Nature Reviews Neuroscience, 10*(3), 211–223. http://dx.doi.org/10.1038/nrn2573

Tyler, W. J., Boasso, A. M., Mortimore, H. M., Silva, R. S., Charlesworth, J. D., Marlin, M. A., … Pal, S. K. (2015). Transdermal neuromodulation of noradrenergic activity suppresses psychophysiological and biochemical stress responses in humans. *Scientific Reports, 5*, 13865. http://dx.doi.org/10.1038/srep13865

The Effects of ALAY and High Beta Down-train Neurofeedback for Patients Who Comorbid with Major Depressive Disorder and Anxiety Symptoms

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

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

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

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

References

Bruder, G. E., Fong, R., Tenke, C. E., Leite, P., Towey, J. P., Stewart, J. E., ... Quitkin, F. M. (1997). Regional brain asymmetries in major depression with or without an anxiety disorder: A quantitative electroencephalographic study. *Biological Psychiatry, 41*(9), 939–948. http://dx.doi.org/10.1016/S0006-3223(96)00260-0

Grin-Yatsenko, V. A., Baas, I., Ponomarev, V. A., & Kropotov, J. D. (2009). EEG power spectra at early stages of depressive disorders. *Journal of Clinical Neurophysiology, 26*(6), 401–406. http://dx.doi.org/10.1097/WNP.0b013e3181c298fe

Mathersul, D., Williams, L. M., Hopkinson, P. J., & Kemp, A. H. (2008). Investigating models of affect: Relationships among EEG alpha asymmetry, depression, and anxiety. *Emotion, 8*(4), 560–572. http://dx.doi.org/10.1037/a0012811

Yamada, M., Kimura, M., Mori, T., & Endo, S. (1995). EEG power and coherence in presenile and senile depression. Characteristic findings related to differences between anxiety type and retardation type. *Nihon Ika Daigaku Zasshi = Journal of Nippon Medical School, 62*(2), 176–185. http://dx.doi.org/10.1272/jnms1923.62.176

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

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

Methods. Participants included 30 children diagnosed with ASD (15.6 ± 3.8 years old, 8 girls), and an age-matched control group of 30 typically-developing children (TD; 15.7 ± 3.9 years old, 7 girls). Subjects performed a modified Posner's attentional cueing task (Posner, 1980). In each trial, subjects were initially presented a visual "cue" stimulus on left or right side of the screen. After a 1- s delay, a "target" stimulus appeared on the same (congruent, 80%) or opposite (incongruent, 20%) side from the cue, and subjects used a left- or right-handed button press to

indicate the position of the target. In half of the trials, a more complex diagonal stimulus presentation was used. EEG data was collected for analysis of several event-related potentials (ERPs), including the lateralized readiness potential (LRP), a measurement of asymmetric brain activity that reflects preparation of contralateral limb movement (Eimer, 1998).

Results. Reaction time (RT) was lower for congruent trials compared to incongruent trials for both ASD (401 vs. 481 ms, $p < .0001$) and TD (339 vs. 374 ms, $p < .001$). Across all four task conditions, ASD group exhibited longer RTs and higher error rates compared to TD (441 vs. 358 ms, $p < .001$; 7.4 vs. 0.8 errors, $p < .0001$). Furthermore, increased task complexity resulted in lengthened RT in ASD group (447 vs. 430 ms, $p < .05$), and in TD group (376 vs. 360 ms, $p < .01$). While the amplitude the early (pretarget) component of LRP was significantly higher in ASD compared to TD (−0.95 vs. −0.23, $p < .05$), the late component (posttarget) showed no group differences (−0.53 vs. −0.50, n.s.). Moreover, analysis of ERPs showed several differences between ASD and TD groups, including frontal N100 amplitude, N100 latency, and N200 amplitude.

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

References

Dowell, L. R., Mahone, E. M., & Mostofsky, S. H. (2009). Association of postural knowledge and basic motor skill with dyspraxia in autism: Implication for abnormalities in disturbed connectivity and motor learning. *Neuropsychology, 23*(5), 563–570. http://dx.doi.org/10.1037/a0015640

Eimer, M. (1998). The lateralized readiness potential as an on-line measure of central response activation processes. *Behavior Research Methods, Instruments, & Computers, 30*(1), 146–156. http://dx.doi.org/10.3758/BF03209424

Posner, M. I. (1980). Orienting of attention. *Quarterly Journal of Experimental Psychology, 32*(1), 3–25. http://dx.doi.org/10.1080/00335558008248231

Weimer, A. K., Schatz, A. M., Lincoln, A., Ballantyne, A. O., & Trauner, D. A. (2001). "Motor" impairment in Asperger syndrome: Evidence for a deficit in propioception. *Journal of Developmental & Behavioral Pediatrics, 22*(2), 92–101. http://dx.doi.org/10.1097/00004703-200104000-00002

PLENARY SESSION PRESENTATIONS

The Importance of Morphology and Montaging in EEG

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Reading the raw EEG is an artform that is essential knowledge-base of any practitioner using EEG to assess and diagnose their patients' conditions. Spindles, triangular shapes, sinusoidal, monomorphic, and archiform waveforms are just a few telling morphological signs that are imperative in understanding what is really going on. Does the waveform wax and wane? Does it travel in spindles or bursts? Does it appear only a few times in the record? What if it is rhythmic? These temporal dynamics are also imperative in a proper assessment of the person. When looking at the raw waveform, you will learn more than what any qEEG, alone, can tell you.

Through exploring the more insidious forms of artifact (i.e., electricity, channel noise, mixed metals, etc.) to detecting less commonly seen morphological forms in the EEG (i.e., lambda, mu, OIRDA, beta spindles, etc.), this lecture will guide the clinician through some of the more advanced ways of interpreting EEG so that the qEEG does not mislead one into misdiagnosis.

We are privileged to have many analysis and diagnostic tools to help us dissect, spatially and temporally analyze, condense, and summate the EEG into neat and tidy diagrams, but we fail our patients and our profession if we miss the devils in the details.

Finally, montages are necessary to understand the many ways in which we can assess and view the EEG. There is no best montage for all purposes. While linked ears can provide a global view, it is prone to contamination if there is a strong temporal finding or if there is contamination otherwise in the ear electrodes. Average and weighted average montages (such as the Laplacian and Hjorth montages), will highlight any local phenomena, and will uncover any significant temporal component, but will fail us to see global information. Bipolar montages are excellent for displaying phase

reversals, which are indispensable in issues of head injury and seizure focus.

References

Buzsáki, G. (2006). *Rhythms of the brain*. New York, NY: Oxford University Press.

Buzsáki, G., & Draguhn, A. (2004). Neuronal oscillations in cortical networks. Science, 304(5679), 1926–1929. http://dx.doi.org/10.1126/science.1099745

Sporns, O., Tononi, G., & Edelman, G. M. (2000a). Connectivity and complexity: The relationship between neuroanatomy and brain dynamics. *Neural Networks, 13*(8–9), 909–922. http://dx.doi.org/10.1016/S0893-6080(00)00053-8

Sporns, O., Tononi, G., & Edelman, G. M. (2000b). Theoretical neuroanatomy: Relating anatomical and functional connectivity in graphs and cortical connection matrices. *Cerebral Cortex, 10*(2), 127–141. http://dx.doi.org/10.1093 /cercor/10.2.127

Stern, J. M. (2005). *Atlas of EEG patterns* (2nd ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

The Impact of Using Effective Connectivity Measures (Granger Causality) in Guiding Neurofeedback

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Over the past several years, we have seen advancements in the ways we assess coherence and connectivity that provide great insights into brain functioning (Coben, Mohammad-Rezazadeh, & Cannon, 2014). This understanding has led to approaching coherence in a multivariate fashion that enhances its accuracy (Kuś, Kamiński, & Blinowska, 2004). Multivariate autoregressive statistical tools have become critical to this endeavor. Such techniques enable us to measure effective connectivity in a source localized fashion such that we can image reciprocal causality and influence. This accuracy in depicting neural networks gets us closer to the real signals in the brain. This led to an enhancement in how we do neurofeedback training which now uses four sensors and trains coherence in a multivariate fashion (Coben, Middlebrooks, Lightstone, & Corbell, 2018).

We have adopted a theory that states the more accurate our assessment of connectivity and the source of the activity then the more effective our attempts at neurofeedback may be. In this pilot study we evaluated the effects of basing our neurofeedback (multivariate coherence training) protocols on coherence measures as compared to multivariate autoregressive effective connectivity that uses sources to estimate reciprocal causality (see Friston, Moran, & Seth, 2013). We sampled 45 subjects with various presenting complaints and divided up into three groups. All subjects had their neurofeedback protocols developed based on qEEG methods. Group 1 was based on ICA and effective connectivity Granger causality, and groups 2 and 3 were based on correlational coherence measures. We used two separate comparison groups, one served as a within-group and the other a between-group comparison.

All subjects underwent between 12 and 15 neurofeedback sessions followed by another assessment period. Dependent measures included EEG comparisons of power and graph theory metrics of effective connectivity. Clinical comparisons were also made based on rating of progress to measure their symptoms change over this period of time. Preliminary data analysis has shown that all groups show changes and gains from their neurofeedback training, but that the group that had their protocols based on effective connectivity measures showed greater clinical and EEG changes. The implications of these findings will be presented.

References

Coben, R., Middlebrooks, M., Lightstone, H. & Corbell, M. (in press). Four- channel multivariate coherence training: Development and evidence in support of a new form of neurofeedback. *Frontiers in Neural Technology*.

Coben, R., Mohammad-Rezazadeh, I., & Cannon, R. L. (2014). Using quantitative and analytic EEG methods in the understanding of connectivity in autism spectrum disorders: A theory of mixed over-and under-connectivity. *Frontiers in Human Neuroscience, 8*, 45. http://dx.doi.org/10.3389 /fnhum.2014.00045

Friston, K., Moran, R., & Seth, A. K. (2013). Analysing connectivity with Granger causality and dynamic causal modelling. *Current Opinion in Neurobiology, 23*(2), 172–178. http://dx.doi.org/10.1016/j.conb.2012.11.010

Kuś, R., Kamiński, M., & Blinowska, K. J. (2004). Determination of EEG activity propagation: Pair-wise versus multichannel estimate. *IEEE Transactions on Biomedical Engineering, 51*(9), 1501–1510. http://dx.doi.org/10.1109 /TBME.2004.827929

Trends in Scientific Research Reflect and Predict the Clinical Relevance of (EEG) Biomarkers

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QEEG-guided neurofeedback is based on interpreting abnormalities in the resting-state EEG in relationship with psychopathology. Setting up effective neurofeedback treatment protocols relies on the correct interpretation of individual qEEG profiles in relationship with the symptoms of the patient. Scientific studies have demonstrated associations between certain deviations in resting-state EEG and

specific psychological disorders. The most well-known link is that of excess theta/beta ratio in relation with ADHD (e.g., Arns, Conners, & Kraemer, 2013).

Recent approval of the FDA for an ADHD diagnostic test based on excess theta/beta ratio illustrates that this “EEG biomarker” is both meaningful and reliable. Other markers for psychopathology include ”alpha asymmetry” for depression (Thibodeau, Jorgensen, & Kim, 2006) and excess beta power for anxiety and insomnia (Pavlenko, Chernyi, & Goubkina, 2009; Perlis, Merica, Smith & Giles, 2001). However, a modern qEEG report will contain many different and detailed analyses of an individual EEG. Moreover, the number of analyses that can be performed on EEG data has been increasing rapidly. Interpreting the clinical relevance of these analyses for the treatment of an individual patient depends on the scientific studies demonstrating links between these measures and the symptoms of the patient.

In the current presentation, the trends in qEEG research will be discussed and an attempt will be made to assess the relevance of different qEEG analyses for clinical purposes using a systematic analysis of the scientific literature. The main approach is to analyze the number of papers published on a particular search term (reflecting a certain EEG biomarker) per year. A secondary approach is to analyze the number of citations per year to seminal papers which describe a new EEG biomarker. Finally, comparisons with scientific literature in different fields may provide useful analogies. For example, the interpretation of an individual blood test for cancer relies on scientific research on the association between certain biomarkers (e.g., certain proteins) and the presence of a tumor. How did scientific research on these ”tumor markers” evolve and eventually lead up to the use of tumor markers in clinical testing today, and what can this tell us about the current state-of-the-art and future clinical relevance of different EEG analyses?

One hypothesis is that papers which demonstrate a promising new biomarker will show a continuous increase in citations per year when the biomarker can be replicated and when it is useful as a diagnostic tool in clinical practice. In contrast, when such a study cannot be replicated it may show an initial increase of citations, but the number of citations per year will inevitably go down after this initial increase. In the latter case, it can be concluded that the biomarker is not useful in clinical practice. Analyzing trends in the scientific literature to predict clinical relevance of potential EEG biomarkers is novel and relevant approach which may have important implications for the scientific and clinical field of qEEG and neurofeedback.

References

Arns, M., Conners, C. K., & Kraemer, H. C. (2013). A decade of EEG theta/beta research in ADHD: A meta-analysis. *Journal of Attentional Disorders, 17*(5), 374–383. http://dx.doi.org/10.1177/1087054712460087

Pavlenko, V. B., Chernyi, S. V., & Goubkina, D. G. (2009). EEG correlates of anxiety and emotional stability in adult healthy subjects. *Neurophysiology, 41*(5), 337–345. http://dx.doi.org/10.1007/s11062-010-9111-2

Perlis, M. L., Merica, H., Smith, M. T., & Giles, D. E. (2001). Beta EEG activity and insomnia. *Sleep Medicine Reviews, 5*(5), 363–374. http://dx.doi.org/10.1053/smrv.2001.0151

Thibodeau, R., Jorgensen, R. S., & Kim, S. (2006). Depression, anxiety, and resting frontal EEG asymmetry: A meta-analytic review. *Journal of Abnormal Psychology, 115*(4), 715–729. http://dx.doi.org/10.1037/0021-843X.115.4.715

Understanding the Mysterious 40 Hz Brain System for Attention, Learning, and Feeling Good

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Evidence from a wide variety of studies and authors supports a new synthesis of understanding regarding the 40 Hz brain scanning system and its role in attention, understanding, learning, and creating positive feelings as a reward for the effort. It is a basic foundation for the individual’s survival, promoting their effective responses to new discoveries and situations. We have therefore named this brain system Neureka! (short for Neural Eureka!). This understanding builds upon Llinas’ discovery of the “Event Binding Rhythm” (Llinas, Ribary, Contreras, & Pedroarena,1998), which scans the cortical layers from front to back 40 times a second and reports back to its origin, the centrally located nuclei in the thalamus, all of which we will review here.

These nuclei synthesize all this information and send out modified 40 Hz scanning rhythms which look for additional information to add to the understanding of a particular new event. This looping information exchange continues until the event is evaluated and a response is created. Salient events are stored in short-term memory, particularly in the prefrontal cortex (PFC). Short-term memory is then converted to longer term memory, particularly if dopamine, norepinephrine, and/or other neuromodulators are released. There is evidence that both of these

neuromodulators are released in the PFC, particularly near FPz. Dopamine also creates a variety of positive feelings when it is released there.

We will review several lines of evidence about the Neureka! brain system from fMRI and other scans (Knutson, Fong, Bennett, Adams, & Hommer, 2003), and complement them with our EEG studies based on the Neureka! measurement, which selectively clarifies this particular 40 Hz rhythm and separates it from the other 40 Hz activity passing through more peripheral parts of the thalamus.

These studies show that neurofeedback training of Neureka! enhances memory and happiness (for at least four months) and decreases depressed feelings. It also improves memory and attention measurements (Sokhadze & Daniels, 2016). Previous studies (Cowan & Albers, 2011; Cowan & Starman, 2017; Rubik, 2011) demonstrate clear relationships between increases in Neureka! amplitude and love, happiness, satisfaction, gratitude, and appreciation. We will review studies where neurofeedback training using the Neureka! measure (Sokhadze, 2012) was used to improve behavioral symptoms in children with autism (Wang et al., 2016). Furthermore, the 40 Hz rhythm was used to distinguish the emotional reaction to drug and stress cues of substance abusers from those with comorbid PTSD (Sokhadze et al., 2009).

References

Cowan, J., & Albers, S. (2017). Manual for the Peak Brain Happiness Trainer. Goshen, KY: Peak Achievement Training.

Cowan, J. D., & Starman, J. D. (2011). Understanding and activating your brain's pleasure systems. Retrieved from http://www.peakachievement.com/UABC.pdf

Knutson, B., Fong, G. W., Bennett, S. M., Adams, C. M., & Hommer, D. (2003). A region of mesial prefrontal cortex tracks monetarily rewarding outcomes: Characterization with rapid event-related fMRI. *NeuroImage, 18*(2), 263–272. http://dx.doi.org/10.1016 /S1053-8119(02)00057-5

Llinás, R., Ribary, U., Contreras, D., & Pedroarena, C. (1998). The neuronal basis for consciousness. *Philosophical Transactions of the Royal Society of London, Series B, Biological Sciences, 353*(1377), 1841–1849. http://dx.doi.org /10.1098/rstb.1998.0336

Rubik, B. (2011). Neurofeedback-enhanced gamma brainwaves from the prefrontal cortical region of meditators and non-meditators and associated subjective experiences. *Journal of Alternative and Complementary Medicine, 17*(2), 109–115. http://dx.doi.org/10.1089/acm.2009.0191

Sokhadze, E. (2012). Peak performance training 7sing prefrontal EEG biofeedback. *Biofeedback, 40*(1), 7–15. http://dx.doi.org /10.5298/1081-5937-39.3.4

Sokhadze, E., & Daniels, R. (2016). Effects of prefrontal 40 Hz-centered EEG band neurofeedback on emotional state and cognitive functions in adolescents. *Adolescent Psychiatry, 6*(4), 116-129.

Sokhadze, E., Stewart, C., El-Baz, A., Ramaswamy, R., Hollifield, M., & Tasman, A. (2009). Induced EEG gamma oscillations in response to drug- and stress-related cues in cocaine addicts and patients with dual diagnosis. *Journal of Neurotherapy, 13*(4), 270–271.

Wang, Y., Sokhadze, E. M., El-Baz, A. S., Li, X., Sears, L., Casanova, M. F., & Tasman, A. (2016). Relative power of specific EEG bands and their ratios during neurofeedback training in children with autism spectrum disorder. *Frontiers in Human Neuroscience, 9,* 723. http://dx.doi.org/10.3389 /fnhum.2015.00723

Gender Differences in Quantitative EEG Volumetric Analysis Shortly After Sport Concussion Injury in High School Athletes

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Introduction. We have previously reported changes in sLORETA quantitative analysis shortly after acute sport-related concussion injury in high school athletes, which persist after clinical recovery (Kerasidis & Ims, 2017). We have also reported the effects of neurofeedback on these changes during the acute recovery period (Ims & Kerasidis, 2018). Studies have identified gender differences in the incidence, severity, and recovery time from sport concussion injury, all increased in females (Cantu, 2010; Hamson-Utley et al., 2013; Miyashita, Diakogeorgiou, & VanderVegt, 2016; Mollayeva, El-Khechen-Richandi, & Colantonio, 2018; Tanveer, Zecavati, Delasobera, & Oyegbile, 2017). The objective of this investigation is to explore gender differences in volumetric qEEG analysis after sport concussion injury in high school athletes.

Methods. Standard electroencephalograms (EEGs) were analyzed in 40 high school athletes (20 males) shortly after concussion injury using sLORETA imaging compared to a normative database (NYU/BrainDx). Peak *Z*-score variation (PZV), and percentage of volume of grey matter activity that fell outside $Z = -2.5$ to 2.5 (PIGMV for increased activity, PRGMV for reduced) were calculated for each of five EEG frequency bands.

Results. PZV was increased in the Delta/Theta/Alpha in both genders with no statistical gender difference (M/F averages: 3.82/3.16, 2.73/2.72, 2.52/2.72, respectively, $p > .05$); Beta in females, not males, Beta-Gamma in males and females which was significantly increased in females (M/F averages: 1.75/2.88, 3.64/5.02 respectively, $p < .01$). PZV was decreased in Beta in males, not females (M/F averages: −2.83/−2.18, $p < .05$). There was a significant difference in reduced Beta-Gamma

activity (M/F averages: −1.11/−0.49, p = .01). Greater than 1% grey matter volume of PIGMV was seen in Delta/Theta/Alpha/Beta and Beta-Gamma activity with no gender difference (M/F averages: 20.94/11.71, 5.87/7.38, 5.62/7.93, 4.09/9.22, p > .05). There was a significant difference in PIGMV in Beta-Gamma (M/F averages: 31.94/60.04, p = .01). Greater than 1% PRGMV in Alpha/Beta in both genders and Theta activity in females, not males.

Conclusions. Slower frequency (Delta, Theta, and Alpha) abnormal variations show no statistical gender differences. In the faster frequency bands (Beta and Beta-Gamma), females demonstrate a larger variation from the norm and larger percent grey matter volume affected by increased Beta and Beta-Gamma activity. Males, not females, exhibit a deficiency in Beta activity after concussion. Further research to correlate these electrophysiologic changes with symptom severity and recovery time is needed.

References

Cantu, R. C. (2010). The role of concussion history and gender in recovery from soccer-related concussion. *Yearbook of Sports Medicine, 2010*, 29–30. http://dx.doi.org/10.1016/s0162-0908(10)79666-5

Hamson-Utley, J. J., Schulte, S., Fowler, L., Glodowski, C., Scharmann, S., Podlog, L., … Ashley, A. (2013, July). Concussion-related neuroproteins: A comparison of gender differences in extreme sports. Poster presented at the 122nd Annual Conference of the American Psychological Association, Honolulu, HI. http://dx.doi.org/10.1037/e620352013-001

Ims, P. D., & Kerasidis, H. (2018, April). Re-training the injured brain: A case series in sLORETA neurofeedback as an acute concussion intervention in youth. Poster session presented at the 49th Annual Association for Applied Psychophysiology and Biofeedback Conference, Orlando, FL.

Kerasidis, H., & Ims, P. D. (2017, July). sLORETA quantitative EEG analysis demonstrates persistent EEG changes beyond clinical recovery from sport concussion in high school athletes: A volumetric study. Poster session presented at 4th Annual American Academy of Neurology Sports Concussion Conference, Jacksonville, FL.

Miyashita, T. L., Diakogeorgiou, E., & VanderVegt, C. (2016). Gender differences in concussion reporting among high school athletes. *Sports Health, 8*(4), 359–363. http://dx.doi.org/10.1177/1941738116651856

Mollayeva, T., El-Khechen-Richandi, G., & Colantonio, A. (2018). Sex & gender considerations in concussion research. *Concussion, 3*(1). http://dx.doi.org/10.2217/cnc-2017-0015

Tanveer, S., Zecavati, N., Delasobera, E. B., & Oyegbile, T. O. (2017). Gender differences in concussion and postinjury cognitive rindings in an older and younger pediatric population. *Pediatric Neurology, 70*, 44–49. http://dx.doi.org/10.1016/j.pediatrneurol.2017.02.001

Social, Spiritual, Psychological, and Physiological Predictors of Well-being of Military Veterans: A Pilot Study of a Viable, Holistic, and Predictive Model of Well-being

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Military leaders are striving to identify and implement innovative and necessary solutions to enhance or optimize military members' well-being, with the ultimate goal of improving the short- and long-term well-being of warriors and their families. This study tested the viability of a holistic model of well-being that was developed as a screening instrument. A secondary goal is to mitigate the detrimental effects of the high operational tempo and the extreme pressures faced by active duty military veteran community. Therefore, to be adequate the model had to be predictive of well-being to serve as such a baselining and monitoring tool. This new model involves a holistic, systems approach, integrating four key life domains that were hypothesized to impact overall well-being: human, psychological, social, and spiritual performance. These domains are interconnected and work together via situational, dispositional, and intentional variables to produce well-being, or the lack thereof (Howell, Kern, & Lyubomirsky, 2007). To test the viability and utility of this model, a stepwise multiple regression analysis was conducted on archival data of 117 military veterans. Based on the literature of, and the shared nomological network between, well-being (e.g., Howell et al., 2007), PsyCap (e.g., Avey, 2014; Lorenz, Beer, Pütz, & Heinitz, 2016), social isolation or connectedness (e.g., Kent, Hawthorne, Kjaer, Manniche, & Albert, 2015), spiritual intelligence (e.g., Faraji, & Begzadeh, 2017), psychological and cognitive performance (e.g., Del Brutto et al., 2015; Lathan, Spira, Bleiberg, Vice, & Tsao, 2013), as well as heart rate variability (HRV; e.g., Fatisson, Oswald, & Lalonde, 2016) and quantitative electroencephalogram (qEEG) metrics (e.g., Thatcher, North, Biver, & Zhou, 2017), it was hypothesized that human performance (Brain Function Index or BFI [qEEG] and SDNN [HRV]), psychological performance (DASS-21 composite score), social performance (Friendship Scale composite score), and spiritual performance (SISRI-24 composite score) would significantly predict well-being (Psychological Capital or PsyCap composite score). This set of predictors is hypothesized to account for a significant proportion of the well-being or PsyCap variance (i.e., CPC-12 composite scores; PsyCap). Furthermore, each predictor is hypothesized to explain a unique and significant

proportion of the PsyCap variance. The expected results would suggest that the positive core construct of PsyCap can be predicted using self-report measures addressing each domain, combined with functional measures (i.e., BFI and SDNN) and cognitive performance assessment outcome measures (i.e., Defense Automated Neuropsychological Assessment [DANA]). Moreover, such findings would support a viable model of well-being, which military leaders can use to baseline and monitor its members.

References

Avey, J. B. (2014). The left side of psychological capital: New evidence on the antecedents of PsyCap. *Journal of Leadership & Organizational Studies, 21*(2), 141–149. http://dx.doi.org/10.1177/1548051813515516

Del Brutto, O. H., Mera, R. M., Del Brutto, V. J., Maestre, G. E., Gardener, H., Zambrano, M., & Wright, C. B. (2015). Influence of depression, anxiety and stress on cognitive performance in community-dwelling older adults living in rural Ecuador: Results of the Atahualpa Project. *Geriatrics & Gerontology International, 15*(4), 508–514. http://dx.doi.org /10.1111/ggi.12305

Faraji, M., & Begzadeh, S. (2017). The relationship between organizational commitment and spiritual intelligence with job performance in physical education staff in east Azerbaijan province. *International Journal of Management, Accounting & Economics, 4*(5), 565–577.

Fatisson, J., Oswald, V., & Lalonde, F. (2016). Influence diagram of physiological and environmental factors affecting heart rate variability: An extended literature overview. *Heart International, 11*(1), e32–e40. http://dx.doi.org/10.5301 /heartint.5000232

Howell, R. T., Kern, M. L., & Lyubomirsky, S. (2007). Health benefits: Meta-analytically determining the impact of well-being on objective health outcomes. *Health Psychology Review, 1*(1), 83–136. http://dx.doi.org/10.1080 /17437190701492486

Kent, P., Hawthorne, G., Kjaer, P., Manniche, C., & Albert, H. (2015). A Danish version of the Friendship Scale: Translation and validation of a brief measure of social isolation. *Social Indicators Research, 120*(1), 181–195. http://dx.doi.org /10.1007/s11205-014-0576-z

King, D. B., & DeCicco, T. L. (2009). A viable model and self-report measure of spiritual intelligence. *The International Journal of Transpersonal Studies, 28(*1), 68–85.

Lathan, C., Spira, J. L., Bleiberg, J., Vice, J., & Tsao, J. W. (2013). Defense Automated Neurobehavioral Assessment (DANA)—Psychometric properties of a new field-deployable neurocognitive assessment tool. *Military Medicine, 178*(4), 365–371. http://dx.doi.org/10.7205/MILMED-D-12-00438

Lorenz, T., Beer, C., Pütz, J., & Heinitz, K. (2016). Measuring psychological capital: Construction and validation of the Compound PsyCap Scale (CPC-12). *PloS ONE, 11*(4), 1–17. http://dx.doi.org/10.1371/journal.pone.0152892

Luthans, F., Youssef, C. M., Sweetman, D. S., & Harms, P. D. (2013). Meeting the Leadership Challenge of Employee Well-Being Through Relationship PsyCap and Health PsyCap. *Journal of Leadership & Organizational Studies, 20*(1), 118–133. http://dx.doi.org/10.1177/1548051812465893

Thatcher, R. W., North, D. M., Biver, C. J. & Zhou, L. (2017). Brain Function Index. Retrieved from http://www.appliedneuroscience.com/Brain_Function_Index.pdf

Altered States NeuroMeditation: Current Approaches, Preliminary Findings, and Future Applications

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Using neurofeedback and other technologies to achieve altered states of consciousness has its roots in the early development of the field of neurofeedback. In fact, some of the initial neurofeedback protocols were designed to replicate many of the effects commonly experienced during a meditative state (Crane, 2007; Trudeau, 2016). Having observed that the practice of meditation often led to an increase in alpha power or increased theta activity crossing over alpha, these two approaches became the foundation of Deep States NeuroMeditation protocols, essentially attempting to facilitate elements of an altered state (Tarrant, 2017).

This work and these protocols are powerful and have been associated with impressive results with difficult clinical populations including alcoholics and those suffering with PTSD. Recent explorations into the study of consciousness has led to some new approaches and protocols for assisting clients into achieving altered states for the purposes of psychological and emotional healing.

This presentation will present a new and novel approach to achieving altered states with neurofeedback by replicating brain-based research on psychedelic therapies. Current research with psilocybin, LSD, DMT, and other psychedelics have all shown tremendous potential in treating a wide range of mental health disorders. It is believed that these impacts are due, at least in part, to the way they alter perception and the dysfunctional creation of a self-identity (Carhart-Harris et al., 2012). Based on this emerging field, certain brain patterns and brain regions have revealed themselves as important in these transformative experiences. By targeting these brain patterns through neurofeedback, in conjunction with additional strategies, we may be on the cusp of a brand-new approach to the use of neurofeedback. In this presentation, we will share preliminary research showing how these altered states can be facilitated using neurofeedback. We will explore approaches, indications and contraindications as well as a variety of adjunctive aids, including vibroacoustics, evocative music, and visual entrainment.

References

Carhart-Harris, R. L., Erritzoe, D., Williams, T., Stone, J. M., Reed, L. J., Colasanti, A., … Nutt, D. J., (2012). Neural correlates of the psychedelic state as determined by fMRI studies with psilocybin. *Proceedings of the National Academy of Sciences (PNAS), 109*(6), 2138–2143. http://dx.doi.org/10.1073/pnas.1119598109

Crane, R. (2007). Infinite potential: A neurofeedback pioneer looks back and ahead. In T. H. M. Press (Ed.), *Handbook of neurofeedback: Dynamics and clinical applications: Haworth series in neurotherapy* (pp. 3–21). Binghamton, NY: CYC Press.

Tarrant, J. (2017). Neuromeditation: An introduction and overview. In T. F. Collura & J. A. Frederick (Eds.), *Clinician's companion to QEEG and neurofeedback* (annotated and with an introduction by J. Kiffer). New York, NY: Taylor & Francis.

Trudeau, D. L. (2016). Experiences with alpha theta: Its origins in studies of meditation. In A. Martins-Mourao, & C. Kerson (Eds.), *Alpha-theta training in the 21st century: A handbook for clinicians and researchers* (pp. 36-64). Murfreesboro, TN: Foundation for Neurofeedback and Neuromodulation Research (FNNR).

Cognitive and Psychophysiological Test Operations as Assessment Tool for Neurofeedback Clinicians: A Pilot Study on Its Preliminary Normative Data and Validity

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Neurofeedback is a paradigm in which individuals are trained to modulate their electroencephalogram (EEG) by providing them feedback about the targeted EEG component to treat symptoms and disorders associated to the neuronal condition. Neurofeedback shows effects on both the alleviation of symptoms and also changes in cognitive performance as an appreciated side effect or as bringing up the desired/expected ability like improved continuous attention, focus, and impulse control. For example, training the alpha band frequency has been associated with improved attentional control and working memory. Investigating attentional control is typically done with tasks such as the Stroop task, in which a color word is shown in a font color that is different (incongruent) than what the word represents and the participant is asked to name only the font color. Tests have shown that there is often a remarkable decrease in response time (delay) and an increase of accuracy from pre- to postneurofeedback sessions. In general, the Stroop test could be used to mark objectively success of neurofeedback over time. The problem is that that clinicians complain about the time which is spent to administer the Stroop test, which makes it interesting for research but not for clinical praxis.

The purpose of this study was to obtain normative data of a battery of informatized tests from the software Cognitive and Psychophysiological Test Operations (CAPITO) and its comparation with classical neuropsychological tests in order to assure construct validity with the goal to create a test which is suitable for use by primary care medical staff, psychologists, and neuropsychologists, since it can be administered in just 10 minutes.

We administered a battery of informatized tests (Stroop, simple reaction time, sustained attention, shifting attention) to 120 subjects who are cognitively normal and range in age from 18 to 65 years, of whom 26 randomly selected subjects also scored in classical tests in order to check battery validity (confidence level of 90%, sampling margin of error 15%). In the case of subjects receiving both modalities (informatized and classical testing) the order of application was balanced, in order to avoid application order bias. Statistics of each test scores were calculated and comparisons between informatized and classical tests were conducted.

Normative data were collected for CAPITO battery and positive correlations with classical tests were found. No effects were found for age and sex in either test. Educational level impacted the Stroop test variables but not the other tests.

References

Brauer-Boone, K., Pontón, M. O., Gorsuch, R. L., González, J. J., & Miller, B. L. (1998). Factor analysis of tour measures of prefrontal lobe functioning. *Archives of Clinical Neuropsychology, 13*, 585–595.

De la Torre, G. G. (2002). El modelo funcional de atención en neuropsicología. *Revista de Psicología General y Aplicada, 55*(1), 113–121.

Fan, J., McCandliss, B. D., Fossella, J., Flombaum, J. I., & Posner, M. I. (2005). The activation of attentional networks. *NeuroImage, 26*(2), 471–479. http://dx.doi.org/10.1016/j.neuroimage.2005.02.004

Fan, J., McCandliss, B. D., Sommer, T., Raz, M., & Posner, M. I. (2002). Testing the efficiency and independence of attentional networks. *Journal of Cognitive Neuroscience, 14*(3), 340–347. http://dx.doi.org/10.1162/089892902317361886

Pineda, D. A., Merchán, V., Rosselli, M., & Ardila, A. (2000). Estructura factorial de la función ejecutiva en estudiantes universitarios jóvenes = Factor structure of the executive function in young university students. *Revista de Neurología, 31*(12), 1112–1118.

Posner, M. I., & Dehaene, S. (1994). Attentional networks. *Trends in Neurosciences, 17*(2), 75–79.

Spikman, J., Kiers, H. A. L., Deelman, B. G., & van Zomeren, A. H. (2001). Construct validity of concepts of attention in healthy controls and patients with CHI. *Brain and Cognition, 47*(3), 446–460. http://dx.doi.org/10.1006/brcg.2001.1320

Training Blood Flow: nHEG Utilization for Specific qEEG Phenotypes in ASD

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The spectrum of autistic disorders is among the most heterogeneous, both in regard to electrophysiology and to metabolism. Specific to brain electrophysiology, research points to multiple noted outlying quantitative EEG features; consistent with the heterogeneity of the symptoms of this disorder. That variation of noted phenomenon has led to various EEG neurofeedback training strategies over the past two decades. Many of these strategies have undergone clinical research and, while encouraging, have presented similarly heterogeneous outcomes. It is still an active discussion with regard to which symptom features of ASD are specific to which neurofeedback protocols. Furthermore, protocols that address frontal pole delta or frontotemporal and temporal beta in low-functioning patients are challenging as these features are often confounded by muscle artifact and eye movement during training. In this presentation, we propose an alternative neurofeedback training protocol that can be used to improve particularly the frontal and temporal region dysfunctions that play a role in regulating attention and response control, and that is nearly impervious to the contamination of eye movement or muscle tension. One of the systemic dysregularities noted in the ASD population is a variety of metabolic imbalances which we argue can be presented as diffuse, low absolute power measures spanning two or more frequency bands in the qEEG. The specific physiological mechanism for this phenotypic pattern is not yet well understood but one rationale is that as a result of underlying metabolic dysregulation, oxygen perfusion is reduced resulting in reduced cell energy and subsequent low power. With this supposition, the authors have been using a nHEG (Toomim) training protocol since 2011. We have selected from 50 of the qualifying clinical nHEG training cases run in 2016 and 2017. The multicase study reveals significant improvement in the qEEG features from baseline to subsequent evaluations following approximately 10 hours of treatment. These results are compared to a protocol of neurofeedback therapy that trains to increase absolute power by rewarding EEG absolute power parameters alone.

References

Chabot, R. J., Coben, R., Hirshberg, L. & Cantor, D. S. (2015). QEEG and VARETA based Neurophysiological Indices of Brain Dysfunction in Attention Deficit and Autistic Spectrum Disorder. *Austin Journal of Autism & Related Disabilities, 1*(2), 1007.

Dias, A. M., Van Deusen, A. M., Oda, E., & Bonfim, M. R. (2012) Clinical efficacy of a new automated hemoencephalographic neurofeedback protocol. *Spanish Journal of Psychology, 15*(3), 930–941.

Edelson, S. B., & Cantor, D. S. (1998). Autism: Xenobiotic influence. *Toxicology and Industrial Health, 14*(6), 799–811. http://dx.doi.org/10.1177%2F074823379801400603

Kouijzer, M. E. J., van Schie, H. T., Gerrits, B. J. L., Buitelaar, J. K., & de Moor, J. M. H. (2013). Is EEG-biofeedback an Effective Treatment in Autism Spectrum Disorders? A Randomized Controlled Trial. *Applied Psychophysiology and Biofeedback, 38*(1), 17–28. http://dx.doi.org/10.1007/s10484-012-9204-3

Wilcox, J., Tsuang, M. T., Ledger, E., Algeo, J., & Schnurr, T. (2002). Brain perfusion in autism varies with age. *Neuropsychobiology. 46*, 13–16. http://dx.doi.org/10.1159/000063570

Applied Innovation in Clinical Practice — Let's Go Beyond Neurofeedback

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Many neurofeedback practitioners utilize multiple modalities in practice to enhance the effects of neurofeedback. Adjunct therapies such as counseling, transcranial direct current stimulation (tCDS), heart rate variability training, and biofeedback, among others, to improve patient outcomes. Two methodologies, bilateral alternating stimulation in tactile form (BLAST) and cranial electrical stimulation (CES) also show promise in altering electrical activity in key networks associated with stress (Feusner et al., 2012; Serin, Hageman, & Kade, 2018) and can be used in conjunction with traditional neurofeedback. However, many clinicians do not have a model for how to apply these modalities in practice, nor have they reviewed the emerging data on the modalities. Beta EEG rhythm has been found to correlate to high situational and personal anxiety (Pavlenko, Chernyi, & Goubkina, 2009) and BLAST has been found to significantly reduce beta activity, subjective distress and physiological body sensations in response to thinking about a stressful event (Serin et al., 2018) by possibly depotentiating amygdala activity (Harper, Rasolkhani-Kalhorn, & Drozd, 2009) which is responsible activating the body's stress response (Ehrlich et al., 2009). The use of CES may result in cortical deactivation, may alter brain activity in the default mode network (DMN), and may create significant changes in intrinsic connectivity networks (Feusner et al., 2013). The body of literature is

growing with regard to these two methodologies, and clinicians can utilize them in conjunction with traditional neurofeedback to achieve specific outcomes in treatment with patients with anxiety, insomnia, depression, and varied diagnoses. A review of clinical data, biometric data, EEG findings, and other research will be presented, along with guidelines for clinical use of the modalities and a system and structure for incorporation into clinical practice. Discussion of how to combine these modalities will also be summarized to advance the field of applied neurofeedback.

References

Busscher, B., Spinhoven, P., van Gerwen, L. J., & de Geus, E. J. C. (2013). Anxiety sensitivity moderates the relationship of changes in physiological arousal with flight anxiety during in vivo exposure therapy. *Behaviour Research and Therapy, 51*(2), 98–105. http://dx.doi.org/10.1016/j.brat.2012.10.009

Ehrlich, I., Humeau, Y., Grenier, F., Ciocchi, S., Herry, C., & Lüthi, A. (2009). Amygdala inhibitory circuits and the control of fear memory. *Neuron, 62*(6), 757–771. http://dx.doi.org/10.1016/j.neuron.2009.05.026

Feusner, J. D., Madsen, S., Moody, T. D., Bohon, C., Hembacher, E., Bookheimer, S. Y., & Bystritsky, A. (2012). Effects of cranial electrotherapy stimulation on resting state brain activity. *Brain and Behavior, 2*(3), 211–220. http://dx.doi.org/10.1002/brb3.45

Harper, M. L., Rasolkhani-Kalhorn, T., & Drozd, J. F. (2009). On the neural basis of EMDR therapy: Insights from qEEG studies. *Traumatology, 15*(2), 81–95. http://dx.doi.org/10.1177/1534765609338498

Lande, R. G., & Gragnani, C. (2013). Efficacy of cranial electric stimulation for the treatment of insomnia: A randomized pilot study. *Complementary Therapies in Medicine, 21*(1), 8–13. http://dx.doi.org/10.1016/j.ctim.2012.11.007

Pavlenko, V. B., Chernyi, S. V., & Goubkina, D. G. (2009). EEG correlates of anxiety and emotional stability in adult healthy subjects. *Neurophysiology, 41*(5), 337–345. http://dx.doi.org/10.1007/s11062-010-9111-2

Serin, A., Hageman, N. S., & Kade, E. (2018). The therapeutic effect of bilateral alternating stimulation tactile form technology on the stress response. *Journal of Biotechnology and Biomedical Science, 1*(2), 42–47. http://dx.doi.org/10.14302/issn.2576-6694.jbbs-18-1887

Multivariate Coherence Training for Developmental Trauma

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Developmental trauma is a major public health concern that has generated increased interest from researchers over the past few decades. The Adverse Childhood Experiences (ACE) study revealed correlational relationships between traumatic childhood experiences and an array of outcomes after several years, which included depression, substance abuse, domestic violence, suicide attempts, and various medical conditions (Felitti et al., 1998). Common domains of impairment observed by children exposed to developmental trauma are multifaceted, consisting of self-concept, attachment, behavioral regulation, affect regulation, dissociation, and biology (Cook et al., 2017). Neuroimaging studies of this population have revealed structural changes in the brain, such as reduced development of the hippocampus, amygdala, corpus callosum, and left neocortex (Teicher et al., 2003). Overall, there are a paucity of neurofeedback studies on developmental trauma. A small handful of projects have focused on power training (van der Kolk et al., 2016), some of which have been qEEG based (Huang-Storms, Bodenhamer-Davis, Davis, & Dunn, 2006).

We are conducting a study on participants with a history of developmental trauma who underwent neurofeedback training. We hypothesize that subjects who undergo neurofeedback training will show significantly decreased levels of mood and trauma-related symptoms compared to controls. Based on the findings of Armes and Coben (2017), we hypothesize that changes in connectivity will be related to success in neurofeedback and reduction of symptoms. Our study consists of 40 participants who were randomly assigned to a four-channel multivariate coherence training group or a control group who received an alternative treatment with no neurofeedback training. Dependent variables included the Beck Depression Inventory-II, Beck Anxiety Inventory, Trauma Symptom Inventory-II, as well as power and graph theory connectivity metrics based on qEEG findings. These measures were all administered at time 1 and time 2 with an intervening period of neurofeedback training. Preliminary findings show enhancements in coherence metrics are associated with decreased depression, anxiety, and trauma-related symptoms.

References

Armes, C. A., & Coben, R. (2017, September). Impact of developmental trauma on brain function and connectivity. Presented at the International Society of Neurofeedback and Research 25th Annual Conference, Foxwoods, CT.

Cook, A., Spinazzola, J., Ford, J., Lanktree, C., Blaustein, M., Cloitre, M., … van der Kolk, B. (2005). Complex trauma in children and adolescents. *Psychiatric Annals, 35*(5), 390–398.

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., ... Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine, 14*(4), 245–258. http://dx.doi.org/10.1016/S0749-3797(98)00017-8

Huang-Storms, L., Bodenhamer-Davis, E., Davis, R., & Dunn, J. (2006). QEEG-guided neurofeedback for children with histories

of abuse and neglect: Neurodevelopmental rationale and pilot study. *Journal of Neurotherapy, 10*(4), 3–16. http://dx.doi.org/10.1300/J184v10n04_02

Teicher, M. H., Andersen, S. L., Polcari, A., Anderson, C. M., Navalta, C. P., & Kim, D. M. (2003). The neurobiological consequences of early stress and childhood maltreatment. *Neuroscience & Biobehavioral Reviews, 27*(1), 33–44. http://dx.doi.org/10.1016/S0149-7634(03)00007-1

van der Kolk, B. A., Hodgdon, H., Gapen, M., Musicaro, R., Suvak, M. K., Hamlin, E., & Spinazzola, J. (2016). A randomized controlled study of neurofeedback for chronic PTSD. *PLoS ONE 11*(12), e0166752. http://dx.doi.org/10.1371/journal.pone.0166752

The Effect of Infraslow Frequency Neurofeedback on Quantitative Electroencephalogram and Autonomic Nervous System Function in Adults with Anxiety and Related Diseases

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Background. Over the last decade it has been observed in clinical practice that Infraslow Frequency (ISF) training shifts clients in physiological state during training. Peripheral body temperature, pupil size, and breathing rate are a few examples of autonomic nervous system (ANS) responses regularly observed during ISF neurofeedback training. ISF electroencephalographic (EEG) biofeedback focuses on the low energy signals produced by the brain. This includes frequencies of less than 0.1 Hz (Smith, Collura, Ferrera, & de Vries, 2014). Evidence suggests that these slow oscillations play a role in synchronizing faster activity and modulates cortical excitability (Bazhenov & Timofeev, 2006). The origins of these slow oscillations are not yet well understood but studies have indicated the involvement of the thalamus and other subcortical structures (Lörincz, Geall, Bao, Crunelli, & Hughes, 2009). The ANS is an important role player in maintaining sympathetic–parasympathetic and cardiovascular homeostasis. It includes vagal cholinergic and sympathetic noradrenergic nerves that supply the heart and sympathetic noradrenergic nerves that enmesh arterioles. Therefore, clinicians and researchers have long sought valid, noninvasive, quantitative means to identify patho-physiologically relevant abnormalities of these systems (Goldstein, Bentho, Park, & Sharabi, 2011). Heart Rate Variability (HRV) is one of the most well-known means of measurement. There is increasing research pointing to the clinical application of HRV in training and exercise due to its apparent result in strengthening sympathetic–parasympathetic balance (Peper, Harvey, Lin, Tylova, & Moss, 2007). Achieving an increased HRV while doing ISF training should be a good indicator of firstly reaching clients Optimum Frequency (OF) and secondly achieving a sympathetic–parasympathetic balance (Camp, Remus, Kalburgi, Porterfield, & Johnson, 2012; Collura, 2014). This study hypothesizes that ISF training has a measurable physiological effect on an individual by measuring certain autonomic functions; namely, HRV, muscle tension, skin temperature, skin conductance, heart rate, respiration rate, and blood pressure. Also, to demonstrate how ISF training impacts the resting state EEG.

Methods. Thirty adults between the ages of 18 and 55 with primarily anxiety will receive a quantitative electroencephalogram (qEEG) to get a baseline before training. The participants will then receive ISF neurofeedback training for 10 sessions while continuous monitoring of ANS changes will be done to determine if there are measurable changes. After 10 sessions we will repeat a qEEG to determine what changes occurred. The same process will be completed for a control group. The control group will receive one-channel power training where Theta and Hibeta activity will be inhibited at 3–7 Hz and 22–30 Hz respectively and Lobeta 12–15 Hz activity enhanced at the C4 location on the head.

Anticipated results. Preliminary results and a pilot study conducted show significant changes that have been observed in participants trained in ISF neurofeedback, both in the activation patterns when looking at the qEEG and the autonomic functions that were measured. No significant changes have been seen thus far in the control group.

Conclusion. The study will possibly demonstrate that autonomic functions are affected by ISF neurofeedback training and that changes occur in the resting state EEG of participants trained.

References

Bazhenov, M., & Timofeev, I. (2006). Thalamocortical oscilations. Scholarpedia, 1(6), 1319. http://dx.doi.org/10.4249/scholarpedia.1319

Camp, R. M., Remus, J. L., Kalburgi, S. N., Porterfield, V. M., & Johnson, J. D. (2012). Fear conditioning can contribute to behavioral changes observed in a repeated stress model. *Behavioural Brain Research, 233*(2), 536–544. http://dx.doi.org/10.1016/j.bbr.2012.05.040

Collura, T. F. (2014). *Technical foundations of neurofeedback*. New York, NY: Routlege/Taylor & Francis.

Goldstein, D. S., Bentho, O., Park, M. Y., & Sharabi, Y. (2011). Low-frequency power of heart rate variability is not a measure of cardiac sympathetic tone but may be a measure of

modulation of cardiac autonomic outflows by baroreflexes. *Experimental Physiology, 96*(12), 1255–1261. http://dx.doi.org/10.1113/expphysiol.2010.056259

Hallman, D., & Lyskov, E. (2012). Autonomic regulation in musculoskeletal pain. Retrieved on July 15, 2017, from Intech Open Science: https://www.intechopen.com/books/pain-in-perspective/autonomic-regulation-in-musculosceletal-pain

Lőrincz, M., Geall, F., Bao, Y., Crunelli, V., & Hughes, S. W. (2009). ATP-dependent infra-slow (< 0.1 Hz) oscillations in thalamic networks. *PLoS One, 4*(2), e4447. http://dx.doi.org/10.1371/journal.pone.0004447

Peper E, Harvey, R., Lin, I., Tylova, H., & Moss, D. (2007). Is there more to blood volume pulse than heart rate variability, respiratory sinus arrhythmia, and cardiorespiratory synchrony? *Biofeedback, 35*(2), 54–61.

Smith, M. L., Collura, T. F., Ferrera, J., & de Vries, J. (2014). Infra-slow fluctuation training in clinical practice: A technical history. *NeuroRegulation, 1*(2), 187–207. http://dx.doi.org/10.15540/nr.1.2.187

The Human Compassion Circuit

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Compassion has been one of the most cherished, acclaimed, practiced, and pursued of human virtues for literally thousands of years—and a foremost part of nearly all organized religions and spiritual quests. Despite its lofty and celebrated status, tragically there are far too many examples in human history of “the far enemy” of Compassion, Cruelty. For a species so enamored with this cherished human virtue, how are we so able to engage in cruel acts? Perhaps the answer, in part, to this critical social question lies in our neurological makeup; perhaps there are specialized structures in our brains that are hardwired for the experience of Compassion, and structures that are similarly hardwired for our expression of Cruelty.

This presentation reports on a recent literature review, analysis, and integration by the author of a growing body of compassion research, detailed in his chapter entitled, “The Brain that Longs to Care for Others: The Current Neuroscience of Compassion” in his soon-to-be-released academic textbook *The Neuroscience of Empathy, Compassion, and Self-Compassion* (Elsevier/ Academic Press, June 2018). This review suggests that such hardwired circuits do indeed exist in each of our brains. A follow-up research investigation by the author was designed to begin the answer to the question above and to more clearly and with greater certainty identify these circuits.

This presentation will thus clarify the temporal and spatial characteristics of a neurological circuit in the human brain identified with the experience of Compassion. The hypothesis of the reported research investigation is that participants will respond to careworthy and to blameworthy compassion scenarios with a specified differential, sequential (temporal) spatial circuit involving (1) an affective amygdala (AMG), anterior insular cortex (AIC), and anterior cingulate cortex (ACC) sub-circuit; (2) motor intentional mirroring structures in the Mirror Neuron System of the premotor cortex (PMC) and inferior parietal lobe (IPL); (3) cognitive regulatory subcircuits in the dorsolateral prefrontal cortex (dlPFC), ventrolateral PFC (vlPFC), dorsomedial PFC (dmPFC), and posterior temporal cortex (PTC); and then (4) simultaneous with the PFC subcircuits above, self–other differentiation Theory of Mind (ToM) subcircuits in the bilateral temporal parietal junction (TPJ), precuneus (PCun), and dmPFC. These temporal and spatial pathways are identified by continuous electroencephalograph (EEG) recordings and during subsequent power spectral and LORETA neuroimaging analyses following the participant’s experiencing of specific, visually-presented compassion scenarios. Exciting Neurotherapy, TMS, Meditation, and Psychotherapy protocols are explored as ways of increasing Compassion in human beings, perhaps even as alternatives to one of the cruelest of compassion "far enemies," torture.

References

Buhle, J. T., Silvers, J. A., Wager, T. D., Lopez, R., Onyemekwu, C., Kober, H., … Ochsner, K. N. (2014). Cognitive reappraisal of emotion: A meta-analysis of human neuroimaging studies. *Cerebral Cortex, 24*(11), 2981–2990. http://dx.doi.org/10.1093/cercor/bht154

de Waal, F. (2009). *The age of empathy*. New York, NY: Three Rivers Press.

Fehse, K., Silveira, S., Elvers, K., & Blautzik, J. (2015). Compassion, guilt and innocence: An fMRI study of responses to victims who are responsible for their fate. *Social Neuroscience, 10*(3), 243–252. http://dx.doi.org/10.1080/17470919.2014.980587

Gallese, V., Keysers, C., & Rizzolatti, G. (2004). A unifying view of the basis of social cognition. *Trends in Cognitive Sciences, 8*(9), 396–403. http://dx.doi.org/10.1016/j.tics.2004.07.002

Goetz, J. L., Keltner, D., & Simon-Thomas, E. (2010). Compassion: An evolutionary analysis and empirical review. *Psychological Bulletin, 136*(3), 351–374. http://dx.doi.org/ 10.1037/a0018807

Kédia, G., Berthoz, S., Wessa, M., Hilton, D., & Martinot, J.-L. (2008). An agent harms a victim: A functional magnetic resonance imaging study on specific moral emotions. *Journal of Cognitive Neuroscience, 20*(10), 1788–1798. http://dx.doi.org/10.1162/jocn.2008.20070

Lindquist, K. A., Wager, T. D., Kober, H., Bliss-Moreau, E., & Barrett, L. F. (2012). The brain basis of emotion: A meta-analytic review. *Behavioral and Brain Sciences, 35*(3), 121–143. http://dx.doi.org/10.1017/S0140525X11000446

Lutz, A., Greischar, L. L., Rawlings, N. B., Ricard, M., & Davidson, R. J. (2004). Long-term meditators self-induce high-amplitude gamma synchrony during mental practice. *Proceedings of the National Academy of Sciences, 101*(46), 16369–16373. http://dx.doi.org/10.1073/pnas.0407401101

Ochsner, K. N., Ray, R. D., Cooper, J. C., Robertson, E. R., Chopra, S. Gabrieli, J. D. E, & Gross, J. J. (2004). For better or for worse: Neural systems supporting the cognitive down- and up-regulation of negative emotion. *NeuroImage, 23*(2), 483–499. http://dx.doi.org/10.1016/j.neuroimage.2004.06.030

Ochsner, K. N., Ray, R. R., Hughes, B., McRae, K., Cooper, J. C., Weber, J., … Gross, J. J. (2009). Bottom-up and top-down processes in emotion generation: Common and distinct neural mechanisms. *Psychological Science, 20*(11), 1322–1331. http://dx.doi.org/10.1111/j.1467-9280.2009.02459.x

Phan, K. L., Wager, T., Taylor, S. F., & Liberzon, I. (2002). Functional neuroanatomy of emotion: A meta-analysis of emotion activation studies in PET and fMRI. *NeuroImage, 16*(2), 331–348. http://dx.doi.org/10.1006/nimg.2002.1087

Porges, S. W. (2003). The Polyvagal Theory: Phylogenetic contributions to social behavior. *Physiology and Behavior, 79*, 503–513. http://dx.doi.org/10.1016/S0031-9384(03)00156-2

Premack, D., & Woodruff, G. (1978). Does the chimpanzee have a theory of mind? *Behavioral and Brain Sciences, 1*(4), 515–526. http://dx.doi.org/10.1017/S0140525X00076512

Singer, T. (2006). The neuronal basis and ontogeny of empathy and mind reading: Review of literature and implications for future research. *Neuroscience and Behavioral Reviews, 30*(6), 855–863. http://dx.doi.org/10.1016 /j.neubiorev.2006.06.011

Singer, T., Critchley, H. D., & Preuschoff, K. (2009). A common role of insula in feelings, empathy and uncertainty. *Trends in Cognitive Sciences, 13*(8), 334–340. http://dx.doi.org/10.1016 /j.tics.2009.05.001

Neurofeedback: An Effective Treatment for Symptoms of Posttraumatic Stress Disorder in Veterans

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POSTERS PRESENTATIONS

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

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

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

psychological questionnaires and EEG, which included the F3 alpha, F4 alpha, and A1 score.

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

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

References

Baehr, E., Rosenfeld, J. P., & Baehr, R. (2001). Clinical use of an alpha asymmetry neurofeedback protocol in the treatment of mood disorders: Follow-up study one to five years post therapy. *Journal of Neurotherapy, 4*(4), 11–18. http://dx.doi.org/10.1300/J184v04n04_03

Cheon, E.-J., Koo, B.-H., & Choi, J.-H. (2016). The efficacy of neurofeedback in patients with major depressive disorder: An open labeled prospective study. *Applied Psychophysiology and Biofeedback, 41*(1), 103–110. http://dx.doi.org/10.1007/s10484-015-9315-8

Choi, S. W., Chi, S. E., Chung, S. Y., Kim, J. W., Ahn, C. Y., & Kim, H. T. (2011). Is alpha wave neurofeedback effective with randomized clinical trials in depression? A pilot study. *Neuropsychobiology, 63*(1), 43–51. http://dx.doi.org/10.1159/000322290

Davidson, R. J. (1998). Anterior electrophysiological asymmetries, emotion, and depression: Conceptual and methodological conundrums. *Psychophysiology, 35*(5), 607–614.

Wang, S.-Y., Lin, I.-M., Peper, E., Chen, Y.-T., Tang, T.-C., Yeh, Y.-C., ... Chu, C.-C. (2016). The efficacy of neurofeedback among patients with major depressive disorder: Preliminary study. *NeuroRegulation, 3*(3), 127–134. http://dx.doi.org/10.15540/nr.3.3.127

A Real-time fMRI Neurofeedback for Mild to Severe Depression Compared to Frontal Alpha-asymmetry Neurofeedback and Cognitive–Behavioral Therapy

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

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

Methods. Thirty subjects (10 males, 20 females, aged 20–50, mean age of 33) were recruited and randomly assigned to experimental or one of two control groups. Participants of experimental group received eight weekly sessions of fMRI neurofeedback targeted bilaterally to the area within medial prefrontal cortex. During each session blocks of enhancing and suppressing the response of the target area were alternating. Subjects continuously received visual feedback reflecting percent of signal change within the region of interest (ROI). The source signal was recorded using Philips Ingenia 3T MR scanner with an EPI sequence, TR = 1000 ms. Temporal dynamics of the signal from ROI was captured from the IViewBOLD graphs and presented on the screen as a yellow circle, the diameter and brightness of which depended on the signal values. Offline fMRI analysis was performed using SPM 12 software. Concurrent EEG was recorded with a 32-channel MR-compatible BrainAmp system, corrected for MR, cardiac, and ocular artifacts and processed in

EEGLab software. First control group patients received sixteen 25-min sessions of frontal alpha-asymmetry neurofeedback. Participants from the second control group were treated with eight individual and eight group sessions of CBT. Each subject underwent psychiatric examination (MADRS), psychological assessment (BDI, SDS [Zung Self-Rating Depression Scale], HADS), and EEG-fMRI recording at rest and during performing an emotionally salient task at start, at middle, and at the end of the course.

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

Conclusions. FMRI-based neurofeedback holds a promise for a targeted regulation of emotional circuits and can be considered as a potentially clinically efficacious technique of self-regulation in mood disorders. However, cost-benefit ratio remains a problem for this application. Studies of EEG-fMRI correlates during the real-time fMRI-neurofeedback sessions may be instrumental for enhancing EEG neurofeedback treatment protocols.

References

Hamilton, J. P., Glover, G. H., Bagarinao, E., Chang, C., Mackey, S., Sacchet, M. D., & Gotlib, I. H. (2016). Effects of salience-network-node neurofeedback training on affective biases in major depressive disorder. *Psychiatry Research, 249*, 91–96. http://dx.doi.org/10.1016/j.pscychresns.2016.01.016

Johnston, S., Linden, D. E. J., Healy, D., Goebel, R., Habes, I., & Boehm, S. G. (2011). Upregulation of emotion areas through neurofeedback with a focus on positive mood. *Cognitive Affective, & Behavioral Neuroscience, 11*(1), 44–51. http://dx.doi.org/10.3758/s13415-010-0010-1

Linden, D. E. J., Habes, I., Johnston, S. J., Linden, S., Tatineni, R., Subramanian, L., … Goebel, R. (2012). Real-time self-regulation of emotion networks in patients with depression. *PLoS One, 7*(6), 38115. http://dx.doi.org/10.1371 /journal.pone.0038115

Young, K. D., Siegle, G. J., Zotev, V., Phillips, R., Misaki, M., Yuan, H., … Bodurka, J. (2017). Randomized clinical trial of real-time fMRI amygdala neurofeedback for major depressive disorder: Effects on symptoms and autobiographical memory recall. *The American Journal of Psychiatry, 174*(8), 748–755. http://dx.doi.org/10.1176 /appi.ajp.2017.16060637

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

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Bilateral alternating stimulation in tactile form (BLAST) technology has been found to significantly reduce subjective distress and physiological body sensations in response to thinking about a stressful event (Serin, Hageman & Kade, 2018). TouchPoints are noninvasive devices that deliver BLAST and are believed to reduce sympathetic nervous system arousal associated with anxiety (Busscher, Spinhoven, van Gerwen, & de Geus, 2013) by depotentiating amygdala activity (Harper, Rasolkhani-Kalhorn, & Drozd, 2009) responsible for activating the body's stress response (Ehrlich et al., 2009). Beta EEG rhythm has been found to correlate to high situational and personal anxiety (Pavlenko, Chernyi, & Goubkina, 2009). The purpose of this study was to utilize quantitative electroencephalography (qEEG) recordings to identify significant changes in electrical brain activity upon thinking of a stressful event and subsequently upon the delivery of BLAST. It was hypothesized that upon thinking of a stressful event beta activity would increase and subsequently reduce significantly upon delivery of BLAST.

Methods. A total of 21 participants (9 male, 12 female), ages 7 to 63 (*M* age = 27.8; *SD* = 16.5), participated in the study and were recruited through the Serin Center. The sample consisted of 14 clinical participants with heterogeneous diagnoses of anxiety, major depressive disorder, and attention-deficit/hyperactivity disorder. The remaining sample consisted of nonclinical participants. QEEG data was collected at the Serin Center's locations in Peoria and Scottsdale, Arizona. Data was collected utilizing a NeuroField Q20 amplifier and was stored using NeuroGuide by Applied Neuroscience Inc. Participants underwent a 5-min baseline recording followed by an instruction to think about a stressful event. Participants were then asked to hold TouchPoints devices. 19-channel qEEG recordings were taken while thinking about the stressful event, during the delivery of BLAST (holding the TouchPoints), and then a baseline was taken again upon removal of the TouchPoints. Paired *t*-test analysis was conducted before and after BLAST with NeuroGuide's Neurostat software.

Results. Preliminary EEG recordings comparing the stress condition to the TouchPoints condition exhibited significantly reduced activity in frontal Theta, specifically in 5 Hz at Fp2 and F4 sites and reduced activity in Beta 1 at 12–14 Hz in the frontal channel locations (Fp1, Fp2, Fp3, Fz, F4). Significant right frontal decreases are shown in Beta 2 at 16–18 Hz, Beta 3 bands at 19 Hz and 23 Hz, and Gamma 1 at 30–35 Hz with activity decreasing along the midline also in Beta 3 at 19 Hz and 23 Hz and in Gamma 1 at 30 Hz.

Conclusion. The significant reduction in beta activity provides preliminary evidence that BLAST technology may have a therapeutic effect on reducing subcortical activity associated with anxiety and stress. Our results are consistent with previous studies (Pavlenko et al., 2009) suggesting beta wave activity is correlated with increased levels of anxiety. This preliminary data implicates the potential efficacy of BLAST as a mediator of SNS arousal and stress through beta-activity reduction in both clinical and nonclinical samples. Follow-up research is required utilizing a comparison control group with a larger sample to assess for qEEG differences in brain activity.

References

Busscher, B., Spinhoven, P., van Gerwen, L. J., & de Geus, E. J. C. (2013). Anxiety sensitivity moderates the relationship of changes in physiological arousal with flight anxiety during in vivo exposure therapy. *Behaviour Research and Therapy, 51*(2), 98–105. http://dx.doi.org/10.1016/j.brat.2012.10.009

Ehrlich, I., Humeau, Y., Grenier, F., Ciocchi, S., Herry, C., & Lüthi, A. (2009). Amygdala inhibitory circuits and the control of fear memory. *Neuron, 62*(6), 757–771. http://dx.doi.org/10.1016/j.neuron.2009.05.026

Harper, M. L., Rasolkhani-Kalhorn, T., & Drozd, J. F. (2009). On the neural basis of EMDR therapy: Insights from qEEG studies. *Traumatology, 15*(2), 81–95. http://dx.doi.org/10.1177/1534765609338498

Pavlenko, V. B., Chernyi, S. V., & Goubkina, D. G. (2009). EEG correlates of anxiety and emotional stability in adult healthy subjects. *Neurophysiology, 41*(5), 337–345. http://dx.doi.org/10.1007/s11062-010-9111-2

Serin, A., Hageman, N. S., & Kade, E. (2018). The therapeutic effect of bilateral alternating stimulation tactile form technology on the stress response. *Journal of Biotechnology and Biomedical Science, 1*(2), 42–47. http://dx.doi.org/10.14302/issn.2576-6694.jbbs-18-1887

Event-related Potential Study of Illusory Figure Processing Deficits in Children with Autism Spectrum Disorder

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

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

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

Results. Children with ASD did not differ from typical children in reaction time (RT), but they committed more errors (12.9% vs. 2.2 %, $F = 14.9$, $p < .001$) and did not show normative posterror RT slowing ($F = 27.6$, $p < .001$). The error-related negativity was lower in ASD ($F = 8.5$, $p = .004$). The early ERPs (P100 and N100) to nontarget stimuli were of higher amplitude and delayed in the ASD group (ps < 0.05). The late ERPs (P3a and P3b) to nontarget stimuli were prolonged in ASD without amplitude differences, though P3a was delayed as well to targets in the ASD (458 vs. 426 ms, $F = 4.9$, $p = .03$).

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

References

Baruth, J. M., Casanova, M., Sears, L., & Sokhadze, E. (2010). Early-stage visual processing abnormalities in high-functioning autism spectrum disorder (ASD). *Translational Neuroscience, 1*(2), 177–187. http://dx.doi.org/10.2478 /v10134-010-0024-9

Bomba, M. D., & Pang, E. W. (2004). Cortical auditory evoked potentials in autism: A review. *International Journal of Psychophysiology, 53*(3), 161–169. http://dx.doi.org/10.1016 /j.ijpsycho.2004.04.001

Cui, T., Wang, P. P., Liu, S., & Zhang, X. (2017). P300 amplitude and latency in autism spectrum disorder: A meta-analysis. *European Child & Adolescent Psychiatry, 26*(2),177–190. http://dx.doi.org/10.1007/s00787-016-0880-z

Kanizsa, G. (1976). Subjective contours. *Scientific American, 234*(4), 48–52.

Kemner, C., van der Gaag, R. J., Verbaten, M., & van Engeland, H. (1999). ERP differences among subtypes of pervasive developmental disorders. *Biological Psychiatry, 46*(6), 781–789. http://dx.doi.org/10.1016/S0006-3223(99)00003-7

Sokhadze, E., Baruth, J., El-Baz, A., Horrell, T., Sokhadze, G., Carroll, T., … Casanova, M. F. (2010). Impaired error monitoring and correction function in autism. *Journal of Neurotherapy, 14(2)*, 79–95. http://dx.doi.org/10.1080 /10874201003771561

Sokhadze, E. M., Baruth, J. M., Sears, L., Sokhadze, G. E., El-Baz, A. S., Williams, E. L., … Casanova, M. F. (2012). Event-related potential study of attention regulation during illusory figure categorization task in ADHD, autism spectrum disorder, and typical children. *Journal of Neurotherapy, 16*(1), 12–31. http://dx.doi.org/10.1080/10874208.2012.650119

Sokhadze, E., Baruth, J., Tasman, A., Sears, L., Mathai, G., El-Baz, A. & Casanova, M. F. (2009). Event-related potential study of novelty processing abnormalities in autism. *Applied Psychophysiology and Biofeedback, 34*(1), 37–51. https://doi.org/10.1007/s10484-009-9074-5

Townsend, J., Westerfield, M., Leaver, E., Makeig, S., Jung, T.-P., Pierce, K., & Courchesne, E. (2001). Event-related brain response abnormalities in autism: Evidence for impaired cerebello-frontal spatial attention networks. *Cognitive Brain Research, 11*(1), 127–145. http://dx.doi.org/10.1016/S0926-6410(00)00072-0

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

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Background. Autism is defined as a spectrum of behavioral disorders that have in common impairments in social interaction and communication skills, language deficits, and a restricted repertoire of interests and stereotyped activities. There are several theoretical models of the neuropathology of autism spectrum disorders (ASD), and one of them suggests the presence of an excessive cortical excitation/inhibition (E/I) ratio (Casanova, Buxhoeveden, & Gomez, 2003; Rubinstein & Merzernich, 2003; Uzunova, Pallanti, & Hollander, 2016) that affects functional connectivity. This model explains atypical event-related potential (ERP) and evoked and induced gamma oscillations observed in ASD during task performance. Repetitive transcranial magnetic stimulation (rTMS), especially using low-frequency inhibitory stimulation, can be considered as a method of modulating the E/I bias.

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

Methods. TMS consisted of trains of 1.0 Hz pulses applied over dorsolateral prefrontal cortex. The experimental task was a three-stimulus visual oddball with illusory Kanizsa figures. Behavioral response

variables included reaction time and error rate along with EEG indices such as ERP and evoked and induced gamma oscillations. One hundred and twelve patients completed the assigned number of rTMS sessions.

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

Conclusions. Our results suggest that rTMS, particularly after 18 sessions, facilitates cognitive control, attention and target stimuli recognition by improving discrimination between task-relevant and task-irrelevant illusory figures in an oddball test. Improvement in executive functions and behavioral symptoms of autism further suggests that TMS has the potential to target core features of ASD. The results of this dosage-response study could serve as important prerequisites that could inform the planning of a blinded randomized clinical trial. Among potential implications of the study should be considered potential of combining rTMS with neurofeedback training (Sokhadze et al., 2014) aimed at reinforcement of neuromodulation effects using operant conditioning in similar manner as reported by our group earlier.

References

Casanova, M. F., Buxhoeveden, D., & Gomez, J. (2003). Disruption in the inhibitory architecture of the cell minicolumn: Implications for autism. *The Neuroscientist, 9*(6), 496–507. http://dx.doi.org/10.1177/1073858403253552

Rubenstein, J. L. R., & Merzenich, M. M. (2003). Model of autism: Increased ratio of excitation/inhibition in key neural systems. *Genes, Brain and Behavior, 2*(5), 255–267. http://dx.doi.org /10.1034/j.1601-183X.2003.00037.x

Sokhadze, E., Baruth, J., Tasman, A., Mansoor, M., Ramaswamy, R., Sears, L., … Casanova, M. F. (2010). Low-frequency repetitive transcranial magnetic stimulation (rTMS) affects event-related potential measures of novelty processing in autism. *Applied Psychophysiology and Biofeedback, 35*(2), 147–161. http://dx.doi.org/10.1007/s10484-009-9121-2

Sokhadze, E. M., El-Baz, A., Baruth, J., Mathai, G., Sears, L., & Casanova, M. F. (2009). Effects of low-frequency repetitive transcranial magnetic stimulation (rTMS) on gamma frequency oscillations and event-related potentials during processing of illusory figures in autism. *Journal of Autism and Developmental Disorders, 39*(4), 619–634. http://dx.doi.org /10.1007/s10803-008-0662-7

Sokhadze, E. M., El-Baz, A. S., Tasman, A., Sears, L. L., Wang, Y., Lamina, E. V., & Casanova, M. F. (2014). Neuromodulation integrating rTMS and neurofeedback for the treatment of autism spectrum disorder: An exploratory study. *Applied Psychophysiology and Biofeedback, 39*(3–4), 237–257. http://dx.doi.org/10.1007/s10484-014-9264-7

Uzunova, G., Pallanti, S., & Hollander, E. (2016). Excitatory/inhibitory imbalance in autism spectrum disorders: Implications for interventions and therapeutics. *The World Journal Biological Psychiatry, 17*(3), 174–186. http://dx.doi.org/10.3109/15622975.2015.1085597

Using Neurofeedback to Lower Anxiety Symptoms: A Follow-up Study

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Introduction. Anxiety represents one of the most commonly diagnosed mental illnesses in the United States, affecting approximately 18% of the population annually (NIMH, 2017). This retrospective study intended to assess whether qEEG-guided amplitude neurofeedback (NF) is viable in symptom reduction of anxiety. This presentation updates a previously presented and published pilot study on treating anxiety symptoms with neurofeedback, based on data from 2014 to 2015 (Dreis et al., 2015). The pilot study involved a retrospective assessment of the efficacy of qEEG-guided one-channel neurofeedback for reduction of anxiety symptoms. The treatment was provided through an urban on-campus community counseling center operated by a university counseling department for the training of master and doctoral level students in neurofeedback. This updated retrospective study follows the same model as the pilot study with increased study size, based on data from 2014 to 2018.

Methods. From 2014 to 2018, 76 total clients were assessed and treated for anxiety using neurofeedback. Retrospectively, 34 clients met inclusion criteria for data analysis. Inclusion required that primary symptoms be related to anxiety, that the client is naive to neurofeedback and completion of pre- and postassessments including qEEG and symptom checklists. It is projected that an additional 12 clients may be included in the final analysis to be concluded by August 2018. Clients of the counseling center were from the local community. Demographics include age ranges from 11 to 61 (*M* =

34.76, *SD* = 15.15), 18 male and 16 female; 17 identified as Caucasian, 13 identified as Hispanic/Latino, and 3 Caucasian/Hispanic ethnicity; one declined to identify their ethnicity. Pre- and postassessments included qEEG, the Zung Self-Rating Anxiety Scale, Screen for Child Anxiety-Related Disorders (SCARED), and the Achenbach System of Empirically Based Assessment (ASEBA). Clients received between 20 to 30 minutes of qEEG-guided NF treatment sessions, twice a week. The range of attended session was 4–19 (*M* = 12.14, *SD* = 3.24).

Results. On the Zung Anxiety Scale mean scores were reduced from 45.5 to 38.1 with $t(32) = 7.20$, $p < .0001$, d = 1.22. SCARED mean scores were reduced from 37.22 to 23.27 with $t(2) = 4.80$, $p < .041$, $d = 2.77$. ASEBA mean scores were reduced on the Anxious-Depressed scale from 68.61 to 64.19 with $t(35) = 3.49$, $p < .001$, $d = 0.58$, on the Anxiety Disorder (DSM) scale from 65.14 to 61.97 with $t(35) = 2.57$, $p < .014$, $d = 0.43$, and on the Total Problems scale (average of eight core scales) from 59.53 to 55.19 with $t(35) = 3.85$, $p < .0001$, $d = 0.64$. Limitations and directions for future research will be discussed along with details of qEEG markers and related treatment protocols.

References

Dreis, S. M., Gouger, A. M., Perez, E. G., Russo, G. M., Fitzsimmons, M. A., & Jones, M. S. (2015). Using neurofeedback to lower anxiety symptoms using individualized qEEG protocols: A pilot study. *NeuroRegulation*, *2*(3), 137–148. http://dx.doi.org/10.15540 /nr.2.3.137

Lu, Y., Wang, C., Su, L., Ma, Z., Li, S., & Fan, Y. (2017). Effects of neurofeedback on panic disorder patients' anxiety. *NeuroQuantology*, *15*(3). http://dx.doi.org/10.14704 /nq.2017.15.3.1083

Mennella, R., Patron, E., & Palomba, D. (2017). Frontal alpha asymmetry neurofeedback for the reduction of negative affect and anxiety. *Behaviour Research and Therapy*, *92*, 32–40. http://dx.doi.org/10.1016/j.brat.2017.02.002

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