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Contents

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Editorial – Volume 2, Number 2: Status Update

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As we go to press with this second issue of Volume 2, *NeuroRegulation* celebrates its one-year publication anniversary! As such, we would like to take this opportunity to update you on the status of the official journal of the International Society for Neurofeedback and Research (ISNR).

ISNR members should be aware that *NeuroRegulation* is better considered an extension, or renaming, of the prior Journal of Neurotherapy (JNT); which published a total of 17 volumes dating back to 1995. Therefore, while *NeuroRegulation* may be midway into its second volume, it has in essence, close to a 20-year history. As was discussed in the inaugural issue (Clarke, 2014), ISNR made the decision to move to an electronic Open Access (OA) format to make neurotherapy- and psychophysiology-related literature more accessible to a much wider audience (consumers and researchers alike), improve the publication speed, and keep pace with growing publishing trends in all academic fields. However, had ISNR moved to an OA format with the JNT publisher at the time, it would have required substantial costs to authors; rather, transitioning to an independent publishing platform facilitated an OA model with no author processing charges. We are pleased to report—in the time frame of 12 months and six issues—we are well on the way to meeting these goals. The objective of expedited publication timelines is being met with current averages of 20 days from submission to review and 60 days from submission to publication.

Evidence of attaining the accessibility goal comes from comparing times viewed reports between the current to prior journals. The *NeuroRegulation* times viewed/downloaded reports, in comparison to JNT most viewed articles, when adjusted for an annualized comparison, reveal that articles are being viewed at a rate more than double that of the JNT articles; with views of abstracts twice that of articles. Moreover, the JNT most viewed article (Hammond, 2011) has been accessed 4,345 times in the 44 months since publication; yet, the *NeuroRegulation* abstract of Montopoli et al. (2015) has been viewed 4,799 times in only 86 *days*. Clearly, then, this journal with the electronic OA format, with all content accessible via Internet search engines, is being seen with greater frequency and reaching a wider audience than the prior subscription-model print journal.

Given the growing expectation of European research venues that scientific works be published in OA outlets, standards for OA journals are coming from that region. In an April press release, Science Europe (2015), a Brussels-based society made up of 50 public research organizations from 27 countries to promote their collective interests, announced their establishment of four essential principles for OA journals. The aim of these OA journal standards is to ensure technical and scholarly quality in all fields, inclusive of science and social science. The standards are as follows:

1. Be indexed in a standard database such as Directory of Open Access Journals (DOAJ), Thomson-Reuter's Web of Science (TR-WoS), or PubMed.
2. Publish under an open license (i.e., Creative Commons) such that the author holds the copyright with no restrictions.
3. Maintain sustainable archiving of content with a persistent address where the full publication can be accessed.
4. Publish the full text, metadata, citations, and OA status in machine-readable format using open standards.

We are pleased to report that *NeuroRegulation* meets *all* criteria. With regard to indexing, earlier this year the journal was indexed in DOAJ, currently have applications pending with both TR-WoS and PsychINFO, and have plans to apply for PubMed indexing as soon as eligible, projected by the end of this year.

We are invested in establishing *NeuroRegulation* as a premier publication in the field of applied

neuroscience. Adhering to high standards for OA publishing and sound science will move us forward to that end. We aim to attract manuscripts from a wide spectrum of neuromodulatory topics and psychophysiology focus, which will position this journal to be an important global scholarly outlet for the neuroscience community.

We are appreciative of the authors who submitted their work to this issue and welcome future submissions to the journal. We invite you to join us in our efforts by contributing research, reviews, case studies, or theoretical papers. Submissions are accepted on an ongoing basis throughout the year; however, deadlines for inclusion in specific issues are posted as Announcements on the home page of the journal website.

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References

Clarke, A. R. (2014). Editorial – Volume 1, Number 1. *NeuroRegulation, 1*(1), 2–3. http://dx.doi.org/10.15540/nr.1.1.2

Hammond, D. C. (2011). What is neurofeedback: An update. *Journal of Neurotherapy, 15*(3), 305–336. http://dx.doi.org/10.1080/10874208.2011.623090

Science Europe. (2015). New Science Europe Principles on Open Access Publisher Services [Press release]. Retrieved from http://www.scienceeurope.org/uploads/PressReleases/270415_Open_Access_New_Principles.pdf

Montopoli, M., Stevens, L., 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

NeuroRegulation

Editorial Perspective: Defining Neurofeedback and Its Functional Processes

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Abstract

Neurofeedback is gaining widespread attention across clinical and research domains. As our knowledge of the brain and its enigmatic mechanisms increase, so does the interest in harnessing these mechanisms to promote improved mental processes and reduce symptomatic issues. Neuroscience advances and neurofeedback will continue to evolve into a primary focus for learning, performance, and reduction of symptoms in psychopathology. Likewise, electroencephalographic (EEG) and source localization techniques will improve our understanding and identification of biomarker EEG patterns to better identify and ultimately classify specific patterns associated with psychological and neurological syndromes. As technology and production of devices become more prevalent, there is a growing need to define the parameters used in neurofeedback, as well as to classify the processes into specific or nonspecific factors to avoid further confounds and problems across disciplines.

Keywords: neurofeedback; operant conditioning; operant learning; self-regulation; neuroplasticity; neural efficiency; neuromodulation

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Introduction

Over the course of the past 50 years, and more recently, neurofeedback has been gaining interest and popularity in the public eye and across disciplines devoted to human mental wellness and performance. The literature is replete with published reports describing the processes involved with neurofeedback and results of empirical studies using this procedure to treat psychological syndromes or functionality in normative groups. However, a clear operant definition for neurofeedback has been elusive. Thus, the term *neurofeedback* has been used widely in recent years in studies that target changes measured through techniques including electroencephalographic (EEG), current source density (CSD), functional magnetic resonance imaging (fMRI), magnetoencephalography (MEG), functional near-infrared spectroscopy (fNRIS) and others (Hammond, 2011; Thibault, Lifshitz, Birbaumer, & Raz, 2015). Likewise, there have been countless devices developed for home use or entertainment that have adopted the term. However, accounts of the learning processes and biological mechanisms underlying neurofeedback are sparse. This is the impetus for this editorial perspective; as methods for operant learning through neurofeedback must be discussed, refined, and adopted into a rational format to further successful use of this method across research settings, peak performance, and mental health disciplines. This paper is not an attempt to validate neurofeedback as a method to treat psychiatric disorders; rather, it is a summary elucidating the mechanisms and procedures important to neurofeedback and learning in general.

Neurofeedback

Neurofeedback (EEG biofeedback, neurotherapy, neuroregulation) is a self-regulation technique that utilizes a brain computer interface (BCI) to influence

the processes of neural plasticity and neural efficiency. Neurofeedback is accomplished by providing the individual with feedback about the electrical activity of the brain within a specific frequency range at a specific target on the scalp. Neurofeedback has traditionally been accomplished by placing one or more sensors on the head to measure the EEG at a particular site, in a specific frequency range, so that auditory and/or visual stimuli are provided contingent on EEG activity reaching a target value. Through feedback human beings, animals, and even single neurons can learn to change and regulate EEG activity (or firing patterns thereof).

Neurofeedback is not to be confused with neurostimulation, transcranial magnetic stimulation, or any methodology that introduces a signal or pulse into the brain. These methods cannot be considered operant conditioning and therefore cannot be considered neurofeedback, because the stimuli involved are not contingent on any defined brain activity.

Neurofeedback may be defined as the presentation of a stimulus change contingent on brain activity that meets a target-specific defined criterion. At this time, three types of EEG neurofeedback can be described. Neurofeedback of the operant conditioning type (NF_{OC}) requires EEG activity to reach a fixed criterion before feedback is delivered. In other cases the response threshold or criterion for feedback varies dynamically based on numerous moment-by-moment calculations of the antecedent EEG activity. Due to lack of specificity and details, such techniques are better classified as neurofeedback of an undifferentiated type (NF_{UT}). Finally, there are the commercially available devices that a user takes home and wears, to improve subjective experiences, typically without clear targets or known functional correlates (e.g., focus, relaxation, stress reduction). These methods can be classified as neurofeedback of the entertainment type (NF_{ET}).

Operant Conditioning

Operant conditioning (OC) describes how we develop behaviors that *operate upon the environment*. OC was first investigated by Thorndike (1898) and later was expanded upon by Skinner (1938). In OC, a response that occurs with some minimum frequency is made to occur more frequently by following it with a particular type of reinforcement, be it positive or negative in form (Pear, 2001). As contrasted with respondent (i.e., classical) conditioning, OC involves directly associating a response with a stimulus event (not reward) rather than a stimulus with a stimulus. This is an important distinction that is often misguided and ill defined in learning research and clinical applications. A reward is a thing of value to an organism, whether it is food, water, points, monies, or any other stimulus. Reward is subject to individual differences except when a deprivation is present. Thus, the stimulus event's covariance with the desired response is the positive reinforcement (e.g., the car driving, ball bouncing, or beeping) and the reward directly follows the stimulus event. Behavior that has been learned through OC is called operant behavior, which may also be interpreted as learning to operate effectively and efficiently on the environment with its contingencies, consequences, and antecedent behaviors. A positive reinforcer is any stimulus whose presentation immediately following a response increases the probability of that response; while a negative reinforcer is any stimulus whose removal immediately following a response increases the probability of that response.

In laboratory experiments with animals it is well known that a deprivation must be present for the animal to engage in experimental protocols. For example, a socially enriched environment with a satiated (food, water, temperature, etc.) animal will be highly unlikely to engage in the process of pushing a lever for food, or other stimulus. Shaping of the response of interest is additionally important in the early stages of the experiment. Importantly and not always considered in the application of neurofeedback procedures, a deprivation must be present in order for shaping and conditioning of behavior to occur. Any human being presenting for neurofeedback training has an awareness of a deprivation (e.g., I can be better at memory, I would like to be less anxious, etc.). This is also true for individuals engaging in neurofeedback training for a particular syndrome. For example, children with Attention-deficit/Hyperactivity Disorder (ADHD) perceive there is a deficit in functionality as contrasted with other normative children, or interactions with teachers and parents. The same is true for the athlete or businessman wanting to perform at a higher level; or an individual with depression, anxiety, or any other problematic issues with emotional, cognitive, or adaptive skill sets. However, there are exceptions to every rule and in the case of OC and awareness of a deprivation, it may not always be discernible due to disorders of communication (autism or traumatic brain injury) yet the EEG and its association with behavioral regulation (excessive movement, emotional

reactivity, etc.) can be shaped and reinforced using OC.

Neurofeedback utilizes OC in a specific context that focuses on the electrical activity of the brain at the scalp, current source density at a specific region of interest, or blood oxygenated level dependent (BOLD) activity at a specific region of interest. Additionally, an EEG frequency that occurs at some minimal rate (e.g., 2 uV of beta to theta power, alpha power of 5 uV, or other designated anomaly) is targeted for conditioning. The most important measure in a neurofeedback learning paradigm is a learning curve that demonstrates acquisition has taken place; or more simply, that the individual has learned to change the EEG or brain activity in the desired direction. The formula for a neurofeedback mechanism using OC consists of a simple, yet elegant paradigm:

1. A value of a specific target frequency or set of target frequencies is selected (e.g., uV value of SMR, theta/beta ratio, or alpha amplitude).
2. A specific electrode site, set of sites, or region of training is selected (e.g., Cz, Fz, or anterior cingulate).
3. An establishing operation induces a motivational state based on the subject's deviation from a specified goal state (e.g., in ADHD, impairment of attention; in anxiety, the presence of an aversive subjective experience; in peak performance training, a greater than usual skill level).
4. Through positive or negative reinforcement, a desired change in EEG activity is documented.
5. Evidence of change is documented at other levels of analysis (e.g., subjective experience, psychopathology scales, neurophysiology assessments, cognitive or behavioral performance).

In many instances, research studies will document all of these elements. In a clinical setting this may be taxing on clinicians and technicians. However, it is not beyond the scope of a practitioner to produce a learning curve to provide evidence that acquisition has in fact occurred. I have conferred with several manufacturers of neurofeedback devices and all have affirmed the data within and across sessions is stored and accessible for production of learning curves; be it microvolt levels, percentage of time in reward, or points scored.

In the figures below, examples of group and individual acquisition curves are provided. Figure 1 shows an example of a learning curve for the average number of points generated for eight individuals with ADHD who had completed 17 to 20 sessions of neurofeedback. Figure 2 shows the average CSD for two study groups across sessions. Figure 3 shows the percentage of reward for an individual within and across 11 sessions. Figure 4 shows a learning trend for points scored in one session of neurofeedback for an individual.

There are two main requirements for demonstrating successful learning. The first is a stable trend in the desired direction. Although a linear trend is typical, nonlinear methods can also contribute to our understanding of learning across time (e.g., quadratic and cubic trends). For example, if one is training SMR uV levels up, then the plot of SMR should show an increasing positive trend. Likewise, if specific frequencies are inhibited, then the plot of voltage in those frequencies should demonstrate a decreasing trend. These can be considered the linear components of learning. Secondly, there should be a decrease in variance across sessions. For example, as the individual learns to self-regulate an EEG frequency, the values of EEG activity or number of points scored should become more consistent across sessions. The method of delivery (neurofeedback, MEG, fMRI, fNRIS) becomes secondary to the original definition of OC and its functional units of measurement: If no acquisition is demonstrated in the variables being trained, then, despite any change in measures at other levels of analysis (e.g., self-report, behavior measures), the process cannot be classified as NF_{oc}.

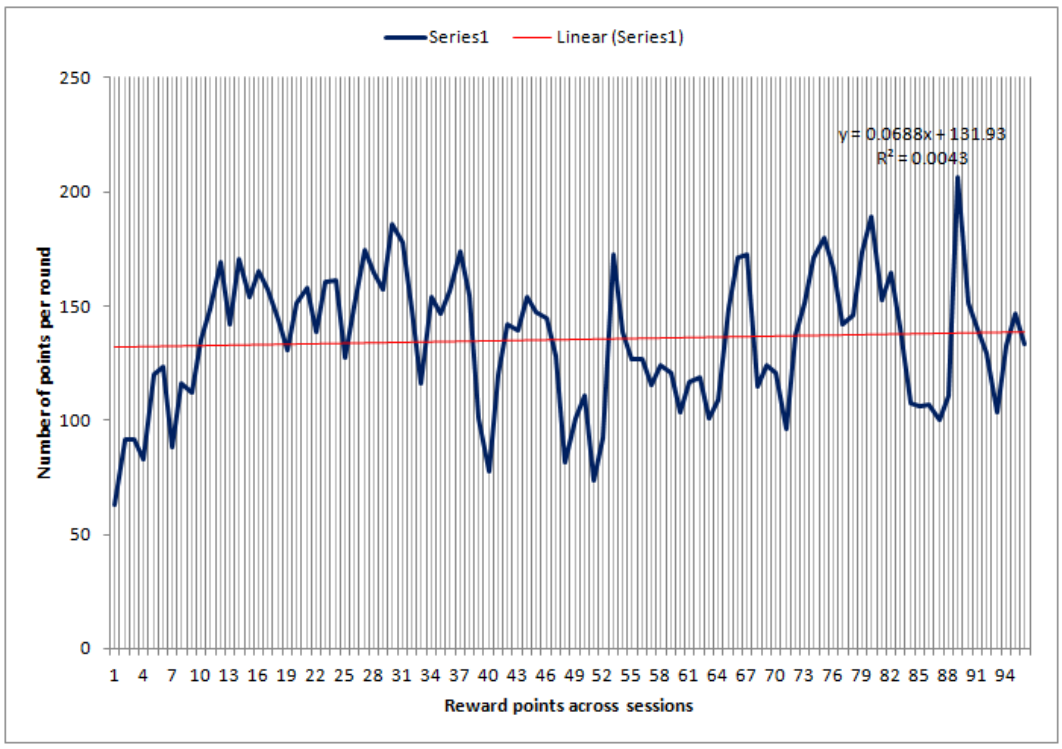

Figure 1. Average points scored across sessions for a group of 8 ADHD patients.

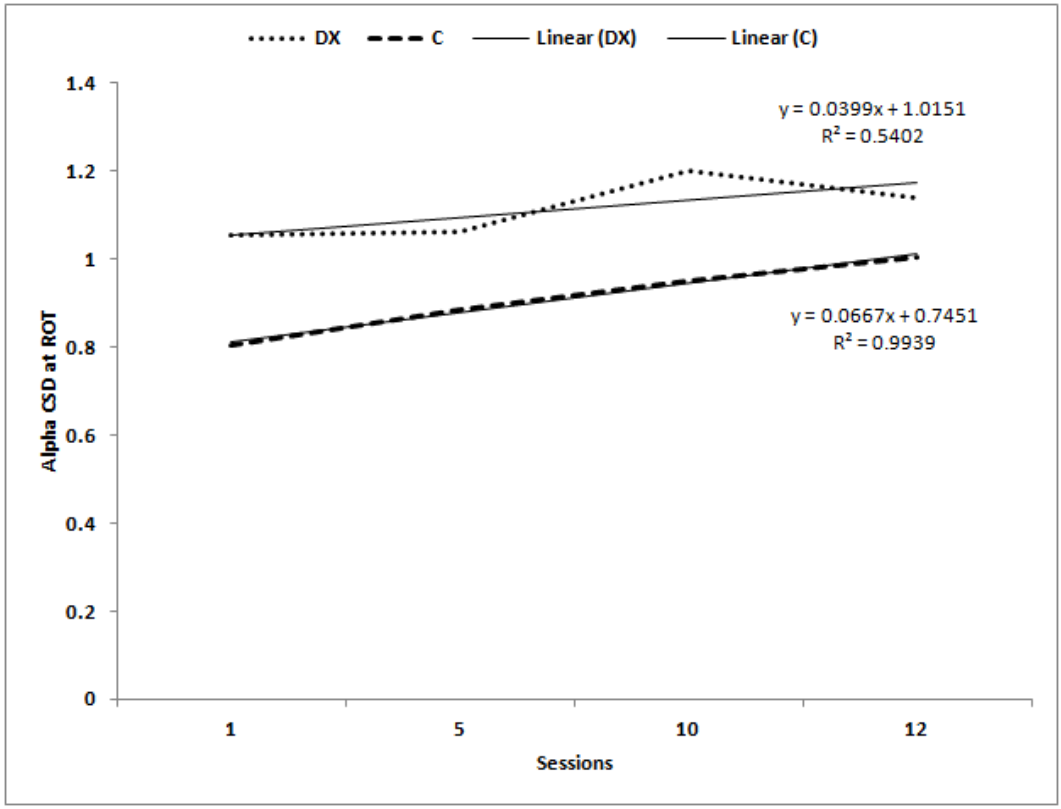

Figure 2. Two groups of individuals and average current source density (CSD) levels at the region of training (ROT) across sessions by using sessions 1, 5, 10 and 12.

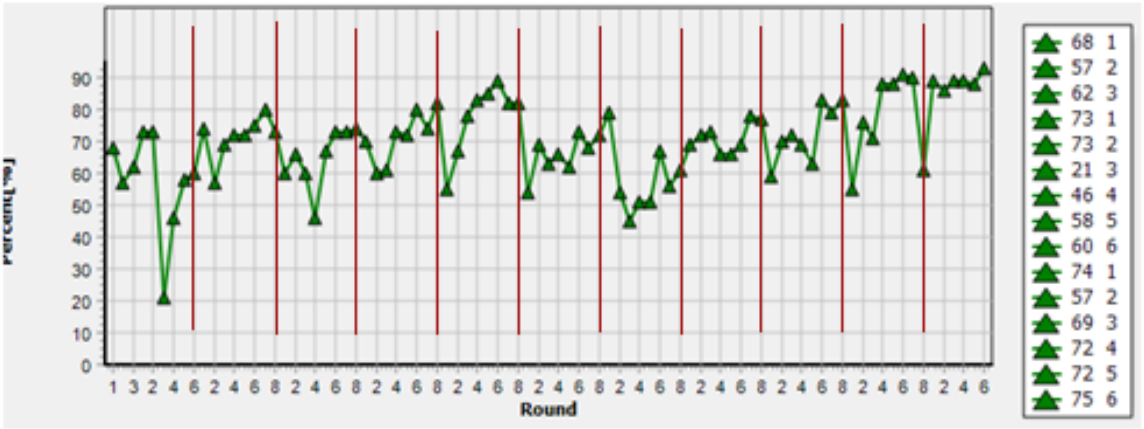

Figure 3. Percentage of time in reward for an individual across 11 sessions and within sessions. Each session consists of six 5-min training rounds.

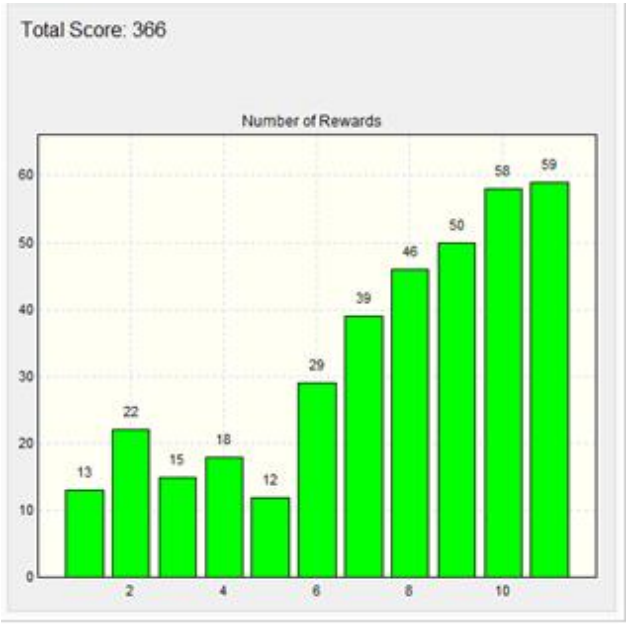

Figure 4. Learning trend generated by an individual within a single neurofeedback session. Number of rewards per 2-min rounds; 11 rounds, for a total of 22 min training time.

Neuroplasticity

Neuroplasticity (NP) is the inherent capacity of the brain to develop new connections and pathways as a compensatory mechanism for injury, or as a function of learning in response to experience and changes in the environment. Practice and learning play a vital role in human brain plasticity over the lifespan (Kelly, Foxe, & Garavan, 2006). Clearly, NP is the driving force in human learning (experience-dependent changes) over the lifespan. This experience-driven effect has refuted the long held position that the adult human brain is both hardwired and resistant to change (Holloway, Broadfield, & Yuan, 2003). Experience-driven changes in the brain have been widely demonstrated in both human and nonhuman primates and these findings present exceptional challenges for observing these mechanisms in vivo. However, technology and human drive to understand have produced more advanced methods to capture how these mechanisms operate. This has contributed to the development of methodologies that influence these processes and to novel treatments and diagnostic techniques for disorders of learning. There is little doubt among neuroscientists that symptoms associated with most psychiatric disorders can be attributed to brain activity and functional network disruptions, regional activation patterns, and lack of the functional integration of systems required for operating effectively on the environment. Currently, the most promising measure of plastic changes in the human brain, as a function of learning and practice, are demonstrated by volumetric magnetic resonance imaging (vMRI). This method shows increases or decreases in white matter and grey matter volume as a function of learning or training (practice) including neurofeedback methods (Beauregard & Lévesque, 2006; Ghaziri et al., 2013; Lévesque, Beauregard, & Mensour, 2006; Petersson, Elfgren, & Ingvar, 1999). Importantly, recent data have shown changes in individual neurons as an individual experiences new data (memories), as well as conditioning of spiking patterns in individual neurons in the hippocampus (Ishikawa, Matsumoto, Sakaguchi, Matsuki, & Ikegaya, 2014; Ison, Quiroga, & Fried, 2015). In most research demonstrating learning in human and nonhuman subjects, the principles of OC are followed and evidence of change is documented (Baxter & Byrne, 2006; Cannon, Baldwin, et al.,

2014; Mozzachiodi, Lorenzetti, Baxter, & Byrne, 2008; Spencer, Syed, & Lukowiak, 1999; Sterman, 2000). Clearly this definition will evolve and adapt as technology and understanding improve. However, in as much as learning and self-regulation are involved, NP is the primary target for all behavioral treatments and processes (including education). As such, NF_{oc} is a viable and reproducible method for improving NP associated with self-awareness, self-regulation, and behavior change.

Neural Efficiency

Neural efficiency (NE) and the effects of practice in the human brain offer the best promise in understanding behavior. Decreases in the extent or intensity of activations or activity are observed in the majority of studies examining task practice. The primary mechanism proposed to underlie activation decreases is increased NE; which, by definition, reflects an increased efficiency within a network such that operant efficiency now occurs with the engagement of fewer neural sources, as well as increased synchronous firing relative to a particular task or stimulus (Babiloni et al., 2009; Foerde et al., 2008; Poldrack, 2000, 2002; Poldrack, Desmond, Glover, & Gabrieli, 1998; Poldrack & Foerde, 2008; Poldrack & Gabrieli, 2001; Poldrack & Logan, 1997, 1998). Decreases in activation are suggested to reflect a more robust and efficient neural representation (Duncan & Miller, 2002) or a more precise functional circuit related to a behavior or function of interest (Garavan, Kelley, Rosen, Rao, & Stein, 2000). In several studies of practice effects in the brain, increases in activation or activity refer to two processes; practice-related expansions in the volume of cortical representations and increases in the strength or amplitude of activations (Kelly et al., 2006). Thus, NE is directly evident by a decrease in the number of resources (energy) allocated to perform a particular task. In many cases this may be referred to as automatic processing and can be thought of in terms of heartbeat, breathing, use of language, and those activities that are well learned and well practiced (the area or function of expertise). In EEG work, specifically we can think of NE in terms of EEG amplitude and global magnitude of the additive signals. For example, as an individual learns to regulate the behavior of sitting still and focusing on a stimulus we would expect an increase in the response of interest (e.g., SMR, low-beta, etc.). As the individual learns to generate this response and sustain it we would eventually expect a decrease in the signal amplitude once the skill is acquired. In sum, once an individual learns and masters a task we can surmise that the brain has adapted to best perform this task with minimal resource depletion.

Self-regulation

Self-regulation (SR) is a highly adaptive and powerful process (Vohs, Baumeister, & Ciarocco, 2005; Vohs et al., 2008). SR refers to the self's capacity to alter its behaviors based in the degree that human beings are adaptive and flexible (Vohs et al., 2005). Alternatively, SR can better be defined as plasticity that relies upon the functional integrity and NE of the brain and its network convergence or divergence in executive processes; including, self-monitoring, self-concept, self-control, self-perception, self-organization, self-related goal setting, planning, and agency (Cannon, Congedo, Lubar, & Hutchens, 2009; Cannon et al., 2007; Cannon & Baldwin, 2012; de Greck et al., 2008; Northoff et al., 2006). This is reinforced by evidence of clinical applications of SR in which Baumeister, Gailliot, DeWall, and Oaten (2006) and de Ridder and de Wit (2006) have proposed that whatever differences and deficits exist in the ability to self-regulate, either innate or learned during development, can be modified by additional learning. Thus, SR is the neural process of data integration and learning as it pertains to the self and its experiential functionality; or more simply, an adaptive data-driven process (Cannon, 2012).

SR is a skill necessary for reliable emotional well-being, or affective constancy. It is proposed that differential variants of SR include emotional, behavioral, and cognitive variants. Importantly, the most overlooked construct necessary for SR is language (defined as the ability to communicate, including internal self-directed speech) and practice. If, like many authors suggest, self-regulation is a skill or set of skills, then mastery of this skill requires extensive practice and learning. The key components for practice in the human brain are NP and NE. These components have been demonstrated in numerous fMRI studies of practice (Frackowiak & Ward, 2004; Fraser et al., 2002; Garrido et al., 2009; Kelly et al., 2006; Neville & Bavelier, 2002). It is well known that practice in effect can induce activation (learning) and decreased activation (well-learnt and less energy required) in numerous experimental conditions, from stringed instruments (Elbert, Pantev, Wienbruch, Rockstroh, & Taub, 1995), to motor functions (Fraser et al., 2002), verbal recall (Andreasen et al., 1995), and working memory (von Bastian, Langer, Jäncke, & Oberauer, 2013). Thus, there is sufficient

evidence that practice (learning by repetition) influences neural networks in both positive and negative fashion. Probably the best example of a negative instance is depression or anxiety. We might consider the influence of negative self-directed speech (Cannon, Lubar, Sokhadze, & Baldwin, 2008; Gilbert, Dumontheil, Simons, Frith, & Burgess, 2007) or processing of derogatory (Baumeister, 2003) appraisals of self by the self or others (Kim et al., 2008) and its potential effects in networks associated with the physiological response to stress. With the large literature of practice and learning and the role of SR across the biobehavioral spectrum we might strongly consider SR as the primary mechanism of action in neurotherapeutic procedures that require operant conditioning or learning (e.g., EEG biofeedback, neurofeedback; Kamiya, 2011; Wood & Peut, 1981).

Experience-dependent changes in the human brain can occur from a synaptic to a cortical level throughout the life span. There is a growing literature base demonstrating these NP effects in both human and nonhuman populations. NP can be thought of in terms of development, such that in our earliest periods of development we assimilate information because of learning (operant behavior) by mimicking, observing, and experiencing the environment. We also begin to organize our self critically based on our perceptions of self in relation to others (operant behavior driven by self-perception and its relation to the environment). As development progresses, so does the data-dependency requirements on the brain through which we learn to adapt an operant efficiency (the result of learning, practice, and specialization) relative to our culture, profession, ethnicity, and so forth. In essence, development is a function of operant learning, and disorders of learning begin and end with the central nervous system and its functional integrity (Cannon, Baldwin, et al., 2014).

Executive functions and self-regulation are better considered as synonymous, rather than independent processes and may best be described with functional neural signatures (e.g., functional integrity of the CNS) within the context of the Papez circuit (1937). One very important research finding—that is often overlooked or unknown in research paradigms investigating the limbic system and its function—is that hippocampal firing (generation of the theta frequency) is directly dependent on septal firing; that is to say, the hippocampus does not function independent of the septal area. Thus, the whole of the Papez circuit is proposed to be a specific network of mechanisms to integrate sensory, internal dialogue, and external information to regulate the hypothalamus and its control over behavioral patterns (Parmeggiani, Azzaroni, & Lenzi, 1971).

It is when some aspect of data integration is compromised and specific neural data-dependency modules become overly practiced and efficient within a maladaptive context (e.g., negative self-perception, abuse, poor affect regulation, or external and internal stressors) relative to the self occurs, that problems in operant efficiency and psychological well-being are compromised. Neurofeedback, in all its forms, holds great potential in providing an evidence-based mechanism for improving emotional and regulatory processes (Johnston, Boehm, Healy, Goebel, & Linden, 2010; Johnston et al., 2011). It is reasonable to consider that the self (organized neural networks) is both malleable and in some aspects more resistant to the effects of new learning (i.e., religion, disciplines, values, morality). It may also be that there are specific genetic mechanisms associated with the homeostatic maintenance of the organism that become disorganized or skewed toward a negative data-selection process. As put forth by Cannon, Baldwin, et al. (2014), SR can be conceptualized within this framework: Behavioral Equilibrium (BE; or Operant Efficiency) is dependent (on the output) of the interaction between emotional equilibrium (EE) and homeostasis (HS); or BE = EE/HS. The mediating variable for Operant Efficiency or Operant Inefficiency is SR or its equivalent executive functions. The primary assumptions underlying this model are NP and NE.

In sum, SR is directly related to NP and NE, and the role of these two processes in theory is based on cognitive and verbal tasks that show specific activations or deactivations as an effect of practice and learning, treatment effects of neurofeedback, transcranial magnetic stimulation, cognitive behavioral therapy, and/or other treatment models that have shown pre-post changes in the cortical landscape. Thus, the fundamental processes (mechanisms of action) underlying all NF_{oc} techniques, regardless of methodology, are NP and NE directed toward improved SR and learning (Johnston et al., 2010; Johnston et al., 2011) for optimal Operant Efficiency.

Conclusions

Neurofeedback continues to gain widespread interest and attention from numerous research, clinical, and performance-related disciplines. It therefore becomes pertinent to define the processes associated with neurofeedback and to differentiate between these methods. A large number of data have shown positive results for neurofeedback across methods. However, not all neurofeedback can be said to involve OC (Cannon, Pigott, et al., 2014). Operant conditioning requires a complete set of fundamental components, some of which are often lacking in both research and clinical realms. Thus, we might consider a classification system for neurofeedback with three designations: NF_{OC}, NF_{UT}, and NF_{ET}. Differentiating neurofeedback methods in these terms may resolve contradictory findings, could aid in reducing the number of confounds in research studies, and provide clients clear information on which to base their consent to treatment. Neurofeedback offers promise for influencing learning and SR across a variety of normative and clinical groups. Its methods and their description must improve along with technological advancements so that better and more consistent outcomes can be achieved.

Author Note:

Author reports no conflicts of interest.

References

Andreasen, N. C., O'Leary, D. S., Cizadlo, T., Arndt, S., Rezai, K., Watkins, G. L., … Hichwa, R. D. (1995). PET studies of memory: Novel versus practiced free recall of word lists. *NeuroImage, 2*(4), 296–305. http://dx.doi.org/10.1006/nimg.1995.1037

Babiloni, C., Marzano, N., Infarinato, F., Iacoboni, M., Rizza, G., Aschieri, P., … Del Percio, C. (2009). "Neural efficiency" of experts' brain during judgment of actions: A high-resolution EEG study in elite and amateur karate athletes. *Behavioural Brain Research, 207*(2), 466–-475. http://dx.doi.org/10.1016/j.bbr.2009.10.034

Baumeister, R. F. (2003). Ego depletion and self-regulation failure: A resource model of self-control. *Alcoholism: Clinical & Experimental Research, 27*(2), 281–284. http://dx.doi.org/10.1097/01.ALC.0000060879.61384.A4

Baumeister, R. F., Gailliot, M., DeWall, C. N., & Oaten, M. (2006). Self-regulation and personality: How interventions increase regulatory success, and how depletion moderates the effects of traits on behavior. *Journal of Personality, 74*(6), 1773–1802. http://dx.doi.org/10.1111/j.1467-6494.2006.00428.x

Baxter, D. A., & Byrne, J. H. (2006). Feeding behavior of Aplysia: A model system for comparing cellular mechanisms of classical and operant conditioning. *Learning and Memory, 13*(6), 669–680. http://dx.doi.org/10.1101/lm.339206

Beauregard, M., & Lévesque, J. (2006). Functional magnetic resonance imaging investigation of the effects of neurofeedback training on the neural bases of selective attention and response inhibition in children with attention-deficit/hyperactivity disorder. *Applied Psychophysiology and Biofeedback, 31*(1), 3–20. http://dx.doi.org/10.1007/s10484-006-9001-y

Cannon, R., Congedo, M., Lubar, J., & Hutchens, T. (2009). Differentiating a network of executive attention: LORETA neurofeedback in anterior cingulate and dorsolateral prefrontal cortices. *International Journal of Neuroscience, 119*(3), 404–441. http://dx.doi.org/10.1080/00207450802480325

Cannon, R., Lubar, J., Congedo, M., Thornton, K., Towler, K., & Hutchens, T. (2007). The effects of neurofeedback training in the cognitive division of the anterior cingulate gyrus. *International Journal of Neuroscience, 117*(3), 337–357. http://dx.doi.org/10.1080/00207450500514003

Cannon, R. L. (2012). *Low resolution electromagnetic brain tomography (LORETA): Basic concepts and clinical applications*. Corpus Christi, TX: BMED Press.

Cannon, R. L., & Baldwin, D. R. (2012). EEG current source density and the phenomenology of the default network. *Clinical EEG and Neuroscience, 43*(4), 257–267. http://dx.doi.org/10.1177/1550059412449780

Cannon, R. L., Baldwin, D. R., Diloreto, D. J., Phillips, S. T., Shaw, T. L., & Levy, J. J. (2014). LORETA neurofeedback in the precuneus: Operant conditioning in basic mechanisms of self-regulation. *Clinical EEG and Neuroscience, 45*(4), 238–248. http://dx.doi.org/10.1177/1550059413512796

Cannon, R. L., Lubar, J. F., Sokhadze, E., & Baldwin, D. R. (2008). LORETA Neurofeedback for Addiction and the possible neurophysiology of psychological processes influenced: A case study and region of interest analysis of LORETA neurofeedback in right anterior cingulate cortex. *Journal of Neurotherapy, 12*(4), 227–241. http://dx.doi.org/10.1080/10874200802501948

Cannon, R. L., Pigott, H. E., Surmeli, T., Simkin, D. R., Thatcher, R. W., Van den Bergh, W., … Koberda, J. L. (2014). The problem of patient heterogeneity and lack of proper training in a study of EEG neurofeedback in children. *Journal of Clinical Psychiatry, 75*(3), 289–290. http://dx.doi.org/10.4088/JCP.13lr08850

de Greck, M., Rotte, M., Paus, R., Moritz, D., Thiemann, R., Proesch, U., …. Northoff, G. (2008). Is our self based on reward? Self-relatedness recruits neural activity in the reward system. *NeuroImage, 39*(4), 2066–2075. http://dx.doi.org/10.1016/j.neuroimage.2007.11.006

de Ridder, T. D., & de Wit, J. B. F. (Eds.). (2006). *Self-regulation in Health Behavior*. West Sussex: John Wiley & Sons Ltd.

Duncan, J., & Miller, E. K. (2002). Cognitive focusing through adaptive neural coding in the primate prefrontal cortex. In D. Stuss, & R. T. Knight (Eds.), *Principles of Frontal Lobe Function* (pp. 278-291). Oxford: Oxford University Press.

Elbert, T., Pantev, C., Wienbruch, C., Rockstroh, B., & Taub, E. (1995). Increased cortical representation of the fingers of the left hand in string players. *Science, 270*(5234), 305–307. http://dx.doi.org/10.1126/science.270.5234.305

Foerde, K., Poldrack, R. A., Knowlton, B. J., Sabb, F. W., Bookheimer, S. Y., Bilder, R. M., … Asarnow, R. F. (2008). Selective corticostriatal dysfunction in schizophrenia: Examination of motor and cognitive skill learning. *Neuropsychology, 22*(1), 100–109. http://dx.doi.org/10.1037/0894-4105.22.1.100

Frackowiak, R. S., & Ward, N. S. (2004). Recovery of function and plasticity in human brain. *Royal Academy of Medicine in Belgium, 159*(Pt 2), 145–155.

Fraser, C., Power, M., Hamdy, S., Rothwell, J., Hobday, D., Hollander, I., … Thompson, D. (2002). Driving plasticity in human adult motor cortex is associated with improved motor function after brain injury. *Neuron, 34*(5), 831–840. http://dx.doi.org/10.1016/S0896-6273(02)00705-5

Garavan, H., Kelley, D., Rosen, A., Rao, S. M., & Stein, E. A. (2000). Practice-related functional activation changes in a

working memory task. *Microscopy Research Technique, 51*(1), 54–63. http://dx.doi.org/10.1002/1097-0029(20001001)51:1<54::AID-JEMT6>3.0.CO;2-J

Garrido, M. I., Kilner, J. M., Kiebel, S. J., Stephan, K. E., Baldeweg, T., & Friston, K. J. (2009). Repetition suppression and plasticity in the human brain. *NeuroImage, 48*(1), 269–279. http://dx.doi.org/10.1016/j.neuroimage.2009.06.034

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

Gilbert, S. J., Dumontheil, I., Simons, J. S., Frith, C. D., & Burgess, P. W. (2007). Comment on "Wandering minds: the default network and stimulus-independent thought." *Science, 317*(5834), 43. http://dx.doi.org/10.1126/science.1140801

Hammond, D. C. (2011). What is neurofeedback: An update. *Journal of Neurotherapy, 15*(4), 305–336. http://dx.doi.org/10.1080/10874208.2011.623090

Holloway, R. L., Broadfield, D. C., & Yuan, M. S. (2003). Morphology and histology of chimpanzee primary visual striate cortex indicate that brain reorganization predated brain expansion in early hominid evolution. *The Anatomical Record, 273A*(1), 594–602. http://dx.doi.org/10.1002/ar.a.10071

Ishikawa, D., Matsumoto, N., Sakaguchi, T., Matsuki, N., & Ikegaya, Y. (2014). Operant conditioning of synaptic and spiking activity patterns in single hippocampal neurons. *The Journal of Neuroscience, 34*(14), 5044–5053. http://dx.doi.org/10.1523/JNEUROSCI.5298-13.2014

Ison, M. J., Quiroga, R. Q., & Fried, I. (2015). Rapid encoding of new memories by individual neurons in the human brain. *Neuron, 87*(1), 220–230. http://dx.doi.org/10.1016/j.neuron.2015.06.016

Johnston, S. J., Boehm, S. G., Healy, D., Goebel, R., & Linden, D. E. J. (2010). Neurofeedback: A promising tool for the self-regulation of emotion networks. *NeuroImage, 49*(1), 1066–1072. http://dx.doi.org/10.1016/j.neuroimage.2009.07.056

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

Kamiya, J. (2011). The first communications about operant conditioning of the EEG. *Journal of Neurotherapy, 15*(1), 65–73. http://dx.doi.org/10.1080/10874208.2011.545764

Kelly, C., Foxe, J. J., & Garavan, H. (2006). Patterns of normal human brain plasticity after practice and their implications for neurorehabilitation. *Archives of Physical Medicine and Rehabilitation, 87*(12 Suppl 2), S20–29. http://dx.doi.org/10.1016/j.apmr.2006.08.333

Kim, J.-W., Choi, E. A., Kim, J.-J., Jeong, B. S., Kim, S.-E., & Ki, S. W. (2008). The role of amygdala during auditory verbal imagery of derogatory appraisals by others. *Neuroscience Letters, 446*(1), 1–6. http://dx.doi.org/10.1016/j.neulet.2008.09.019

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

Mozzachiodi, R., Lorenzetti, F. D., Baxter, D. A., & Byrne, J. H. (2008). Changes in neuronal excitability serve as a mechanism of long-term memory for operant conditioning. *Nature Neuroscience, 11*(10), 1146–1148. http://dx.doi.org/10.1038/nn.2184

Neville, H., & Bavelier, D. (2002). Human brain plasticity: Evidence from sensory deprivation and altered language experience. *Progress in Brain Research, 138*, 177–188. http://dx.doi.org/10.1016/S0079-6123(02)38078-6

Northoff, G., Heinzel, A., de Greck, M., Bermpohl, F., Dobrowolny, H., & Panksepp, J. (2006). Self-referential processing in our brain—A meta-analysis of imaging studies on the self. *NeuroImage, 31*(1), 440–457. http://dx.doi.org/10.1016/j.neuroimage.2005.12.002

Parmeggiani, P. L., Azzaroni, A., & Lenzi, P. (1971). On the functional significance of the circuit of Papez. *Brain Research, 30*(2), 357–374. http://dx.doi.org/10.1016/0006-8993(71)90086-2

Pear, J. (2001). *The science of learning*. Psychology Press.

Petersson, K. M., Elfgren, C., & Ingvar, M. (1999). Dynamic changes in the functional anatomy of the human brain during recall of abstract designs related to practice. *Neuropsychologia, 37*(5), 567–587.

Poldrack, R. A. (2000). Imaging brain plasticity: Conceptual and methodological issues—A theoretical review. *NeuroImage, 12*(1), 1–13. http://dx.doi.org/10.1006/nimg.2000.0596

Poldrack, R. A. (2002). Neural systems for perceptual skill learning. *Behavioral and Cognitive Neuroscience Reviews, 1*(1), 76–83. http://dx.doi.org/10.1177/1534582302001001005

Poldrack, R. A., Desmond, J. E., Glover, G. H., & Gabrieli, J. D. (1998). The neural basis of visual skill learning: An fMRI study of mirror reading. *Cerebral Cortex, 8*(1), 1–10. http://dx.doi.org/10.1093/cercor/8.1.1

Poldrack, R. A., & Foerde, K. (2008). Category learning and the memory systems debate. *Neuroscience and Biobehavioral Reviews, 32*(2), 197–205. http://dx.doi.org/10.1016/j.neubiorev.2007.07.007

Poldrack, R. A., & Gabrieli, J. D. E. (2001). Characterizing the neural mechanisms of skill learning and repetition priming: Evidence from mirror reading. *Brain, 124*(1), 67–82. http://dx.doi.org/10.1093/brain/124.1.67

Poldrack, R. A., & Logan, G. D. (1997). Fluency and response speed in recognition judgments. *Memory and Cognition, 25*(1), 1–10. http://dx.doi.org/10.3758/BF03197280

Poldrack, R. A., & Logan, G. D. (1998). What is the mechanism for fluency in successive recognition? *Acta Psychologica, 98*(2-3), 167–181. http://dx.doi.org/10.1016/S0001-6918(97)00041-3

Skinner, B. F. (1938). *The Behavior of Organisms: An Experimental Analysis*. New York: Appleton-Century.

Spencer, G. E., Syed, N. I., & Lukowiak, K. (1999). Neural changes after operant conditioning of the aerial respiratory behavior in Lymnaea stagnalis. *The Journal of Neuroscience, 19*(5), 1836-1843.

Sterman, M. B. (2000). Basic concepts and clinical findings in the treatment of seizure disorders with EEG operant conditioning. *Clinical EEG and Neuroscience, 31*(1), 45–55. http://dx.doi.org/10.1177/155005940003100111

Thibault, R. T., Lifshitz, M., Birbaumer, N., & Raz, A. (2015). Neurofeedback, self-regulation, and brain imaging: Clinical science and fad in the service of mental disorders. *Psychotherapy and Psychosomatics, 84*(4), 193–207. http://dx.doi.org/10.1159/000371714

Thorndike, E. L. (1898). Animal Intelligence: An experimental study of the associative process in animals. *Psychological Monographs: General and Applied*, *2*(4), i–109.

Vohs, K. D., Baumeister, R. F., & Ciarocco, N. J. (2005). Self-regulation and self-presentation: Regulatory resource depletion impairs impression management and effortful self-presentation depletes regulatory resources. *Journal of Personality and Social Psychology, 88*(4), 632–657. http://dx.doi.org/10.1037/0022-3514.88.4.632

Vohs, K. D., Baumeister, R. F., Schmeichel, B. J., Twenge, J. M., Nelson, N. M., & Tice, D. M. (2008). Making choices impairs subsequent self-control: A limited-resource account of decision making, self-regulation, and active initiative. *Journal*

of Personality and Social Psychology, 94(5), 883–898. http://dx.doi.org/10.1037/0022-3514.94.5.883

von Bastian, C. C., Langer, N., Jäncke, L., & Oberauer, K. (2013). Effects of working memory training in young and old adults. *Memory & Cognition, 41*(4), 611–624. http://dx.doi.org/10.3758/s13421-012-0280-7

Wood, D. J., & Pesut, D. J. (1981). Self-regulatory mental processes and patient recovery. *Western Journal of Nursing Research, 3*(3), 262–267. http://dx.doi.org/10.1177/019394598100300322

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NeuroRegulation

Frontal Gamma Asymmetry in Response to Soft Skills Stimuli: A Pilot Study

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Abstract

Objective: The purpose of this pilot study was to explore associations between self-reported rank ordering of a set of 23 job-related soft skills and frontal gamma (38 to 42 Hz) asymmetry emerging during exposure to the same set of soft skills. **Method:** Fourteen individuals responded to a soft skill assessment, then were exposed to a randomized list of the same soft skills' key words while collecting electroencephalographic (EEG) data, using a new implementation of standardized low-resolution brain electromagnetic tomography (sLORETA) to analyze and view voxel images of real-time brain activity. A differential calculation, as a measure of approach or avoidance to the key word stimulus (Approach-Avoidance-Differential; AAD), was used to quantify the asymmetry in response to the stimuli. Spearman's Rank correlations (r_s) were calculated for the paired occurrences between the self-reported ranking of the soft skills and the AAD. **Results:** Overall, 71% of the cases resulted in correlations, indicating soft skill directionality response. Reduction in gamma response intensity was seen when participants' ranked their highest to lowest soft skills, as indicated in their self-reported assessments. **Conclusions:** These results will inform further organizational neuroscience research which has potential to lead to a new approach to self-report validation and methods to detect individual approach or avoidance biases which impact self-reporting assessments.

Keywords: gamma asymmetry, soft skills, precognition, industrial/organizational psychology, organizational neuroscience, sLORETA, approach-avoidance

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Introduction

The concept of self-report is a key element in many phases of psychology, counseling, as well as industrial-organizational coaching and human resource management. It is often the only means to probe the internal world of a person. However, self-reports are limited by key issues, including the level of awareness, honesty, and ability to express internal thoughts and feelings, which is highly variable across individuals (Kanai & Rees, 2011). Self-reporting assessments, frequently in the form of ipsative or force choice assessments, are therefore suspect in the absence of cross-verification. Objective measures, including physiological responses, nonverbal expressions, and other observables, can be considered to supplement self-reports.

A further limitation of ipsative assessments, as outlined in Bedwell, Fiore, and Salas (2011, 2014), are that several particular biases can influence self-report measures: Consistency motif, social desirability, acquiescence biases, and self-serving biases. While a comprehensive discussion of these influences is beyond the scope of this paper, a brief

overview will add clarity to this aspect of self-report assessments. The *consistency motif* implies that individuals may attempt to create consistency in their thoughts and feelings and, as a result, maintain consistency in their responses rather than dealing with each question individually. *Social desirability* addresses the need for social approval and acceptance that can lead to behaviors deemed culturally acceptable, thus presenting themselves in a favorable manner, regardless of their true feelings or tendencies. *Acquiescence bias* occurs when respondents generally agree (or disagree) with questionnaires, regardless of content. This may result in some components of an assessment seeming to be related, when in fact, they are not. *Self-serving bias* may occur when people attribute the more positive aspects of their performance to their own traits or dispositions and poor performance to external factors. This bias may result in higher self-ratings on assessment questions regarding mastery levels. Thus, it is crucial that participants be truthful to themselves and that the assessment output is only as accurate as the input. To address these concerns, assessments many times use checks of internal consistency, validity and reliability, and norm comparisons. Ultimately, however, a brain-based measure would be optimal, if it could provide a meaningful indicator of emotional, cognitive, and motivational states and serve as correlation to self-reported responses.

To begin investigating the potential for such a brain-based measure, we hypothesized that a soft skill assessment tool could be administered and then correlated to real-time brain activity. The concept leading to these soft skills, also referred to as competencies, were first described by McClelland (1961); thus, leading to the present-day competency-based job descriptions commonly used in the field of industrial-organizational psychology.

Building on this workplace skill concept, cognitive research has begun to illuminate how intrapersonal and interpersonal competencies are crucial to daily interactions (e.g., National Research Council, 2008, 2012). When matching a person to a job, it becomes crucial that we be able to predict not just what a person knows, but rather, what they are capable of knowing. Furthermore, research has shown that these competencies are not fixed but are developed by prior experience (Yeager & Walton, 2011), and thus should be tied to memories and emotions associated with those experiences; and therefore, should be traceable in brain imaging. The concept of *capacity* to know becomes paramount as capacity implies that something needs to be in place for learning to occur. The work of Dweck (2006) refers to potential barriers to learning as a "fixed mindset." A fix mindset is a belief about one's ability that can block learning; and many times are associated with past negative events. It is for this reason that we must separate what a person knows from what they are capable of knowing. Simply providing the learning opportunity without first exposing hidden fix mindsets may not result in learning. It is these negative memories and corresponding emotions that many times must be addressed before learning can occur.

The National Research Council's 2012 report of 21st century skills challenges us to recognize the critical role soft skills play in our lives, both on and off the job. They stress the need for students and working adults to be able to identify and develop skills that include innovation, creativity, problem solving, critical thinking, communication, collaboration, and self-management; all of which are among the job-related competencies assessed in this study. In other words, the challenge has been to develop valid and accurate assessment instruments that allow an individual, or an employer, the ability to assess these soft skills quickly and with confidence.

In the quest to investigate the neural underpinnings of individuals' internal processes, as related to industrial and organizational psychology, an entire field termed organizational neuroscience (also called neuroleadership) has begun to emerge; which can further be conceptualized as a branch of the greater field of social cognitive neuroscience (Lafferty & Alford, 2010; Rock, 2008). The advent of functional magnetic resonance imaging (fMRI) has greatly advanced these efforts. The fMRI signal is produced when changes in blood oxygenation and flow are detected secondary to neuronal activity; thus, when more oxygen is consumed in response to increased brain activity this response is represented in brain activation maps indicating localization of mental processes (Arthurs & Boniface, 2002). Its use in cognitive neuroscience began in the 1990s, with its most prevalent contribution being the discovery that small areas of brain function can be associated with the act of mentalizing, together with its spatial resolution at the size of 1 or 2 mm (Arthurs & Boniface, 2002; Mitchell, 2008). As an example of this line of research, Tabibnia, Satpute, and Lieberman (2008) were able to compare brain activity and self-reported measures to evaluate perceptions of fairness versus unfairness. However, some research from this field is beginning to incorporate quantitative electroencephalography (qEEG) technology to investigate neural measures,

or signatures, of internal processes. For example, Waldman, Balthazard, and Peterson (2011) used qEEG to link coherence and inspirational leadership traits, wherein they found a correlation between right frontal coherence and socialized visionary communication. In addition to being far less costly to implement and easier to access, one distinct advantage of qEEGs over fMRIs is improved temporal resolution; meaning that qEEGs can record neural activity faster, in terms of milliseconds (Hüsing, Jäncke, & Tag, 2006).

One such qEEG technology is low-resolution brain electromagnetic tomography (LORETA). LORETA incorporates a mathematical inverse solution of surface EEG data, which can provide cortical source localization, and generates three-dimensional images, similar to those produced by fMRI data (Thatcher, 2013). The LORETA algorithm creates an estimate of brain activity, termed current source density (CSD), in a virtual space representing cortical structures, encompassing 2,394 coordinates, expressed as 7 mm^3 sized voxels (The KEY Institute for Brain-Mind Research, 2014). A new generation of the algorithm, standardized LORETA (sLORETA; Pascual-Marqui, 2002), advances this concept, and bases the computations on a standardized CSD such that the voxel size is 5 mm^3 for a total of 6,239 voxels.

The Role of Approach-Avoidance Asymmetry

While advancements in EEG imaging, quantitative, and source localization analysis are key to this paper, the underlying concept of frontal lobe asymmetry provides the theory bases and can be traced back to Davidson, Schwartz, Saron, Bennett, and Goleman (1979) where they first described the use of scalp-recorded EEG asymmetry and the possible connection to emotional processes. What followed was a plethora of studies documenting the role of frontal lobe approach-avoidance asymmetry in emotional processing and decision making (Davidson, 1992, 2000, 2002, 2004; Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Gordon, Barnett, Cooper, Tran, & Williams, 2008; Harmon-Jones 2004; Nitschke, Heller, Etienne, & Miller, 2004; Rock, 2008; Rolls, 1999). Davidson, Pizzagalli, Nitschke, and Kalin (2003) summarized the research up to that point by proposing that greater left-side prefrontal cortex activity appeared to be associated with approach-related and goal-directed action planning, while the right suggests avoidance-related emotions.

While the ability to differentiate approach (reward) from avoid (threat) is in itself noteworthy, it is important to understand the bases of this process is directly tied to emotional expressions. Gordon et al. (2008) defines emotions as "adaptive actions tendencies that are mobilized by signals of potential danger or reward. They involve a 'feedforward' mode of brain and body activity that is triggered automatically and without the need for conscious awareness of the triggering signal" (p. 349). They refer to this response to stimuli as a nonconscious emotional reaction, while Collura, Zalaquett, Bonnstetter, and Chatters (2014) define this limbic system processing prior to cognitive awareness as a *precognition*. Naccache et al. (2005) explains that the limbic networks can process threat and reward cues within 200ths of a second, thus supplying a continuous nonconscious response to every interaction we have, all day long. Being able to detect the corresponding brain activity, when these precognitions occur, provides a direct link to the emotions and experiences behind our decisions, and exposes our thought processing before conscious thoughts or self-regulation can take place (Gordon et al., 2008).

It is important to note that through the 1990s research examining and documenting the concept of approach-avoidance was confined to slower frequency analysis, primarily alpha asymmetry. However, this began to change with a series of experiments (Pizzagalli, Greischar, & Davidson, 2003; Pizzagalli, Nitschke, et al., 2002; Pizzagalli, Pascual-Marqui, et al., 2001) which included a focus on frequencies in addition to alpha, such as theta, beta, and to a lesser extent, gamma; wherein frontal asymmetries were at times found (Davidson, 2004). Then, Oakes et al. (2004) correlated LORETA CSD to regional glucose metabolism with positron emission tomography, where they found that while alpha did show an expected asymmetry relation, the frequency band most consistently and strongly associated with glucose metabolism was gamma; with localization primarily in the frontal lobe region. As a result of these studies, Davidson (2004) concluded an important aspect in advancing this line of research would be exploring frequencies other than alpha to garner additional information. This identified gap in the research, combined with the noted aspect of gamma discovered by Oakes et al. (2004), provides the basis for this study with its focus on gamma asymmetry. Moreover, the vast majority of this line of research has implemented surface-recorded EEG data, with an inclusion of LORETA source localization to a lesser degree. Yet, recent advances in qEEG applications have

demonstrated it may be possible to use a new implementation of sLORETA to both analyze and view voxel images of real-time gamma brain activity that may reflect emotional states as related to precognitive activity (Collura, Bonnstetter, & Zalaquett, 2014; Collura, Zalaquett, et al., 2014).

Our past research and data in assessing workplace soft skills has shown that past experiences, combined with emotional connections to those experiences, are a key component to soft skill development. This research, coupled with frontal asymmetry literature, leads us to hypothesize that frontal asymmetry would correlate to the emotionally laden component of self-reported soft skill rankings. Therefore, this pilot study sought to detect sLORETA-derived real-time brain activity within this approach-avoidance framework, and focused on the comparison of soft skills assessment and precognitive response gamma (38 to 42 Hz) asymmetry. The main purpose of the study, with a single-subject design, was to measure correlations between self-reported rank ordering of a set of 23 soft skills and frontal gamma asymmetry emerging during exposure to the same set of soft skills stimuli, while simultaneously acquiring EEG data.

Methods

Participants

Fourteen individuals participated in the study over a 4-month period. The participants ranged in age from 29 to 67 (M = 47.8, SD = 11.5), with 9 being male and 5 female. Ethnicity was primarily white; education included five each master's and bachelor's degrees; all were employed. See Table 1 for a breakout of age, gender, ethnicity, education, and occupation. All participants had normal or corrected-to-normal vision and no history of traumatic brain injury; there was no screening for substance and/or alcohol use/abuse. The external Institutional Review Board (IRB) of the Center for Applied Cognitive Research approved this study for the protection of human subjects in research, and all participants signed an informed consent document.

Table 1
Demographic Data

Case #	Age	Gender	Ethnicity	Education	Occupation
1	67	Male	White	Master's	Chief Executive Officer
2	48	Male	White	Bachelor's	Facilitator/Trainer
3	48	Male	White	Bachelor's	Fitness Trainer
4	47	Male	White	High School	Consultant Supervisor
5	60	Male	White	Master's	Consultant
6	29	Female	White	Associate	Customer Support
7	59	Female	White	Bachelor's	Consultant
8	62	Male	White	Bachelor's	Executive Coach
9	37	Female	Hispanic/Latino	Bachelor's	Sales Associates
10	41	Male	White	Bachelor's	Lead Programmer/Analyst
11	56	Male	White	Master's	Vice President
12	36	Female	Middle Eastern	N/A	N/A
13	40	Male	Hispanic/Latino	Master's	N/A
14	39	Female	White	Master's	Executive Coach

Note. N/A = not available due to participant not providing that information.

Materials

To assess the extent to which participants have developed a set of 21st century competencies, the TriMetrix® DNA, (Target Training International, Ltd; Scottsdale, AZ) assessment was used to gain insights into the developmental level of these work-related competencies. Table 2 provides a listing and definitions of each soft skill assessed.

Table 2
Definitions of 23 Professional/Personal Soft Skill Competencies

Analytical problem solving	Anticipating, analyzing, diagnosing, and resolving problems.
Conflict management	Addressing and resolving conflict constructively.
Continuous learning	Taking initiative in learning and implementing new concepts, technologies, and/or methods.
Creativity/innovation	Adapting traditional or developing new approaches, concepts, methods, models, designs, processes, technologies, and/or systems.
Customer service	Anticipating meeting and/or exceeding customer needs, wants, and expectations.
Decision making	Utilizing effective processes to make decisions.
Diplomacy	Effectively handling difficult or sensitive issues by utilizing tact, diplomacy, and an understanding of organizational culture, climate, and/or politics.
Empathy	Identifying with and caring about others.
Employee development/coaching	Facilitating and supporting the professional growth of others.
Flexibility	Agility in adapting to change.
Futuristic thinking	Imagining, envisioning, projecting, and/or predicting what has not yet been realized.
Goal orientation	Energetically focusing efforts on meeting a goal, mission, or objective.
Interpersonal skills	Effectively communicating, building rapport, and relating well to all kinds of people.
Leadership	Achieving extraordinary business results through people.
Management	Achieving extraordinary results through effective management of resources, systems, and processes.
Negotiation	Facilitating agreements between two or more parties.
Personal effectiveness	Demonstrating initiative, self-confidence, resiliency, and a willingness to take responsibility for personal actions.
Persuasion	Convincing others to change the way they think, believe, or behave.
Planning/organizing	Using logical, systematic, and orderly procedures to meet objectives.
Presenting	Communicating effectively to groups.
Self-management (time/priorities)	Demonstrating self-control and an ability to manage time and priorities.
Teamwork	Working effectively and productively with others.
Written communication	Writing clearly, succinctly, and understandably.

A crosswalk of these 23 skills against 21st century soft skills is shown in Table 3, as originally published in Gosselin, Cooper, Bonnstetter, and Bonnstetter (2013). Human resource personnel have found that an individual's hierarchy of competencies is key to their success and knowing what they are is essential to reaching their goals (National Research Council, 2012). This assessment is designed to assist in managing and developing a career. For many jobs, personal skills are as important as technical skills in producing superior performance. The TriMetrix® DNA report describes what an individual "has done" in 23 research-based capacities related to the business environment.

Data from over 25,000 participants are used, on an annual basis, to validate total variance of the TriMetrix® DNA, meaning that each of the 92 Likert-scaled questions has a response range that encompasses the one through six choices from agree to disagree. For inter-rater reliability, a 360-degree feedback survey is also used to assess the perception of others on an individual's evidence-based competencies; thus, triangulating between at least three auditors (e.g., peers, supervisors, subordinates, customers) to check perceptual agreement.

Table 3
Cross-walk of 23 Competencies Assessed Using the TTI TriMetrix® DNA™ System and Categorized Using the Domains Identified by the National Research Council

Domains from National Research Council (2012)	TTI TriMetrix® DNA Competencies	
Cognitive Competencies: n = 5	• Planning and Organizing • Analytical Problem Solving • Decision Making	• Creativity/Innovation • Futuristic Thinking
Intrapersonal Competencies: n = 5	• Continuous Learning • Goal Orientation • Self-Management	• Flexibility • Personal Effectiveness
Interpersonal Competencies: n = 13	• Employee Development/Coaching • Presenting • Diplomacy • Management • Customer Service • Interpersonal Skills • Leadership	• Teamwork • Conflict Management • Empathy • Persuasion • Written Communication • Negotiation

Note. Adapted from Gosselin et al. (2013).

Procedures

In the first phase of the procedure, each of the 14 participants completed the 30- to 40-min TriMetrix® DNA assessment online, which included a set of questions concerning their personal perception of 23 competency accomplishment, as well as questions regarding others' perceptions of their abilities. This assessment was completed approximately two weeks prior to the EEG data collection phase. In this second phase, each participant was exposed to a randomized list of the same soft skills' key words while EEG was simultaneously acquired. Prior to commencement of EEG recording, subjects were told that the experiment was concerned with collecting their reaction to a set of words or short phrases that may or may not describe them. Once background and demographic data was collected participants were readied for EEG collection.

Each subject was fitted with an electrode cap (Electro-Cap International; Eaton, OH) with 19 tin electrodes (plus a ground electrode), positioned to the International 10-20 system of electrode placement. The EEG amplifier was the Discovery 24E (Brainmaster Technologies; Bedford, OH) with a sampling rate of 1,024 samples per second (data rate to the computer of 256 samples per second), an A/D conversion of 24-bit resolution, EEG bandwidth of 0.43–80 Hz, and input impedance of 1,000 Gohm. EEG was acquired with the BrainAvatar software (Brainmaster Technologies; Bedford, OH) with linked ears reference; electrode impedance was adjusted to be below 10 kohm. During the stimuli presentation, two auxiliary channels of the amplifier were used to record event start and stop markers. These markers were generated using a predesigned random set of soft skill stimuli built into an E-Prime 2.0 software (Psychology Software Tools, Inc.; Sharpsburg, PA). Prior to presenting the stimuli, 2 min of eyes-open and 2 min of eyes-closed EEG was collected for further analysis, if deemed necessary.

The procedure next involved presenting a series of screens shots positioned on a 20-inch monitor, 1.5 m from the participant. They were told to watch the screen and focus attention on the words presented. The stimuli, presented in random order, appeared on the screen and remained for 1.5 s, followed by a random blank screen from 1 to 5 s. Further explanation of the basic setup and procedures used for data collection can be found in Bonnstetter, Collura, Hebets, and Bonnstetter (2012). This process allows for a series of *modified* event-related potential (ERP) type experiments. While not ERPs in the traditional sense, these events are still a time-locked stimulus to surface brain activity and measure transient electrical potential gamma shifts during cognitive processing. This patented process, referred to as Validating Ipsative Decision-making with Electroencephalography (VIDE; U.S. Patent No. 9,060,702, 2015) provides the intensity of a person's emotional response to a stimulus, by measuring voxel activation and emotional directionality, by differentiating approach versus avoidance responses within the prefrontal cortex.

Data Analysis

During post-processing analysis, averaged voxel values and sLORETA images were matched to the individual soft skill stimuli presented, to examine any associations. The region of interest (ROI) for analysis was identified as "frontal lobe," as predefined in the BrainAvatar imaging software (as designated by the Key Institute sLORETA model) and included only the left and right frontal lobes. In this software, the frontal lobe ROI is a very large area containing 2,176 voxels, which encompasses 35% (2,176 of 6,239) of all voxels.

Included in this ROI are the right and left Brodmann areas as follows: All of BA 6, 8, 9, 11, 44, 46; a majority or most of BA 4, 5, 10, 25, 45, 47; and a relative few voxels from 3, 13, 31, 32, 34, 43. In essence, this constitutes the first 2,176 voxels from The Key Institute sLORETA voxel index (http://www.uzh.ch/keyinst/loreta.htm). Table 4 provides a breakout, sorted by voxel number, of the specific voxels included in each BA making up the sLORETA frontal lobe ROI.

The resulting quantitative values were the average of the ROI voxels for the right and left frontal lobes. A measure of *acceptance* versus *avoidance* was calculated in the form of a numeric difference, termed the Approach-Avoidance-Differential (AAD), indicating the relative amount of energy in the right frontal lobe ROI, compared to the left. The AAD calculation is the average of the right hemisphere ROI voxels minus the average of the left hemisphere ROI voxels (1,088 voxels per hemisphere). A negative value indicates greater left hemisphere activation and implies *approach* (i.e., a sense of accepting thoughts, feelings, and behavior) towards the stimulus word, a positive value indicates greater right hemisphere activation and implies *avoidance* (i.e., a sense of aversion) against the stimulus word, and a value near zero implies a neutral response.

Table 4

Brodmann Area Voxels Included in sLORETA Frontal Lobe ROI

Brodmann Area	Total Number of Voxels in BA	Voxel Number Range Included	Number of Voxels Included in BA	Percentage BA Included
BA-10	272	1–268	268	99%
BA-11	239	269–507	239	100%
BA-13	248	508–517	10	4%
BA-25	45	518–550	33	73%
BA-3	129	551–553	3	2%
BA-31	194	554–574	21	11%
BA-32	155	575–596	22	14%
BA-34	33	597–604	8	24%
BA-4	146	605–743	139	95%
BA-43	26	744–753	10	38%
BA-44	56	754–809	56	100%
BA-45	58	810–866	57	98%
BA-46	46	867–912	46	100%
BA-47	217	913–1125	213	98%
BA-5	90	1126–1179	54	60%
BA-6	554	1180–1733	554	100%
BA-8	174	1734–1907	174	100%
BA-9	269	1908–2176	269	100%

Note. Total number of voxels in frontal lobe ROI = 2,176.

Correlations were calculated for the paired occurrences between the self-reported ranking of the soft skills and the AAD for the gamma asymmetry in response to the presented stimulus, for each participant. Given the ranked items are an ordinal variable, the Spearman's Rank correlation (r_s) was calculated. This statistic does not evaluate linear relationships, but rather the strength of monotonic relationships (i.e., variables which change together in the same direction, but not necessarily at a constant rate). Therefore the r_s results in a measure of directionality as well as the strength of the relationship. It is important to note, however, that the r_s does not provide a predictive measure of linearity; therefore regression lines are not drawn on the data graphs in Figures 1 and 2.

As a qualitative method of analysis, using sLORETA imaging, together with the VIDE process, a visual examination was made of the asymmetry in the frontal cortex, identifying gamma (38 to 42 Hz) bursts to assess the underlying precognitive decisions behind the self-reported responses, at the moment of decision making (Collura, Zalaquett, et al., 2014). This process theoretically provides evidence that an evoked, emotionally laden response results in corresponding brain activity and exposes the match to TriMetrix® DNA assessment-reported findings. This imaging process documents both the intensity of human emotional response as well as the directionality of the response. The process can be used, as in this study, for examining mental processes; but also has the potential for immediate open dialog with a client concerning issues that may become evident from this real-time mental imaging, as described in Collura, Bonnstetter, et al. (2014) as well as Collura, Zalaquett, et al. (2014).

Results

As shown in Table 5, when examining the top and bottom stimuli for all 14 individuals, we discovered a relationship between an individual's *approach* versus *avoidance* to a stimulus, based on the AAD and an examination of the sLORETA imaging. When the 14 participants' highest ranked personal skills are compared against their EEG responses to those same stimuli, there is a trending toward *approach* for their top five skills. This was indicated by a negative AAD score, together with an increased activation in their left frontal lobe, when examining gamma asymmetry in their frontal cortex.

Similarly, when we compared all 14 participants' five lowest ranked personal skills against their EEG responses to those same stimuli, we found an overall decrease in both their left and right frontal lobes, and AADs trending towards zero, indicating a neutral or reduced response, as if they did not recognize the stimuli/skill as being significant.

The rest of the 13 personal skills, those grouped between the top and bottom, seemed to shift or switch between *approach*, *avoidance*, or a neutral response. The response may depend on each individual's personal level of development, with a trend supporting the participants' likelihood of being emotionally more removed or disconnected from a skill, when they were moderately developed, or not developed at all in it. (The terms *moderately developed* and *not developed* come directly from the TriMetrix® DNA and are based on assessment population norms and the application of standard deviations.)

The resulting Spearman Rank correlations between the AAD and the soft skill rankings are shown in Table 6; for all cases n = 23 with df = 21. Four cases (4, 5, 6, 8) yielded, in essence, no correlations with r_s values ranging from −0.05 to 0.11. Two cases (1, 7) resulted in low correlations with r_s values of 0.29 and −0.28, respectively. Two cases (2, 3) were moderately correlated with r_s values of 0.52 and p = .011 for both. Four cases (10, 12, 13, 14) yielded strong correlations with r_s values of −0.69, 0.73, −0.69, 0.65, respectively and p values ranging from .000 to .001. Two cases (9, 11) were nearly perfect correlations with r_s values of 0.99 and 0.92, respectively and p = .000 for both. Overall, 71% (10 of 14) of the cases resulted in monotonic correlations, indicating soft skill directionality; moreover, in many of these cases, a degree of intensity reduction can be seen as they move from their top skills to their bottom. Figures 1 and 2 provide a graphical representation of each participant's AAD in response to the 23 stimuli; Figure 1 includes the 10 cases that resulted in correlations, while Figure 2 includes the four cases for which there were no correlations.

Table 5

Approach-Avoidance Differential (AAD) per Soft skill Ranked Order per Participant

Ranked Order	Participant Number													
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1st	-0.91	-1.81	-0.04	1.05	0.33	0.02	0.48	-0.19	-0.91	0.72	-1.63	-0.47	0.61	-1.10
2nd	-0.89	-1.59	-0.10	0.41	-0.07	-0.03	-0.75	0.26	-0.83	0.59	-1.42	-0.43	0.41	-0.57
3rd	-0.86	0.90	-0.02	0.30	-0.27	0.52	-0.04	0.51	-0.81	0.44	-1.25	-0.40	0.22	-0.30
4th	-0.40	-1.52	-0.10	0.22	-0.04	0.35	-0.16	0.17	-0.72	0.89	-1.61	-0.42	0.78	-0.55
5th	-0.37	-1.14	-0.04	0.38	-0.34	0.21	-0.50	0.04	-0.59	0.63	-1.22	-0.39	0.52	-0.55
6th	0.21	-1.75	-0.01	0.80	0.11	-0.25	-0.33	0.10	-0.59	0.69	-1.28	-0.32	0.58	-0.49
7th	-0.39	-0.55	0.11	0.62	0.14	0.47	0.16	0.21	-0.57	0.78	-1.35	-0.30	0.67	-0.36
8th	0.66	-0.53	0.23	0.78	-0.05	-0.08	0.42	-0.07	-0.56	0.49	-1.05	-0.29	0.38	-0.63
9th	0.58	-0.80	0.39	1.00	0.11	0.15	-0.49	0.55	-0.53	1.08	-1.61	-0.27	0.97	0.02
10th	0.47	-0.59	0.29	1.09	0.13	0.14	-1.30	-0.03	-0.49	0.46	-0.95	-0.35	0.35	-0.52
11th	0.32	-0.51	1.01	1.07	-0.33	0.28	-1.08	-0.03	-0.49	0.46	-0.95	-0.31	0.35	-0.52
12th	0.17	-0.63	0.29	1.36	0.16	-0.15	0.00	0.20	-0.46	0.66	-1.12	-0.28	0.55	-0.26
13th	0.19	-1.03	0.44	1.11	-0.09	0.22	0.48	0.05	-0.45	0.50	-0.95	-0.25	0.39	-0.40
14th	0.31	-0.84	0.39	1.74	-0.04	0.64	-0.24	0.01	-0.42	0.43	-0.85	-0.43	0.32	-0.41
15th	0.27	-0.80	0.56	1.21	-0.14	0.12	-0.56	0.34	-0.42	0.76	-1.18	-0.36	0.65	-0.08
16th	0.11	-0.47	0.52	1.16	0.41	0.15	0.12	0.02	-0.41	0.43	-0.84	-0.33	0.32	-0.39
17th	0.09	-0.83	0.26	1.27	0.29	0.14	-0.38	0.08	-0.40	0.48	-0.88	-0.28	0.37	-0.32
18th	0.10	-0.50	0.65	0.56	-0.11	0.07	-0.69	0.00	-0.39	0.39	-0.78	-0.20	0.28	-0.39
19th	0.07	-0.67	0.05	0.69	0.25	0.17	-0.59	0.22	-0.27	0.49	-0.37	-0.26	0.19	-0.05
20th	0.19	-0.45	0.15	0.55	0.02	0.27	-1.12	0.03	-0.36	0.39	-0.75	-0.21	0.28	-0.33
21st	0.21	-0.71	0.14	0.24	0.08	0.16	-0.24	0.14	-0.18	0.39	-0.57	-0.20	0.14	-0.04
22nd	0.15	-0.30	0.15	0.17	-0.07	0.46	-0.91	0.10	-0.21	0.28	-0.30	-0.19	0.12	-0.11
23rd	0.03	-0.32	0.34	0.14	0.03	-0.05	-0.13	0.11	-0.23	0.21	-0.44	-0.29	0.18	-0.12

Note. Items shaded in pink are the AADs for the five highest ranked personal skills; items shaded in blue are the AADs for the five lowest ranked personal skills.

Table 6
Spearman Rank-Order Correlations Between AAD and Soft skills Rankings (n = 23; df = 21)

Case#	r_s	*p*
1	**0.29**	.174
2	**0.52**	.011
3	**0.52**	.011
4	-0.05	.833
5	0.11	.623
6	0.05	.819
7	**-0.28**	.199
8	-0.04	.846
9	**0.99**	.000
10	**-0.69**	.000
11	**0.92**	.000
12	**0.73**	.000
13	**-0.69**	.000
14	**0.65**	.001

Note. Bold indicates presence correlation (low = 0.28 to nearly perfect 0.99).

In keeping with the single-subject design, with the 10 (71%) cases provide in Figure 1, it is important to note that each graph must be read independently because each individual enters the experience with a different brain activity baseline. For example, Figure 1, graph 1, is the same data and image set depicted in Table 7. This individual shows both a movement from the left frontal cortex to the right, as well as a reduction in gamma activity as they approach their 23rd choice of skill development. Moreover, Collura, Zalaquett, et al. (2014) posit persons may have an idiosyncratic *approach* or *avoidance* bias, to which this study may lend credence. For example, graphs 9, 11, 12 and 14, depict individuals having left hemisphere dominate gamma activity, with all negative AADs, which may be indicative of an *approach* bias as a baseline; then, show a reduction in intensity as they move from soft skills mastered to their last choice. Conversely, graphs 10 and 13 depict individuals having right hemisphere dominate gamma activity, with all positive AADs, which may be indicative of an *avoidance* bias as a baseline; and again, show intensity reduction from their first ranked item to a more neutral response to their last soft skills rankings. Here, again as suggested by Collura, Zalaquett, et al. (2014), these individuals may hold an overall bias towards a negative worldview, finding it difficult to embrace or to have a positive attitude towards much of anything. Rock (2008) describes dramatic effects that a negative baseline bias can have on perceptions, problem solving, decision making, stress management, collaboration, and even motivation. An intense avoidance baseline has been tied to an overly vigilant amygdala that results in a person being more tuned into threats than rewards. Thus the threat response is easily triggered. In fact, Baumeister and Leary (1995) explain how an avoidance baseline generates far more arousal in the limbic system than approach responses, thus leading to more intense and longer lasting effects from these perceived threats. Therefore, while Figure 1 shows directionality and, in some cases, an intensity reduction shift when examining their soft skill ranked ordering, it is important to note that each participants has a different baseline and therefore must be viewed from different baseline perspectives.

Figure 1. The AAD values are plotted for the 23 ranked order items for each participant. Included here are cases with monotonic correlations.

Figure 2 depicts 4 participants (29%) whose gamma asymmetry in response to the presented stimuli did not yield correlations with the soft skill ranking; thus, lacking directionality. As well, intensity reduction was not consistently seen. While data in these cases do show emotional expression, this expression does not occur in any predictable manner.

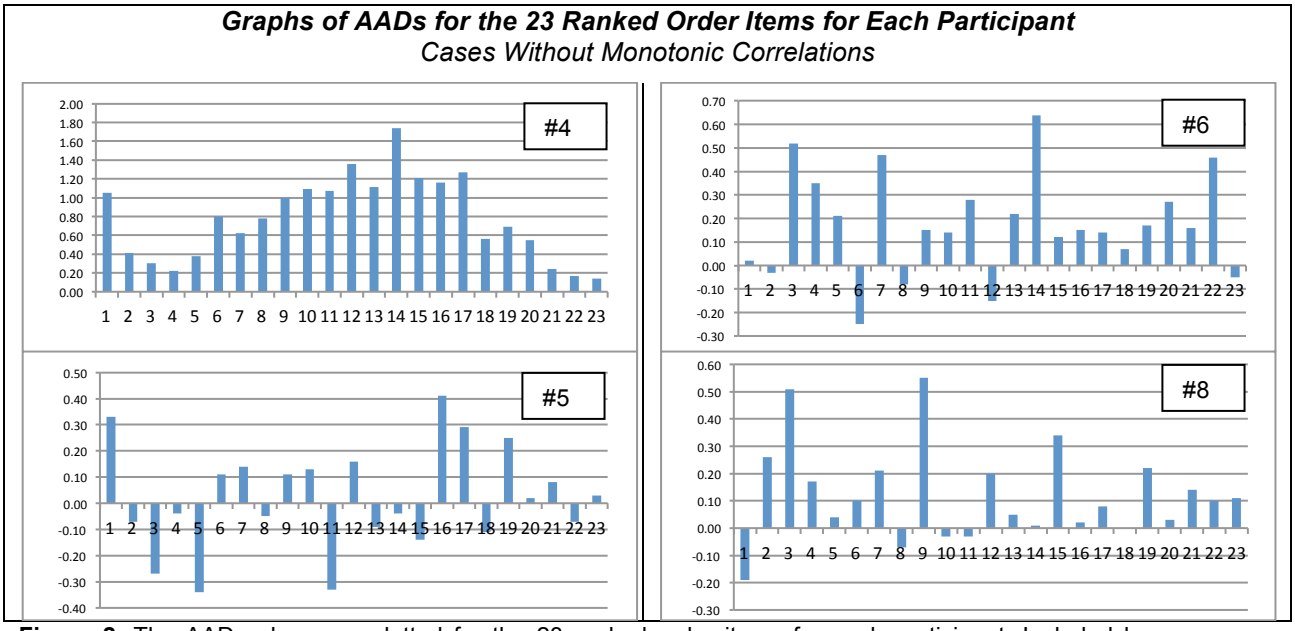


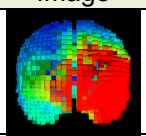
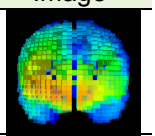
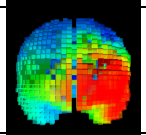
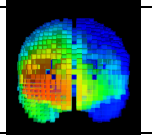
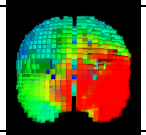
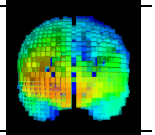
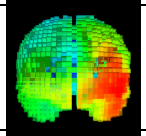
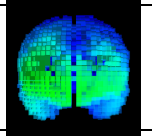
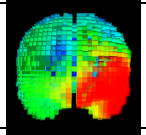
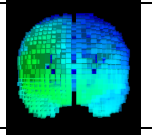
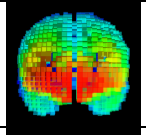
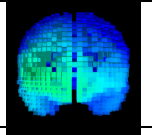
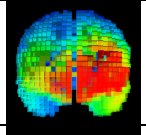
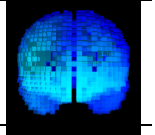
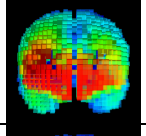
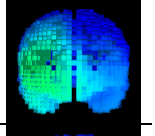
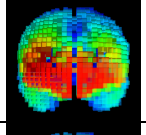
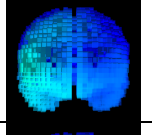
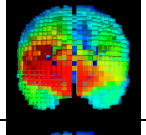
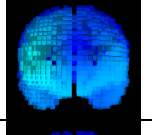
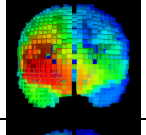
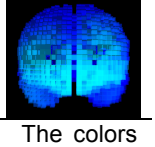
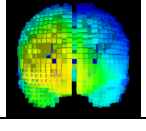
Figure 2. The AAD values are plotted for the 23 ranked order items for each participant. Included here are cases without monotonic correlations.

Single Case Illustrative Example

As an illustrative, case study example, Table 7 summarizes the graphical (qualitative) and the AAD (quantitative) results for a single participant (#1). This case also provides an example of the real-time voxel imaging available with this sLORETA implementation, using the VIDE process. The items are ranked in order of the individual's own self-rating of competence. Each item consists of the probe stimulus word (i.e., soft skill) presented, the AAD score, and the associated brain image responses shown as instantaneous activation patterns. In these images, the colors correspond to brain activity, gamma frequency; with red being the highest gamma activation and dark blue the lowest or no gamma activation. In this case, the AAD results are observed to rank in a manner associated with the individual ranking. In conjunction with these AADs, the brain images also show increased activity in the right side of the image (the client's left hemisphere) when AAD values are negative (indicating *approach* or positive response), then moving toward more right-hemisphere activity when AAD values are positive (indicating *avoidance* or negative response), as interest and self-assessment of competence decreases. Of particular note is the fact that the lowest ranked interest items seem to show an overall lack of response of any type, whether *approach* or *avoidance*, indicating an absence of response, not simply a negative response.

Table 7

Frontal Gamma Asymmetry in Response to Soft Skills Stimuli in a Single Case (Participant #1)

Rank Order	TriMetrix® DNA Soft Skill	AAD	Gamma Image	Rank Order	TriMetrix® DNA Soft Skill	AAD	Gamma Image
1	Presenting	-0.91		13	Teamwork	0.19	
2	Diplomacy	-0.89		14	Management	0.31	
3	Customer Service	-0.86		15	Conflict Management	0.27	
4	Self-Management	-0.40		16	Analytical Problem Solving	0.11	
5	Interpersonal Skills	-0.37		17	Decision Making	0.09	
6	Employee Development/ Coaching	0.21		18	Creativity/ Innovation	0.10	
7	Continuous Learning	-0.39		19	Personal Effectiveness	0.07	
8	Planning/ Organizing	0.66		20	Futuristic Thinking	0.19	
9	Persuasion	0.58		21	Leadership	0.21	
10	Written Communication	0.47		22	Negotiation	0.15	
11	Empathy	0.32		23	Flexibility	0.03	
12	Goal Orientation	0.17					

Note. AAD = Approach-Avoidance-Differential. The colors correspond to brain activity, gamma frequency; with red being the highest and dark blue the lowest. The right side of the image is the participant's left hemisphere.

Discussion

The ability to match frontal gamma activity to self-reporting assessments would represent a new approach to self-report validation. To that end, the primary goal of this pilot study was to use sLORETA to measure the degree of association between self-reported rank ordering of a set of 23 soft skills and frontal gamma asymmetry during simultaneous exposure to the same set of soft skills. In this study, when individuals were asked to make choices leading to a rank ordering, or forced choice, the brain imaging and corresponding quantitative data yielded correlations in most cases (71%; 10 of 14). However, in four cases (29%), while evidence of emotional expression was apparent, no correlations were found; thus no distinguishable pattern of directionality or intensity reduction emerged. Therefore, the degree that results of this pilot study can be generalized is limited. Yet, nonetheless, the use of sLORETA in this pilot study, together with real-time voxel imaging and the VIDE process, does present a novel approach to advancing this line of research in measuring gamma asymmetry with source localization EEG data. Further, in evaluating results on a case study basis, insights can be gained in how this data may be useful when interpreted on an individual level.

For example, in the case illustrated in Table 7, and in keeping with the model posited by Collura, Zalaquett, et al. (2014), while the individual appears to have a firm grasp on their top five soft skills, the brain precognitive responses to the remaining 17 skills may need to be discussed. For instance, it appears that the participant's precognitive position on planning/organizing, persuasion, written communication, empathy, and management tend toward *avoidance*. In other words, at a level outside of conscious awareness, this person may have deep beliefs or mindsets in place that would need to be exposed and altered before real progress toward new skill acquisition could occur. It might actually be easier to develop their worst rated skills (leadership, negotiation, and flexibility). At least these three skills appear to lack any previous emotional response and, therefore, may be easier to develop. Then, extending this perspective may also help to explain the lack of directionality and intensity reduction for the participants depicted in Figure 2. Within this context, a plausible theory may be that this group of individuals could have a poor sense of internal awareness such that it was difficult for them to provide accurate self-reports and/or rank orders of skills.

Limitations and Directions for Future Research

While this study found beginning evidence of correlations between soft skill acquisitions (as defined by a self-reporting assessment) and gamma asymmetry, further refinement of the protocols are needed to build a reliable model. The time lag between the two data collection processes is one area to refine. Efforts need to be made to capture simultaneous data while filling out the self-report questionnaire and the corresponding EEG recordings. The 2-week window between events may have added uncontrolled variables. Moreover, the impact of the stimuli presentation order needs to be further addressed.

Then, comparison to some measure of self-awareness would be of benefit, as suggested as a hypothesis for Figure 2 participants. To investigate this, we intend to embark on cross-referencing participants with an additional instrument, to determine if other variables may adversely influence asymmetry. This triangulation will involve examining internal and external dimensional balance using an assessment designed to provide insights into external factors, including understanding of others, practical thinking, and system judgment, as well as internal factors including sense of self, role awareness, and self direction. Just as it is crucial to collect demographic and background histories, which may impact a study, we may have uncovered the need to document a person's ability to understand self in order to accurately assess precognitions.

Another variable that needs better control is the fact that within the population studied a wide range of behavioral styles and motivational factors were identified along with the soft skill data. Moreover, gender differences should be a future area of focus. Because of the limited number of participants, analysis of these subgroup factors was not possible. We may find that different behavioral styles react differently to soft skill acquisition. Thus, replication with larger sample sizes is needed.

Revisiting the research design in light of the limitations stated above represents the primary directions for future research. More so, further statistical validation of these methods is necessary for furthering this line of research. To take full advantage of the benefits of sLORETA source localization, isolating and investigating asymmetries in the most relevant BAs included in the frontal lobe ROI, as well as other frequency bands, may prove advantageous. However, administering these protocols in real world contexts, such as during

coaching sessions, job interviews, and possibly even in psychotherapeutic milieus (given proper ethical constraints), are promising areas for additional study, to evaluate the impact of potentially exposing hidden decision-making mechanisms of the preconscious mind.

Conclusion

The use of sLORETA real-time EEG data acquisition, and imaging, to investigate frontal gamma asymmetry in response to soft skill stimuli is innovative in its methodology as a new approach to the original work of Davidson, Pizzagalli, and other researchers. The correlation of self-reports' skill set by corresponding sLORETA brain imaging is encouraging; but more surprising, was the lack of brain response to those skills not possessed (i.e., the neutral responses). This may present beginning evidence of skills being experience-based and support a position that, in the lack of exposure to a skill, an individual may simply not have an emotional or memory connection to trigger a measurable brain-activation reaction. Nonetheless, it is important to remain mindful this pattern was found in 71% of participants; thus, concrete implications may be limited.

While much work remains, these results will inform further research, which has potential to lead to a means of validating survey results and at the same time exposing attempts at assessment manipulation. In spite of the exploratory nature of this study, advancing this line of research is warranted to investigate potential brain-based measures, which can objectively validate self-reported responses, and thereby provide meaningful indicators of emotional, cognitive, and motivational states, thus establishing advantageous tools in the field of organizational neuroscience.

References

Arthurs, O. J., & Boniface, S. (2002). How well do we understand the neural origins of the fMRI BOLD signal? *Trends in Neurosciences, 25*(1), 27–31. http://dx.doi.org/10.1016/S0166-2236(00)01995-0

Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychological Bulletin, 117*(3), 497–529. http://dx.doi.org/10.1037/0033-2909.117.3.497

Bedwell, W. L., Fiore, S. M., & Salas, E. (January, 2011). *Developing the 21st century (and beyond) workforce: A review of interpersonal skills and measurement strategies*. Paper presented at the meeting of The National Academies, Board on Testing and Assessment and The National Academy of Education, Irvine, CA. http://www.eastbaycharterconnect.org/uploads/7/1/7/6/71762220/developing_the_21st_century_and_beyond_workforce-_a_review_of_interpersonal_skills__measurement_strategies_bedwell_et_al._2011.pdf

Bedwell, W. L., Fiore, S. M., & Salas, E. (2014). Developing the future workforce: An approach for integrating interpersonal skills in the MBA classroom. *Academy of Management Learning and Education, 13*(2), 171–186. http://dx.doi.org/10.5465/amle.2011.0138

Bonnstetter, B. J., Bonnstetter, R. J., Hebets, D., & Collura, T. F. (2015). *U.S. Patent No. 9,060,702*. Washington, DC: U.S. Patent and Trademark Office.

Bonnstetter, R. J., Collura, T. F., Hebets, D., & Bonnstetter, B. J. (2012, Winter) Uncovering the belief behind the action. *NeuroConnections,* 20–23.

Collura, T. F, Bonnstetter, R. J., & Zalaquett, C. (2014). Neurocounseling: Bridging Brain and Behavior. Seeing inside the client's mind. *Counseling Today, 57*(6), 24-27.

Collura, T. F., Zalaquett, C. P., Bonnstetter, R. J., & Chatters, S. J. (2014). Toward an operational model of decision making, emotional regulation, and mental health impact. *Advances in Mind-Body Medicine, 28*(4), 18–33.

Davidson, R. J. (1992). Emotion and affective style: Hemispheric substrates. *Psychological Science, 3*(1), 39–43. http://dx.doi.org/10.1111/j.1467-9280.1992.tb00254.x

Davidson, R. J. (2000). Affective style, psychopathology, and resilience: Brain mechanisms and plasticity. *American Psychologist, 55*(11), 1196–1214. http://dx.doi.org/10.1037/0003-066X.55.11.1196

Davidson, R. J. (2002). Anxiety and affective style: Role of prefrontal cortex and amygdala. *Biological Psychiatry, 51*(1), 68–80. http://dx.doi.org/10.1016/S0006-3223(01)01328-2

Davidson, R. J. (2004). What does the prefrontal cortex "do" in affect: perspectives on frontal EEG asymmetry research. *Biological Psychology, 67*(1–2), 219–234. http://dx.doi.org/10.1016/j.biopsycho.2004.03.008

Davidson, R. J., Ekman, P., Saron, C. D., Senulis, J. A., & Friesen, W. V. (1990). Approach-withdrawal and cerebral asymmetry: Emotional expression and brain physiology. *Journal of Personality and Social Psychology, 58*(2), 330–341. http://dx.doi.org/10.1037/0022-3514.58.2.330

Davidson, R. J., Pizzagalli, D., Nitschke, J. B., & Kalin, N. H. (2003). Parsing the subcomponents of emotion and disorders of emotion: Perspectives from affective neuroscience. In R. J. Davidson, H. H. Goldsmith, K. Scherer (Eds.), *Handbook of Affective Sciences* (pp. 8–24). New York: Oxford University Press.

Davidson, R. J., Schwartz, G. E., Saron, C., Bennett, J., & Goleman, D. J. (1979). Frontal versus parietal EEG asymmetry during positive and negative affect. *Psychophysiology, 16,* 202–203.

Dweck, C. S. (2006). *Mindset: The new psychology of success*. New York, NY: Ballantine Books.

Gordon, E., Barnett, K. J., Cooper, N. J., Tran, N., & Williams, L. M. (2008). An "Integrative Neuroscience" platform: Application to profiles of negativity and positivity bias. *Journal of Integrative Neuroscience, 7*(3), 345–366. http://dx.doi.org/10.1142/S0219635208001927

Gosselin, D., Cooper, S., Bonnstetter, R. J., & Bonnstetter, B. J. (2013). Exploring the assessment of 21st century professional competencies of undergraduate students in environmental studies through a business-academic partnership. *Journal of Environmental Studies and Sciences, 3*(3), 359–368. http://dx.doi.org/10.1007/s13412-013-0140-1

Gosselin, D., Cooper, S., Bonnstetter, R. J, & Bonnstetter, B. J. (2014). Erratum to: Exploring the assessment of 21st century professional competencies of undergraduate students in environmental studies through a business-academic partnership. *Journal of Environmental Studies and Sciences, 4*(2), 188–189. http://dx.doi.org/10.1007/s13412-014-0164-1

Harmon-Jones, E. (2004). Contributions from research on anger and cognitive dissonance to understanding the motivational functions of asymmetrical frontal brain activity. *Biological Psychology, 67(*1–2), 51–76.

http://dx.doi.org/10.1016/j.biopsycho.2004.03.003
Hüsing, B., Jäncke, L., & Tag, B. (2006). *Impact Assessment Of Neuroimaging, Final report*. Zürich: vdf Hochschulverlag.
Kanai, R., & Rees, G. (2011). The structural basis of inter-individual differences in human behaviour and cognition. *Nature Reviews Neuroscience, 12*(4), 231–242. http://dx.doi.org/10.1038/nrn3000
Lafferty, C. L., & Alford, K. L. (2010). Neuroleadership: Sustaining research relevance into the 21st century. *SAM Advanced Management Journal, 75*(3), 32–40.
McClelland, D. (1961). *Human motivation*. Cambridge, UK: Cambridge University Press.
Mitchell, J. P. (2008). Contributions of functional neuroimaging to the study of social cognition. *Current Directions in Psychological Science, 17*(2), 142–146. http://dx.doi.org/10.1111/j.1467-8721.2008.00564.x
Naccache, L., Gaillard, R., Adam, C., Hasboun, D., Clémenceau, S., Baulac, M., … Cohen, L. (2005). A direct intracranial record of emotions evoked by subliminal words. *Proceedings of the National Academy of Sciences of the United States of America, 102*(21), 7713–7717. http://dx.doi.org/10.1073/pnas.0500542102
National Research Council. (2008). Research on future skill demands: A workshop summary. In M. L. Hilton & Rapporteur (Eds.), *Planning Committee on Research Evidence Related to Future Skill Demands. Center for Education. Division of Behavioral and Social Sciences and Education*. Washington, DC: The National Academies Press.
National Research Council. (2012). Education for Life and Work: Developing Transferable Knowledge and Skills in the 21st Century. Committee on Defining Deeper Learning and 21st Century Skills. In J. W. Pellegrino & M. L. Hilton, (Eds.), *Board on Testing and Assessment and Board on Science Education, Division of Behavioral and Social Sciences and Education*. Washington, DC: The National Academies Press.
Nitschke, J. B., Heller, W., Etienne, M. A., & Miller, G. A. (2004). Prefrontal cortex activity differentiates processes affecting memory in depression. *Biological Psychology, 67(*1–2), 125–143. http://dx.doi.org/10.1016/j.biopsycho.2004.03.004
Oakes, T. R., Pizzagalli, D. A., Hendrick, A. M., Horras, K. A., Larson, C. L., Abercrombie, H. C., … Davidson, R. J. (2004). Functional coupling of simultaneous electrical and metabolic activity in the human brain. *Human Brain Mapping, 21*(4), 257–270. http://dx.doi.org/10.1002/hbm.20004
Pascual-Marqui, R. D. (2002). Standardized low-resolution brain electromagnetic tomography (sLORETA): Technical details. *Methods and Findings in Experimental and Clinical Pharmacology, 24*(Suppl. D), 5–12.
Pizzagalli, D. A., Greischar, L. L., & Davidson, R. J. (2003). Spatio-temporal dynamics of brain mechanisms in aversive classical conditioning: high-density event-related potential and brain electrical tomography analyses. *Neuropsychologia, 41*(2), 184–194. http://dx.doi.org/10.1016/S0028-3932(02)00148-3
Pizzagalli, D. A., Nitschke, J. B., Oakes, T. R., Hendrick, A. M., Horras, K. A., Larson, C. L., … Davidson, R. J. (2002). Brain electrical tomography in depression: the importance of symptom severity, anxiety, and melancholic features. *Biological Psychiatry, 52*(2), 73–85. http://dx.doi.org/10.1016/S0006-3223(02)01313-6
Pizzagalli, D., Pascual-Marqui, R. D., Nitschke, J. B., Oakes, T. R., Larson, C. L., Abercrombie, H. C., … Davidson, R. J. (2001). Anterior cingulate activity as a predictor of degree of treatment response in major depression: Evidence from brain electrical tomography analysis. *The American Journal of Psychiatry 158*(3), 405–415. http://dx.doi.org/10.1176/appi.ajp.158.3.405
Rock, D. (2008). SCARF: a brain-based model for collaborating with and influencing others. *NeuroLeadership, 1*, 1–9.
Rolls, E. T. (1999). *The Brain and Emotion*. New York: Oxford University Press.
Tabibnia, G., Satpute, A. B., & Lieberman, M. D. (2008). The sunny side of fairness: Preference for fairness activates reward circuitry (and disregarding unfairness activates self-control circuitry). *Psychological Science, 19*(4), 339–347. http://dx.doi.org/10.1111/j.1467-9280.2008.02091.x
Thatcher, R. W. (2013). Latest developments in live *z*-score training: Symptom check list, phase reset, and LORETA *z*-score biofeedback. *Journal of Neurotherapy, 17*(1), 69–87. http://dx.doi.org/10.1080/10874208.2013.759032
The KEY Institute for Brain-Mind Research. (2014). Zürich, Switzerland. http://www.uzh.ch/keyinst/
Waldman, D. A., Balthazard, P. A., & Peterson, S. J. (2011). Leadership and Neuroscience: Can We Revolutionize the Way That Inspirational Leaders Are Identified and Developed? *Academy Of Management Perspectives*, *25*(1), 60–74. http://dx.doi.org/10.5465/AMP.2011.59198450
Yeager, D. S., & Walton, G. M. (2011). Social-psychological interventions in education: They're not magic. *Review of Educational Research, 81*(2), 267–301. http://dx.doi.org/10.3102/0034654311405999

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NeuroRegulation

EEG Neurofeedback in the Treatment of Chronic Pain: A Case Series

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Abstract

Neurofeedback has been used to treat a variety of problems and symptoms related to central nervous system dysregulation, including chronic pain conditions. However, there is limited published work describing the application and efficacy of neurofeedback for chronic pain. This case series describes the outcomes of neurofeedback treatment of four patients with diverse diagnoses and pain symptoms. Although there was variability in patient response, all patients reported improvements in pain and other symptoms with treatment. The findings indicate that more research to (1) clarify the benefits of neurofeedback for different conditions and (2) identify the most effective protocols for individual patients is warranted.

Keywords: Neurofeedback; chronic pain; migraine; gastrointestinal pain; testicular pain

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Introduction

Neurofeedback procedures, by which individuals are trained, through operant conditioning, to increase or inhibit the magnitude ("power") of oscillations in specific bandwidths as a way to self-regulate brain activity, have been used to improve arousal, alertness, emotional control, and symptom expression (Othmer, Othmer, & Kaiser, 1999); symptoms of attention deficit disorder (Arns, de Ridder, Strehl, Breteler, & Coenen, 2009; Linden, Habib, & Radojevic, 1996; Lubar, 1991; Monastra, Monastra, & George, 2002); learning and cognitive functioning (Albert, Andrasik, Moore, & Dunn, 1998; Cunningham & Murphy, 1981; Egner & Gruzelier, 2003; Fenger, 1995; Gruzelier, Egner, & Vernon, 2006; Murphy, Darwin, & Murphy, 1977; Nall, 1973; Rasey, Lubar, McIntyre, Zoffuto, & Abbott, 1996; Thompson & Thompson, 1998; Vernon et al., 2003; Whisenant & Murphy, 1977); and artistic performance (Gruzelier, 2009).

One class of symptoms to which neurofeedback has been applied is chronic pain (Jensen, Sherlin, Hakimian, & Fregnia, 2009). Chronic pain is defined with respect to acute pain by the National Institute of Neurological Disorders and Stroke (NINDS) of the National Institutes of Health (2015) as follows:

> While acute pain is a normal sensation triggered in the nervous system to alert you to possible injury and the need to take care of yourself, chronic pain is different. Chronic pain persists. Pain signals keep firing in the nervous system for weeks, months, even years. There may have been an initial mishap—sprained back, serious infection, or there may be an ongoing cause of pain—arthritis, cancer, ear infection, but some people suffer chronic pain in the absence of any past injury or evidence of body damage (para. 1).

Although early theories of chronic pain focused more on the periphery as the site of presumed physical damage that is likely "causing" pain, current models of chronic pain acknowledge the critical role that the brain plays in creating the pain experience. As a result, there has been an increased interest in

treatments that might impact pain via their direct effects on brain activity, including neurofeedback.

Consistent with the possibility that neurofeedback could benefit individuals with chronic pain, a growing body of evidence supports pain perception as being mediated by structures and neural networks in the brain and as being influenced by multiple, interactive neural processes that modulate pain information at many levels, including the cortex. Multiple cortical sites have been identified as involved, including the somatosensory cortex, the insular cortex, the anterior cingulate, the prefrontal cortex, and thalamic nuclei (Apkarian, Bushnell, Treede, & Zubeita, 2005; Babiloni et al., 2003; Chen, 2001; Craig, 2003a, 2003b; DeCharmes et al., 2005; DeLeo, 2006; Jensen, 2010; Katz & Rothenberg, 2005; Melzack, Coderre, Katz, & Vaccarino, 2001; Miltner & Weiss, 1998; Peyron, Laurent, & Garcia-Larrea, 2000; Rainville, Duncan, Price, Carrier, & Bushnell, 1997; Tinazzi, Fiaschi, Rosso, Faccioli, Grosslercher, & Aglioti, 2000). Furthermore, there is evidence of neural adaptation to pain stimuli, indicating neural plasticity (Flor, 2003; Katz & Melzack, 1990). Consistent with this idea, repeated exposure to painful stimuli has been shown to increase one's sensitivity to stimulation and, therefore, to the tendency to interpret stimulation that may not be damaging as "pain," contributing to chronicity (Bromm & Lorenz, 1998). The pain circuitry in the brain also overlaps the circuitry involved in depression, providing further evidence of central nervous system involvement in pain modulation (Lindsay & Wyckoff, 1981).

A number of studies have identified specific frequencies of brain wave activity that are associated with pain. For example, the presence of more activity in the alpha frequency range (8–13 Hz) is known to be associated with, and to reflect a general inhibition of, cognitive activity and central nervous system (CNS) processing (Pfurtscheller, 2003); hence, lower alpha activity has been associated with increased pain perception and higher alpha activity with decreased pain perception (Babiloni et al., 2008; Nishigami, Nakano, Osumi, Tsujishita, Mibu, & Ushida, 2014). Because neurons and neuronal ensembles that fire in the alpha frequency tend to inhibit activity in "downstream" neurons that are influenced by these ensembles, lower alpha power is associated with more information processing, including information that is transferred through thalamo-cortical and cortico-cortical channels. Suppression of the alpha rhythm could therefore allow for more processing of nociceptive input from the periphery. Consistent with this idea, suppression of alpha power (also known as alpha event-related desynchronization) has been found to occur in the primary somatosensory cortex in anticipation of aversive or painful electrical stimulus (Burroughs, 2011). Lower alpha activity has also been implicated in the perception of pain accompanying sensorimotor incongruent information (Nishigami et al., 2014) and in the anticipation of painful motor stimuli (Babiloni et al., 2008). Thus, it is reasonable to hypothesize that the stronger the magnitude of event-related desynchronization of alpha in anticipation of pain, the greater the subjectively rated experience of pain. In addition, research has shown that individuals with chronic pain exhibit increased beta and decreased alpha CNS activity, with additional increased theta activity in individuals with spinal cord injury and chronic pain (Jensen et al., 2009).

Consistent with this research linking brain oscillation patterns to the severity of pain, a number of studies have demonstrated improvements in pain sensation/perception in various pain conditions following neurofeedback training (Jensen et al., 2009; Prinsloo, Gabel, Lyle, & Cohen, 2012). However, there are important gaps in our knowledge regarding the potential of neurofeedback for treating pain. For example, there is not yet a sufficient empirical or theoretical basis for deriving protocols from a common understanding of the specific brain oscillations to reward and inhibit. We also do not yet know if the protocols that can be derived from the limited understanding we do have would be equally effective across different pain conditions. Moreover, questions remain regarding the adaptability of such protocols to the realities of an outpatient clinical practice, in which there is less an emphasis on standardization than on the ability to modify the treatment approach in response to clinical considerations, such as client response. Such tailoring of training protocols has been a recent focus for researchers (Arns, Conners, & Kraemer, 2013; Arns, Heinrich, & Strehl, 2014; Escolano, Navarro-Gil, Garcia-Campayo, Congedo, & Minguez, 2014; Lansbergen, van Dongen-Boomsma, Buitelaar, & Slaats-Willemse, 2011; Logemann, Lansbergen, Van Os, Böcker, & Kenemans, 2010).

Given these considerations, the aim of this paper is to provide additional information regarding how neurofeedback training might be used for treating individuals with chronic pain in the context of an active clinical practice. This information could be useful to clinicians by providing them with specific procedures and treatment protocols to consider in

patients they treat with chronic pain. The information could also be useful to researchers by providing them with specific treatment protocols to evaluate in future clinical trials.

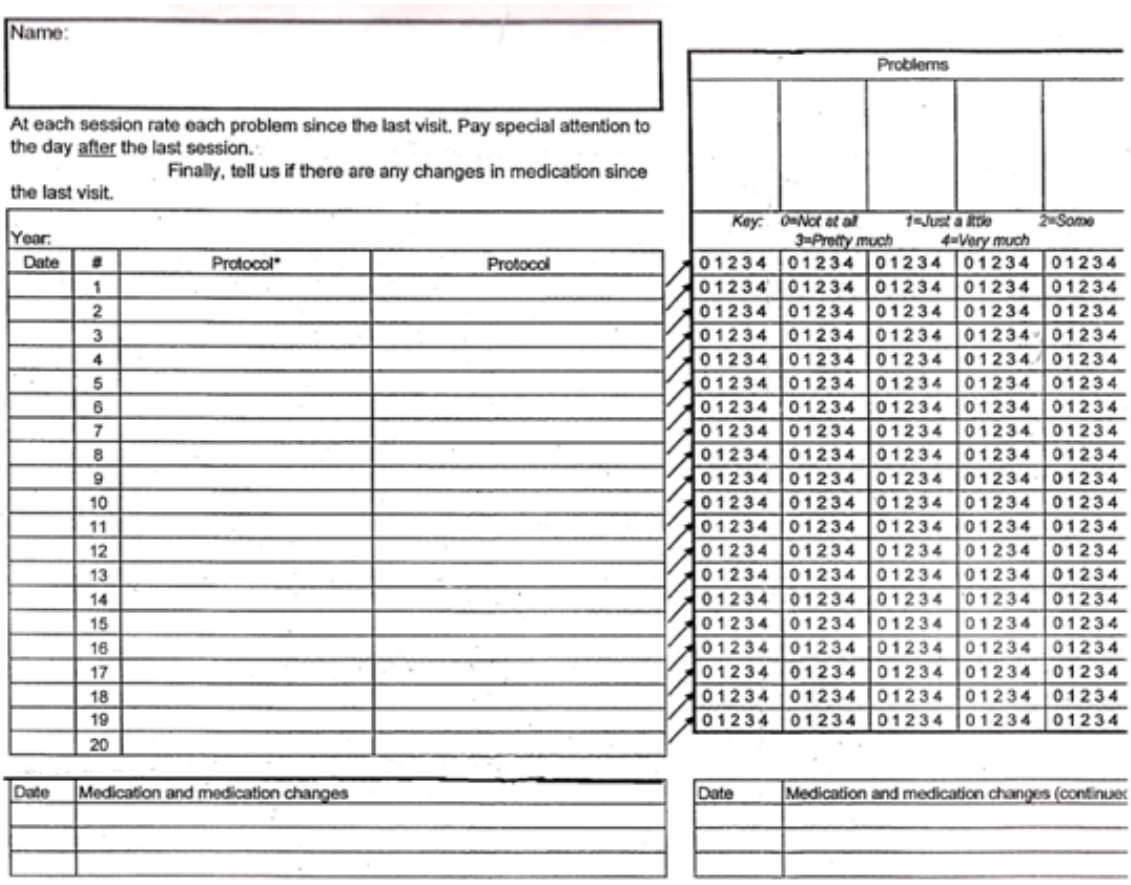
Methods

Patient selection

The subjects of this case series were four consecutive patients in the outpatient practice of the first author who met the following criteria: (1) presenting with a primary complaint of chronic pain, (2) not presenting with severe psychological problems other than anxiety and/or depression. Thus, the participants were not selected based on their response to treatment. The patients were screened using a comprehensive intake interview without the use of standardized psychological measures.

Intake interviews

Each patient first underwent an initial 45-min intake interview in which the patient's presenting problems, current functioning, and psychosocial and medical histories were taken. This was followed by a second intake session, which consisted of a 45-min introduction to the equipment and procedures of the neurofeedback training. Baseline levels of the outcome variables were assessed during the second session, and monitored throughout treatment, using a Neurofeedback Progress Chart (NPC; Matthew Fleischman, PhD, The Center for Attention and Learning, Eugene, OR) as shown in Figure 1. With the NPC, patients are told to describe each problem or symptom they would like to see improve with treatment, and then asked to rate the severity of each one by circling a number on a 0–4 Likert scale (0 = *Not at all a problem* to 4 = *Very much a problem*). They were also told that they could place a circle between the numbers, which essentially turned the measure into a 9-point scale.


Name:

At each session rate each problem since the last visit. Pay special attention to the day after the last session.

Finally, tell us if there are any changes in medication since the last visit.

Year:

Problems

Key: 0=Not at all 1=Just a little 2=Some 3=Pretty much 4=Very much

Date	#	Protocol*	Protocol	01234	01234	01234	01234	01234
	1			01234	01234	01234	01234	01234
	2			01234	01234	01234	01234	01234
	3			01234	01234	01234	01234	01234
	4			01234	01234	01234	01234	01234
	5			01234	01234	01234	01234	01234
	6			01234	01234	01234	01234	01234
	7			01234	01234	01234	01234	01234
	8			01234	01234	01234	01234	01234
	9			01234	01234	01234	01234	01234
	10			01234	01234	01234	01234	01234
	11			01234	01234	01234	01234	01234
	12			01234	01234	01234	01234	01234
	13			01234	01234	01234	01234	01234
	14			01234	01234	01234	01234	01234
	15			01234	01234	01234	01234	01234
	16			01234	01234	01234	01234	01234
	17			01234	01234	01234	01234	01234
	18			01234	01234	01234	01234	01234
	19			01234	01234	01234	01234	01234
	20			01234	01234	01234	01234	01234

Date	Medication and medication changes

Date	Medication and medication changes (continued)

Figure 1. Neurofeedback Progress Chart.

Equipment

Training was conducted using software developed by EEG Spectrum International (later called EEG Education and Research; Granada Hills, CA) version 4.30. The software was run on a single computer–dual monitor system, with one Dell Inspiron 620, i5-2310, 2.90 GHz, 64-bit desktop computer and two Optiquest monitors. The signal was processed with a Thought Technology ProComp+ (Quebec, Canada) amplifier, and 16" silver electrodes were applied to the scalp and ears with a preparation of NuPrep gel and Ten20-Conductive paste with an impedance of $\leq$ 20 kohm. The patient received visual and auditory feedback through the display of a "game" interface. The visual and auditory feedback was responsive to the patient simultaneously increasing the microvoltage reading of a specified frequency band of brain oscillation activity ("reward band") and decreasing the microvoltage reading of two other specified frequencies of brain wave activity ("inhibit bands"), above or below their respective thresholds, which were programmed into the computer by the clinician before each treatment session. The thresholds were also adjusted at times during each treatment session to increase the level of difficulty for the patient when the patient's success resulted in the training being much easier than previously.

Training procedure

At the beginning of each neurofeedback training session, beginning with the second session, the patient was presented with the NPC and instructed to: "…*complete the ratings for each symptom or problem based on how much of a problem it has been for you since the last session, with particular emphasis on the day or two immediately after the last session*."

Electrodes were placed on the patient's scalp and earlobes after prepping the skin. Electrode placement was made according to the standard 10-10 electrode placement system in which electrode sites are identified with a letter and a number (see Figure 2).

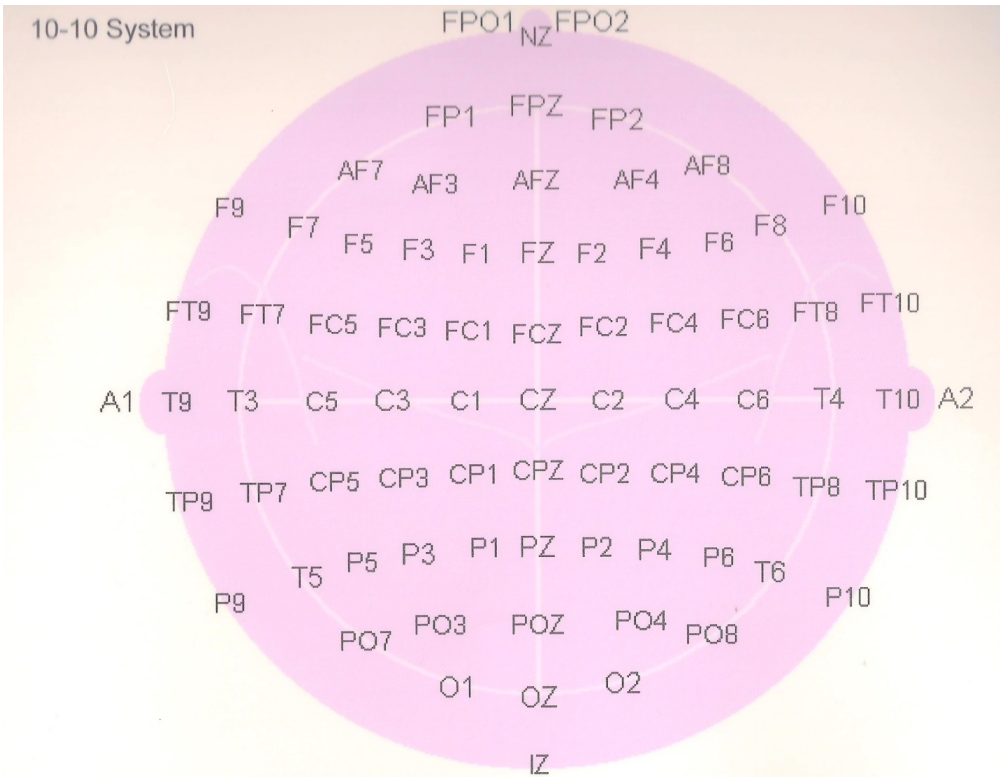


Figure 2. 10-10 Electrode Placement System.

Three electrodes were placed for each protocol: one for the active training site, one for the reference site, and one for ground. The first site was the active site and always involved placement somewhere on the scalp over a cortical region that was the target of training. The second (reference) electrode was placed either at another scalp location (in bipolar training) or on an earlobe (in unipolar training). The third electrode was placed on what was considered an electroencephalography (EEG) neutral site, such as an earlobe, as a ground for the amplifier. Placements are identified by the active site followed by the reference (e.g., C4-A2). All treatment protocols were determined by the clinician and at the time of patient presentation or during the course of treatment. Given the authors' interest in determining the efficacy of electrode sites and bandwidths cited in the literature (e.g., Jensen et al., 2009), treatment protocols were based on this literature rather than on quantitative EEG (qEEG) findings.

During treatment, the patients were seated upright in a chair facing the computer monitor, which displayed the game or the interface. The therapist sat on the patient's left, facing the screen, which displayed the patient's EEG activity. The patient was instructed to "*Sit comfortably, relax and focus."* If the patient asked what he or she was supposed to "do" to play the game, he or she was reassured using words similar to the following: "*Just relax all of your muscles, relax your jaw and your neck, and get into a state of relaxed focus. Your job is to* [language inserted appropriate to what would happen if the patient earned points in the game, such as *'…keep the circle moving through the maze eating dots*']. Y*our brain will learn from the feedback*."

For all patients, the thresholds were set to begin at 20-60-15, where 20 referred to the percentage of time that the microvoltage of the lower frequency inhibit band exceeded the threshold, making the patient ineligible for a reward (visual or auditory), 60 referred to the percentage of time that the microvoltage of the reward band exceeded the threshold, making the patient eligible for a reward, and 15 referred to the percentage of time that the microvoltage of the lower frequency inhibit band exceeded the threshold, making the patient ineligible for a reward. In order for the patient to receive a visual and auditory reward, all three threshold criteria had to be met simultaneously. The thresholds were adjusted throughout the training to control for the level of difficulty of reaching criteria for reward in order to balance the need for an appropriate level of challenge (i.e., not make meeting the training criterion too easy, reducing the opportunity for learning) with the need to make meeting the training criterion possible (i.e., to limit anxiety and frustration). This was a clinical decision made individually for each patient.

When a patient sits down to begin a neurofeedback training session, there is often a shift in the amplitude of the EEG activity. In order to start the training session at the appropriate level of difficulty (with the first inhibit band starting out at 20% above threshold, the reward band at 60% above threshold, and the second inhibit band at 15% above threshold), the therapist autothresholded the computer about 30 seconds after the patient first began training. The therapist remained in the room for the entire session, occasionally (but conservatively) adjusting the thresholds. Each training session lasted for 30 min, with at least 18 of those minutes spent in actual neurofeedback training.

Results

Case 1

Presenting problem

A 19-year-old female high school senior presented with chronic abdominal pain, which she described as "sharp," "pulsing," and "aching." On a 1–10 pain scale, where 1 represented *No pain*, and 10 represented *The worst pain you have ever experienced*, she rated her typical daily pain at an 8. In addition to severe pain, she complained of depressed mood, excessive sleep, and diminished energy and motivation. The patient was a high school senior and, for the week prior to her initial appointment, she had been out of school the entire week. She did not believe that she would feel well enough to attend the end of senior year social activities that had been planned.

History

When the patient was 13-years-old, she became ill with gastrointestinal symptoms—gastrointestinal pain and diarrhea—while at an overnight summer camp. Her symptoms persisted into the fall of that year, and she missed a large portion of 8th grade. At age 14, she experienced painful menstruation secondary to endometriosis and was prescribed Lupron and Prempro. At age 15, after swimming in a lake in the summer, she contracted a waterborne parasite (microsporidium) and developed stomach pain and diarrhea. These symptoms never resolved. Starting in the 8th grade, and continuing to the time she began treatment, the patient often missed weeks of school at a time. She also described a long history of anxiety and depression associated

with the pain, and reported that, when these psychological symptoms became worse, so did her pain.

The patient had been treated at the Mayo Clinic and at Massachusetts General Hospital. She had been prescribed paroxetine, sertraline, escitalopram oxalate, duloxetine hydrochloride, and pregabalin, the latter two of which she was taking at the time of treatment. She also took melatonin to help her sleep. She had seen a therapist for cognitive-behavior therapy to help her cope with the pain. A neuropsychological evaluation in 2009 found her to be of average to above average intelligence and also diagnosed her with Attention-deficit/Hyperactivity Disorder (ADHD). Prior to treatment, the patient completed the NPC and identified the following two areas of concern: Pain (rated as a 4 at pretreatment) and depression (rated as a 3).

Course of treatment

The patient was then seen for 41 neurofeedback sessions over an 8-month period. She was seen twice weekly for approximately 16 weeks, once every 2 weeks for approximately 12 weeks after that, and then once a month for approximately 9 weeks.

For sessions 1–3, a single channel, bipolar protocol was used with a ground to the right earlobe (A2). The bipolar protocol was T3-T4 with the active electrode at T3 and the reference electrode at T4. This protocol was chosen because of clinical reports of its effectiveness in emotional stabilization (Othmer, 2005). Various 3 Hz-wide reward bands between 9–12 Hz (alpha) and 12–15 Hz (low beta) were used, out of interest in determining if a particular reward band would yield a fast, positive response. The inhibits used were 2–7 Hz (theta) and 22–32 Hz (fast beta). At the end of the three sessions, the symptom ratings remained the same. In the clinician's experience, patients often experience some subjective sense of improved well-being during the session at some frequency setting with this protocol. Because the patient did not experience any subjective improvements and was in a good deal of distress, the decision was made to switch to a different protocol in order to determine if the patient could experience some immediate relief.

Sessions 4–5 were conducted using a single channel, unipolar protocol, with the electrode either at CZ or FZ, with the reference electrode on the left earlobe (A1) and the ground on the right earlobe (A2). The CZ and FZ sites were chosen for two reasons. First, they are over the anterior cingulate cortex, known to be important in the affective experience of pain (Rainville et al., 1997). Second, research has indicated that training at these sites is effective for reducing inattention symptoms in individuals who have a diagnosis of ADHD (Arns et al., 2009). The reward frequencies varied within the beta range in 3 Hz-wide bands, ranging from 12–15 Hz to 20–23 Hz, in order to find a frequency which felt most comfortable to her, particularly one in which she felt most focused, with the inhibits at 4–7 Hz and 22–32 Hz. No changes in the problem symptoms were reported following this protocol.

Sessions 6–10 were conducted with the unipolar placement at FZ-A1, with a reward of 8–13 Hz, and a second, simultaneous, reward in the beta band, ranging between 12–15 Hz and 21–24 Hz. The alpha reward band was expanded by 1 Hz in both directions in order to capture more of the alpha spectrum. The inhibits remained at 4–7 Hz and 22–32 Hz during these sessions. Significant improvement was noted in both symptoms following these sessions, with pain reducing to a 2 and depression to a 1.

Given these improvements, training at FZ-A1 continued for sessions 11–18, with rewards at 8–13 Hz and 12–15 Hz, and inhibits at 4–7 Hz and 22–32 Hz. The 12–15 Hz reward was added because of aforementioned research indicating that enhancements of alpha and slow beta frequencies could be effective in chronic pain treatment. Following these sessions, the previous reduction was maintained at a 2 and depression dropped further to a 0.

For sessions 19–20, an attempt was made to train posteriorly, because pain had not improved as much as depression had, and because posterior training was thought to be consistent with the somatosensory nature of the pain. Four studies have reported positive outcomes with training that included occipital electrode placements (Andreychuk & Skriver, 1975; Cohen, McArthur, & Rickles, 1980; Gannon & Sternbach, 1971; Melzack & Perry, 1975) and two reported positive outcomes with training that included parietal placements (Cohen et al., 1980; Jensen, Grierson, Tracy-Smith, Bacigalupi, & Othmer, 2007). A unipolar O2-A2 protocol rewarding 8–13 Hz and inhibiting 4–7 Hz and 22–32 Hz was used first, followed by a unipolar P4-A2 protocol with the same rewards and inhibits. Following these two training sessions, the patient reported a worsening of her symptoms, with pain increasing to a 3 and depression to a 4.

In an effort to return to the previous levels of improvement, the decision was made to return to training at the FZ-A1 site for session 21, with the rewards at 8–13 Hz and 12–15 Hz, in order to separately reinforce alpha and slow beta activity, and the inhibits at 2–7 Hz and 22–32 Hz. The decision to broaden the band of the slow wave inhibit was made because a significant amount of activity was observed by visual analysis in that part of the spectrum. The pain rating remained at 3. The decision was made to switch to unipolar training at C4-A2 in order to see if using a protocol that has been linked to calming rather than activating the nervous system might be effective. Sessions 22–29 were conducted at that site, with rewards being 8–13 Hz and 12–15 Hz, and inhibits being at 2–7 Hz and 22–32 Hz. The symptom ratings improved back to their previous lower levels, with pain rated as a 2 and depression a 0.

With the previous level of symptom improvement reestablished, sessions 30–33 employed a two-channel set up at the following sites: F1-A1 and F2-A2, as suggested in Jensen et al. (2009), based on research indicating a role for the dorsal anterior cingulate in the affective experience of pain (Rainville et al., 1997). This was conducted with a reward band of 8–13 Hz and inhibits at 4–7 Hz and 15–32 Hz, setting a broader fast frequency inhibit in order to address possible overarousal problems that might be contributing to the patient's pain symptoms. Following these sessions, the pain rating reduced further (to a 1) and the low level of depression (at a 0) maintained.

Because of the improvement in pain intensity, we continued with the same training sites for the final six sessions (sessions 34–41), with one change in the protocol: the slow wave inhibit band was further widened to 0–7 Hz because of the high level of activity observed through visual inspection of the single Hz display on the therapist monitor. Following these sessions, the symptom ratings were 0 for both problems.

Summary of treatment and behaviors outside of the treatment sessions

After the first four sessions, there was no change in the symptom ratings, and the patient reported experiencing a slight increase in pain and depression. Starting with session 5, the patient reported substantial decreases in pain and depression. She also reported having more energy, having improved sleep, and feeling more relaxed. After session 17, the patient took her GED (she had missed most of the end of her senior year), and was planning to attend senior prom. After session 22, the patient started looking for a job. She also reported that she started having periods of reduced pain outside of the sessions rather than constant severe pain. Following session 24, the patient started working 2 days per week. Through sessions 22–30, the patient experienced "flare ups" of her pain, but she reported that these were not as severe as her pain intensity before treatment, and felt manageable to the patient. After session 39, the patient started college and enrolled in three classes. At session 41, the patient reported that she was getting As in all of her classes and had registered for classes for the next semester.

Case 2

Presenting problems

A 14- year-old male ninth grade boy presented with chronic left testicular pain, which he experienced as an aching, and sometimes "surging" pain in his left testicle. He was taking anti-inflammatories, pain medication, and anti-depressant medication. He rated his daily pain as between 4 and 6 on a 1–10 numerical rating scale, even when he was taking pain medication. At intake, the patient was taking fluoxetine 40 mg. and tramadol 50 mg. The pain was worse with physical activity, including walking between classes in school. The patient attended a high school that consisted of several buildings on a campus, so walking between classes sometimes involved walking between buildings. Because of the pain, the patient had been attending school for half-days for the past 6 months and had stopped sports activities (basketball and snowboarding) that he used to enjoy. The patient completed the NPC and identified a single area of concern—pain (rated as a 4 at pretreatment). EEG recordings over several cortical sites indicated higher than average beta activity over the posterior sites O1 and O2, and F4 beta power clearly greater than F3 beta.

History

About one year prior to intake, the patient experienced sharp pain in his left testicle while in gym class. He described the pain as the worst pain he had ever experienced, which made him cry. A urological examination indicated blood in his urine and he was diagnosed with epididymitis at Children's Hospital Boston.

Course of treatment

The patient was seen for 22 neurofeedback sessions approximately twice a week over a 3-month period. For sessions 1–11, the following two-channel protocol was used: O1-A1 and O2-A2, 8–11 Hz and 10–13 Hz rewards, and 4–7 Hz and 15–22

Hz inhibits. Occipital placements were attempted both because these placements had been used successfully in mixed chronic pain conditions (Melzack & Perry, 1975) and because of the high beta activity at those regions. Following session 3, he rated his pain problem as 2. Following session 7 and through session 11, he rated his pain problem as 1.

The patient was then seen for sessions 12–22 with the training site being F4-A2, because of the high pretreatment beta activity at that site, with only a 15–22 Hz inhibit and no reward frequency band. By session 17 through the end of treatment, his rating of the pain problem dropped to 0.

Summary of treatment and behaviors outside of the treatment sessions

After session 2, the patient reported that he had exerted himself more than usual outside of the office; specifically, he attended graduation parties and started playing basketball again. He experienced less pain when he played basketball and he did not ice himself after these activities, as he had been doing after any physical activity, including walking, before treatment. After session 11, the patient played more rigorous basketball and reported that this did not increase his pain, as it had prior to treatment. At session 13, the patient reported that he had stopped experiencing random bursts of pain unrelated to activity. After session 14, the patient reported that he had played basketball and swam with little pain. He decided to discontinue fluoxetine. After session 15, the patient reported that he felt "grumpy" off of fluoxetine, so he resumed taking it. After session 16, the patient reported that he had experienced no pain except for experiencing a sharp pain with no precipitating event, lasting 15–20 minutes once a week, for which he took pain medication. At session 18, the patient reported that his pain had disappeared and that he had gone jet skiing and had played basketball with no pain. At session 21, the patient reported that he had been able to walk around his school with no pain. He had experienced one incident of pain after school, for which he had taken pain medication.

Case 3

Presenting problem

A 56-year-old married woman presented with migraine headaches, which had been occurring five times a week. The headaches often lasted all day. Her migraines could get triggered by lifting her head off her pillow at night, turning in her bed, or getting out of bed. She was also experiencing anxiety and depression, which were triggered by family problems. In addition to mood disturbance, her symptoms included teeth clenching, crying, overeating, delayed sleep onset, and poor sleep maintenance. She also complained of memory problems. She was taking a combination analgesic containing acetaminophen, butalbital, and caffeine. The patient identified the following six areas of concern:

- migraine (rated as a 4),
- physical anxiety symptoms (by which she meant perceived muscle tension associated with anxiety, rated as a 4),
- depression (rated as a 4),
- sleep (rated as a 4),
- overeating (rated as a 4), and
- organizational skills (rated as a 3).

History

The patient reported that she had been experiencing migraines at the same frequency since she was 18 years old. She also experienced a great deal of stress raising her four children, all of whom she was homeschooling.

Course of treatment

The patient was seen in neurofeedback treatment twice weekly for 32 sessions. For sessions 1–5, a bipolar placement at T3-T4 was used, because her migraines and the attendant mood dysregulation were seen as signs of an unstable arousal pattern (Othmer, 2005). Because the patient often experienced pain during the sessions, the inhibits and rewards were frequently changed during the sessions in order to determine if the patient could experience an immediate, positive response to training at specific frequencies. Slow inhibits varied (2–7 Hz, 6–9 Hz, 7–10 Hz, 8–11 Hz), and the reward band varied as well (7–10 Hz to 12–15 Hz in 3-Hz wide bands). There was a constant inhibit of 22–32 Hz. The patient had difficulty controlling fast beta (22–32 Hz) activity and keeping it under threshold. She also reported a great deal of stress and guilt about family matters, and some time was spent engaging in problem solving with the patient on these matters at the beginning and end of each session, as she was being prepared for the neurofeedback training.

The patient's headaches varied in intensity during the sessions. To help address this, the patient was taught diaphragmatic breathing and was trained to increase Heart Rate Variability (HRV; McCraty, Atkinson, & Tomasino, 2001) with the assistance of a computer software program installed on a laptop computer (emWave PC Stress Relief System,

www.heartmath.com) on the fourth session, HRV was monitored simultaneously on the EEG feedback and the HRV equipment during this and most subsequent sessions. Following the fourth session, the migraine problem was rated as 2, physical anxiety as 2, depression was rated as 1, sleep as 2, overeating as 0 and organizational skill problem as 2.

Sessions 6–7 combined the following protocols: T3-T4 with 2–7 Hz and 22–32 Hz inhibits and a 7–10 Hz reward, and FZ-A1 with a 22–32 Hz inhibit and a reward of 12–15 Hz or 14–17 Hz. The FZ-A1 protocol was added because of the aforementioned role of the dorsal anterior cingulate in regulating the emotional aspect of pain and because of the role of the frontal cortex in executive skills, such as organization, which was one of the patient's complaints. Half of the session was spent on the first protocol and half on the second. The first protocol continued the T3-T4 placement with consistent inhibits and rewards set where the patient reported less anxiety and a greater sense of well-being. The FZ placement was chosen to attempt to decrease the fast beta activity. The 14–17 Hz reward was introduced in an attempt to improve the patient's executive functioning, including her organizational skills. Simultaneous HRV training was integrated into almost every neurofeedback session, except when the patient felt too tired or overwhelmed. The patient was also instructed in hand warming and was loaned a hand thermometer with which to practice at home. Her headaches decreased in frequency, intensity, and duration, even as her anxiety fluctuated. She often felt a headache coming on in the morning, but these were short-lived and did not persist for the entire day, as they had prior to treatment. Her depression problem score on the NPC also decreased; she was not experiencing depression daily, and when she began to feel depressed, the feeling quickly subsided. At the end of these sessions, migraine was rated as 2, physical anxiety was rated as 2, depression was rated as 1, sleep problems were rated as 2, overeating was rated as 0, and organization problems were rated as 2.

Sessions 8–11 involved two-channel training at T3-T4 with inhibits at 2–7 Hz and 22–32 Hz and reward at 7–10 Hz, and F3-F4 with inhibits at 1–7 Hz and 22–32 Hz with no reward. At the end of these sessions, migraine was rated as 1, physical anxiety was rated as 2, depression was rated as 0, sleep problems were rated as 2, overeating was rated as 4, and organization problems were rated as 2.

Sessions 12–13 combined the following two protocols: T3-T4 with inhibits as 2–7 Hz and 22–32 Hz and a 7–10 Hz reward, and F3-F4 with inhibits at 2–7 Hz and 22–32 Hz and a 10–13 Hz reward, varying the frontal placements in an attempt to better impact her organizational skills and also to address her depressed mood, given the role of the frontal lobes in depression (Baehr, Rosenfeld, Baehr, & Earnest, 1999). Half of each session was spent on each protocol. However, the patient did not experience any additional relief, so passive infrared hemoencephalography (pIR HEG) training was introduced.

Sessions 14–21 alternated between pIR HEG training and EEG neurofeedback within each session. During the neurofeedback training, some additional protocols were introduced due to the increased variability of the patient's headaches and other symptoms. Half of each EEG neurofeedback session was spent with either T3-T4 or C3-C4 with inhibits at 2–7 Hz and 22–32 Hz and reward at 7–10 Hz. Half of the session was spent at Fp1-A1, with a 4–8 Hz inhibit and a 15–18 Hz reward. The reward band at Fp1 was introduced in an attempt to activate the inhibitory capacity of the prefrontal cortex. At the beginning of this series of sessions, the patient's headache would start in the morning, but would stop before noon and not return for the rest of the day. Then, in 1 week, she experienced migraine headaches for 4 days, lasting all day, with the pain level at 5 on a 1–10 numerical scale. She rated her migraine problem between 0 and 3.5 on the NPC before the treatment sessions during this period. Her ratings of the overeating problem were also highly variable, ranging from 0 to 3. Her depression was rated as 0, physical anxiety was rated between 1 and 3, problems with sleep were rated between 1 and 3, and organization was rated between 2 and 3.

For sessions 22–25, the patient was trained at T3-T4 with inhibits of 2–7 Hz and 22–32 Hz and reward at 7–10 Hz. During this period, her migraine pain varied from 0 to 3. At the end of this period, physical anxiety was rated as 1, depression was rated as 0, sleep problems were rated as 1, overeating was rated as 0, and organization problems were rated as 2.

Sessions 26–32 were conducted at F3-F4 with inhibits at 2–7 Hz and 22–32 Hz and a 10–13 Hz reward to concentrate more on training the frontal cortex. The patient also reported that she had started taking a beta blocker. The patient's rating was 0 for migraines, 1 for physical anxiety, 0 for depression, 0 for sleep, 0.5 for overeating, and 2 for

organization. On the HRV monitor, her heart rate was lower and more consistent.

Summary of treatment and behaviors outside of the treatment sessions

This patient presented with migraine symptoms and complications in her mood and family situation. Although EEG neurofeedback approaches provided at the start of treatment appeared to be associated with some initial improvements in pain and other symptoms, there was variability in her symptom intensity over the course of treatment. She also did not experience as much relief with the neurofeedback training as she had hoped, and felt that her functioning continued to be impaired. Rather than persist in continuing to treat her solely with EEG neurofeedback, the decision was made to introduce and include the complementary modalities of HRV (used consistently beginning with session 4) and pIR HEG training (used intermittently beginning with session 14), both of which seemed to provide enhanced relief for the patient. An additional confound was the patient's decision to start a beta blocker prior to the final two sessions. In addition, the patient and the therapist discussed the patient's family stresses and strategies for dealing with them throughout treatment. Overall, there was some improvement in the problems she identified prior to treatment. Specifically, the patient's symptom ratings decreased as follows: migraines, from 4 to 0; physical anxiety, from 4 to 1; depression, from 4 to 0; sleep problems, from 4 to 0; overeating, from 4 to 0.5; and problems organizing, from 3 to 2. However, despite these improvements in the problem ratings, the patient reported that she was not satisfied with the outcome, perhaps due to an attribution of the bulk of these improvements to the beta blocker she initiated just before the final two sessions.

Case 4

Presenting problem

A 47-year-old divorced man, living with his female partner, presented with daily, severe gastrointestinal pain and diarrhea several times a day, starting between 3 and 5 a.m. and persisting throughout the day. These symptoms caused the patient extreme discomfort and disrupted his work routines and responsibilities. The man worked two jobs as a mechanic, which left him only 1 hour to sleep each day. He further reported, "I have no energy and I'm forgetful." He said, "I can't keep food in me," and said that he had "constant diarrhea." He reportedly ate a healthy diet. The patient had been in talk therapy previously and had not found it to be helpful; he was not interested in engaging in that form of treatment. Prior to treatment, the patient identified the following two areas of concern: diarrhea and pain (rated as a 4) and lack of ability to focus (rated as a 3).

History

The patient reported that the pain and diarrhea started to afflict him daily 14 years earlier. Prior to that, he was experiencing these symptoms more episodically. His symptoms started during a period of time when he was living with his mother and his mother moved frequently because she "got bored." He experienced some remittance of his symptoms one year prior to intake when he went on a plant-based diet for 2 months. His diarrhea decreased to two incidents a month. He went off the diet and was fine for 4 months, but then his symptoms returned to their previous levels.

His surgical history included an appendectomy at age 5, two surgeries on his right shoulder, surgery on his right knee, and surgery to repair a hernia. He reported a history of pain in his lower back, hips, and knees. He consumed two to three cola beverages on weekends, but denied consuming alcohol, tobacco, or recreational drugs. Family history was significant for Chron's Disease in the patient's older brother and Bipolar Disorder in his younger brother. The patient denied any history of childhood trauma or illness.

Course of treatment

The patient was seen in neurofeedback treatment, first twice weekly and then once weekly, for 26 sessions. For sessions 1–14, a bipolar placement at T3-T4 was used, because the patient's arousal pattern was not clear and his symptoms seemed indicative of an unstable pattern. The inhibits were at 4–7 Hz and 18–32 Hz, and the reward bands were 8–12 Hz and 15–18 Hz. Within five sessions, his diarrhea and pain rating improved to 0, and his focus rating improved to a 2. During the course of the five sessions, the patient also reported a gradual reduction in the number of days in which he was afflicted with pain and diarrhea and a decrease in the frequency, intensity, and duration of his symptoms during the days when he was symptomatic. He reported that his mood was better and that he was getting more and better sleep, and had been able to sleep for 5 hours at night. At the eighth session, the patient reported that he had had 6 consecutive symptom-free days, followed by a single day of discomfort, 2 symptom-free days, and then another single day of discomfort. At the eleventh session, the patient reported only one incident of symptom occurrence, without an

identified precipitating event, with the symptoms lasting 3 hours in the morning.

The decision was jointly made to decrease the frequency of sessions to once per week. At the twelfth session, the patient reported only 1 day of symptoms in the previous week. There followed an interruption of treatment for 3 weeks, due to scheduling difficulties associated with the winter holiday season. When the patient returned, he reported that he had been symptom free until New Year's Eve, when he overate and had 3 days of diarrhea. Since then, however, he had not had gastrointestinal problems. He also reported that his sleep and his memory had worsened in the intervening period. His diarrhea and pain problem was rated as 0, and his cognitive focus problem was rated as 2.

For sessions 15–16, we agreed to change the protocol to more directly address the focus problem. We began the protocol CZ-A1 with inhibits at 4–7 Hz and 22–32 Hz, and the reward band at 15–18 Hz. For session 16, the reward band was increased to 16–19 Hz. After session 16, the patient reported that his diarrhea and pain had worsened and that he was now experiencing these symptoms every morning and after each meal. However, these symptoms were not persisting constantly throughout each day, as they had prior to treatment, and his pain continued to be less intense than previously. He still rated his diarrhea and pain as 0 and focus as 2. We scheduled a second session that week and, at session 17, we decided to train at T3-T4 with inhibits at 4–7 Hz and 18–21 Hz and rewards at 8–12 Hz and 12–15 Hz. At session 18, the patient reported that he had been symptom free since session 17, and the decision was made to train half the session at T3-T4 and half the session at CZ-A1. For sessions 18–21, we continued with the T3-T4 protocol for half the session and added CZ-A1, inhibiting 4–7 Hz and 22–32 Hz and rewarding 16–19 Hz to improve focus. At session 19, the patient reported experiencing diarrhea again that morning. At session 20, the patient reported having had no diarrhea and pain, but was increasingly troubled by forgetfulness. He understood that this latter problem might be related to his lack of sleep. His diarrhea and pain symptoms remained a 0 on the NPC, but his forgetfulness (not rated on the NPC, because this was not identified as a problem initially) did not improve, so the decision was therefore made to discontinue training at CZ-A1 and to initiate training at FZ-A1 with the same inhibits and rewards, in order to see if that protocol could be more beneficial to his cognitive functioning. This was done for sessions 22–26. By session 24 the patient was reporting improved sleep with more energy, with no resumption of his diarrhea and pain. He reported experiencing bursts of increased energy. This combined protocol had been continued through session 24. At that point, the patient's diarrhea and pain was rated 0, and his focus was rated 1.5. At session 25, the reward on the FZ-A1 protocol was increased to 17–20 Hz. His diarrhea and pain were rated as 0, and focus was 1.5. At session 26, the patient reported that he had experienced explosive diarrhea all day at work from Saturday through Tuesday. He rated his diarrhea and pain as 4, and focus as 1.5. The training was changed back to the T3-T4 protocol for the entire session.

The patient called the next week to cancel his appointment because of an illness that, he said, had been coming on for the past week or two. The patient then missed his next scheduled appointment. Attempts were made to reach the patient by telephone but the calls were not returned. Two months later, the patient's partner called to report that their relationship had deteriorated and that the patient was very angry, depressed, passive-aggressive and non-communicative. She said that his diarrhea and pain had returned on a daily basis. At a follow-up appointment with the patient and his partner, the patient reported that he did not want to continue with neurofeedback training.

Summary of treatment and behaviors outside of the treatment sessions

This patient presented with diarrhea and severe gastrointestinal pain of over 14 years duration, with symptoms occurring daily and throughout each day for the past 14 years with a reduction in frequency for several months after he had changed his diet. He also complained of problems focusing his attention. During treatment, he identified a third problem area (memory problems) that he attributed to lack of sleep and to working two full-time jobs. The patient experienced a gradual but rapid resolution of his diarrhea and pain symptoms over the first five sessions covering 2 weeks of training. Attempts to reduce the frequency of his training were met with a partial resumption of symptoms, but these resolved over time. He also reported improvements in his ability to focus and improved sleep and mood after the introduction of a protocol that targeted those areas of functioning. By the end of the 25th session, the patient's diarrhea and pain had resolved for several weeks and his cognitive functioning had improved. His diarrhea and pain rating had improved from 4 to 0, and his problems focusing rating had improved from 3 to 1.5.

However at the 26th session, the patient reported a resumption of his symptoms during most days of the preceding week. He later reported that he had been coming down with an illness. The patient cancelled the next session due to illness and missed the appointment after that. He then did not return phone calls. A phone call from the patient's significant other indicated that his symptoms had returned to their previous level and that there was significant conflict in their relationship. At a follow-up session, the patient said that he was not interested in additional neurofeedback training.

Discussion

These case studies document the improvement in chronic pain symptoms and related improvement in quality of life for patients of various ages, with a variety of chronic pain conditions, who underwent neurofeedback training. All patients had previously undergone conventional medical treatments for their conditions, and two had been in some form of psychological therapy. All of the patients reported substantial symptom relief after the neurofeedback training. However, one of these patients expressed dissatisfaction with the training and attributed her improvement (in headaches) to a beta blocker medication that was initiated towards the end of treatment, even though she had reported substantial improvements in her symptoms prior to this. A second patient who had made significant improvement had a relapse during treatment—deterioration in his condition, which might have been related to an incipient illness—and his symptoms continued following this relapse. He also elected to discontinue treatment, even though he had reported complete relief from his pain and gastrointestinal symptoms to an extent that he had not experienced in years. Whether he would again experience symptom improvements had he continued with treatment, and whether those improvements would have maintained after any additional treatment was completed, cannot be known.

Although the cortical training sites, and changes in those sites over the course of treatment, were tailored for each patient based on the treatment goals and the patients' response (see summary of protocols in Table 1), treatment protocols common to all four patients included inhibiting theta (4–7 Hz) and fast beta (22–32 Hz) activity and rewarding alpha (8–12 Hz) and slow beta (12–15 Hz) activity. All four patients were trained at more than one cortical site during the course of the training. These sites included prefrontal, frontal, central, temporal, parietal, and occipital placements; although all four of the patients trained at frontal sites at some point. Three of the patients also trained at temporal sites.

These promising preliminary findings indicate that additional research to study the efficacy of neurofeedback for chronic pain is warranted. Based on these findings, and to the extent that research in this area requires standardization, researchers would do well to consider protocols that include the common elements of the training provided to the patients in this case series—that is, protocols that include inhibition of theta and fast beta and rewarding of alpha and slow beta, with variable training sites. Research comparing these protocols to standard care would be important. It would also be important to assess the effects of nonspecific aspects of the training. This could potentially be done in an experimental study in which one group of subjects receives training with the rewards and inhibits stated above, and another group receives training in a control condition not expected to impact pain.

The selection of the control protocol in a clinical trial would need to take some thought, however. From a scientific perspective, perhaps a "negative protocol" in which theta and fast beta are rewarded and alpha and slow beta are inhibited would be ideal, as this represents the opposite of a protocol hypothesized to be beneficial. However, because of concerns that the negative protocol might have negative effects—a possibility that requires use of such a protocol to confirm—in our view if such a protocol were used, it should be provided for only brief periods of time. Also, in designs using such a protocol, it would be important, from an ethical perspective, for the treatment protocol to be made available to the subjects following the control protocol and for appropriate informed consent and human subjects review to be implemented to protect the subjects from the possible negative effects of the control protocol.

Table 1

Summary of Neurofeedback Protocols and Responses for Each Case

Patient	Session #	Training Site(s)	Reward(s) (Hz)	Inhibit(s) (Hz)	Pain intensity
#1 19-year-old female with GI pain	Pre-tx				4
	1–3	T3-T4	Frequency ranging from 9–12 to 12–15	2–7, 22–32	4
	4–5	CZ-A1 or FZ-A1	Beta frequency ranging from 12–15 to 20–23	4–7, 22–32	4
	6–10	FZ-A1	8–13	4–7, 22–32	2
	11–18	FZ-A1	8–13, 12–15	4–7, 22–32	1
	19	O2-A1	8–13	4–7, 22–32	3
	20	P4-A2	8–13	4–7, 22–32	3
	21	FZ-A1	8–13, 12–15	2–7, 22–32	
	22–29	C4-A2	8–13, 12–15	2–7, 22–32	2
	30–33	2 channel: F1-A1, F2-A2	8–13	4–7, 15–32	1
	34–41	2 channel: F1-A1, F2-A2	8–13	0–7, 15–32	0
#2 14-year-old male with testicular pain	Pre-tx				4
	1–5	T3-T4	Frequency ranging from 7–10 to 12–15	22–32, plus 2–7 or 6–9 or 7–10 or 8-11	2
	6–7	T3-T4 FZ-A1	7–10 12–15 or 14–17	2–7, 22–32 2–7, 22–32	2
	8–11	2 channel: T3-T4 FZ-A1	7–10 No reward	2–7, 22–32 1–7, 22–32	1
	12–13	T3-T4 F3-F4	7–10 10–13	2–7, 22–32 2–7, 22–32	
	14–21	pIR HEG or T3-T4 or C3-C4	7–10	2–7, 22–32	0–3.5
	22–25	T3-T4	7–10	2–7, 22–32	0–3
	26–32	F3-F4	10–13	2–7, 22–32	0
#3 56-year-old female with migraines	Pre-tx				4
	1–5	T3-T4	Frequency ranging from 7–10 to 12–15	22–32, plus 2–7 or 6–9 or 7–10 or 8–11	2
	6–7	T3-T4 FZ-A1	7–10 12–15 or 14–17	2–7, 22–32 2–7, 22–32	2
	8–11	2 channel: T3-T4 FZ-A1	7-10 No reward	2-7, 22-32 1–7, 22–32	1
	12–13	T3-T4 F3-F4	7–10 10–13	2–7, 22–32 2–7, 22–32	
	14–21	pIR HEG or T3-T4 or C3-C4	7–10	2–7, 22–32	0–3.5
	22–25	T3-T4	7–10	2–7, 22–32	0–3
	26–32	F3-F4	10–13	2–7, 22–32	0
#4 47-year-old male with GI pain	Pre-tx				4
	1–14	T3-T4	8–12, 12–15	4–7, 18–32	0
	15–16	CZ-A1	15–18	4–7, 22–32	0
	17	T3-T4	8–12, 12–15	4–7, 22–32	
	18–21	CZ-A1 T3-T4	16–19 8–12, 12–15	4–7, 22–32 4–7, 22–32	0
	22–24	FZ-A1 T3-T4	16–19 8–12, 12–15	4–7, 22–32 4–7, 22–32	0
	25	FZ-A1 T3-T4	17–20 8–12, 12–15	4–7, 22–32 4–7, 22–32	0
	26	T3-T4	8–12, 12–15	4–7, 22–32	4

A second type of control protocol would be one that might be hypothesized to result in some general benefits (e.g., increased ability to concentrate, improved mood), but that would not necessarily be expected to have direct beneficial effects on pain (for example, a protocol involving rewarding 15–18 Hz activity, and inhibiting 4–7 Hz and 22–32 Hz activity as measured at CZ). While detecting an effect of an "active" (focused on pain reduction) protocol over such a "pain neutral" protocol might be more difficult than detecting an effect over a negative protocol because of indirect beneficial effects of general improvements in mood on pain, such a design might also be considered more ethical; especially, if a negative protocol is indeed found to have negative effects on pain or function. Pilot research to explore the effects of various protocols on outcomes would be useful prior to finalizing any specific design.

Limitations

This study has a number of important limitations that need to be considered when interpreting the results. Primary among them, of course, is that the study is an uncontrolled case series. Thus, it is not possible to draw conclusions regarding the factors that contributed to the benefits observed. For any one patient, the benefits observed could have been due to time effects or any one or more of many nonspecific factors associated with treatment (e.g., patient or clinician motivations and expectations, therapeutic rapport) or even specific factors (e.g., initiation of a beta blocker) not associated with the neurofeedback treatment. Properly controlled clinical trials are needed to determine which of the benefits that occur with neurofeedback are specific to the neurofeedback training, including the specific training protocol used.

A second important issue is that protocol selection was made based on research on the very limited frequency bands and cortical sites that have been shown to be responsive to pain perception, as well as on research and publications on neurofeedback with other populations. It is possible that other protocols might have been even more effective than the protocols used here. Given the wide variety of chronic pain conditions, including the multiplicity of symptoms, precipitating conditions, levels of intensity, and types of coexisting emotional and behavioral impairment, there is a need for a more systematic process of protocol selection, which only more extensive, controlled research can provide.

A third issue is that the improvements in the patients' conditions were recorded using a general rating of how much of a problem each of the symptoms was in each patient's life. Although this allowed the patients a great deal of latitude to judge for him or herself the impact of their pain, more targeted and specific measures of pain and pain interference might have allowed for a better ability to compare progress and treatment effect across patients. Future researchers in this area—including clinicians who plan to present the results of their clinical work as case studies—would do well to consider using standardized measures of pain and pain-related outcomes in their work (Dworkin et al., 2005; Jensen, 2010; Jensen, Karoly, & Braver, 1986).

A fourth concern is that decisions to change protocols were based on the clinician's interpretations of the patients' responses, rather than on objective or prearranged criteria. While a large part of the decision making in neurofeedback must be clinical in nature, the lack of any systematically applied criteria for changing protocols results in uncertainty as to whether any one protocol was used for a long enough period of time to see if it was effective. A related point is whether a protocol change was the primary factor in symptom reduction of if the treatment effect was a cumulative effect of all of the protocols used. In case 1, for example, the patient began to demonstrate improvement after an alpha reward was introduced in a certain protocol starting in session 6. It would be important to further investigate whether this was an essential factor in the patient's improvement.

An additional limitation of the current study was the lack of assessment of EEG activity, as an outcome or process variable, given that the model underlying the use of neurofeedback for pain management hypothesizes a causal role for brain activity in general, and brain oscillations in particular, in the experience of pain (Jensen, Day, & Miró, 2014). Future researchers (and clinicians presenting case studies) would do well to use the results of qEEGs to help understand the extent to which improvements in pain are accompanied by changes in brain oscillation activity when possible. It is possible that such research could also be used to guide protocol selection and could lead to more effective and efficient treatment.

Summary and Conclusions

Four patients with a variety of chronic pain conditions were treated with multiple (between 22 and 41) sessions of neurofeedback training. All reported substantial reductions in chronic pain intensity and improvements in other domains of

quality of life during treatment. However, one patient reported dissatisfaction with the treatment and attributed her improvements to a medication that was initiated towards the end of neurofeedback training, and a second patient relapsed following a brief illness and an interruption in treatment, and subsequently discontinued treatment. The findings from this case series provide additional evidence suggesting that neurofeedback might be an effective method for helping some (and perhaps many) individuals with chronic pain learn self-management skills that would give them more control over, and alleviate, their suffering, and also result in improvements in other related symptoms and problems. This latter point is supported by the findings of Choobforoushzadeh, Neshat-Doost, Molavi, and Abedi (2015), who found that neurofeedback reduced symptoms of depression and fatigue in patients with multiple sclerosis. More research is also warranted to evaluate the efficacy of neurofeedback, relative to other pain treatments, to distinguish the specific from the nonspecific effects of neurofeedback, and to identify the training protocols that are most effective for patients with different pain conditions.

References

Albert, A. O., Andrasik, F., Moore, J. L., & Dunn, B. R. (1998). Theta/beta training for attention, concentration and memory improvement in the geriatric population. *Applied Psychophysiology and Biofeedback, 23*(2), 109.

Andreychuk, T., & Skriver, C. (1975). Hypnosis and biofeedback in the treatment of migraine headache. *International Journal of Clinical and Experimental Hypnosis*, *23(3),* 172–183.

Apkarian, A. V., Bushnell, M. C., Treede, R.-D., & Zubeita, J.-K. (2005). Human brain mechanisms of pain perception and regulation in health and disease. *European Journal of Pain, 9*(4), 463–484. http://dx.doi.org/10.1016/j.ejpain.2004.11.001

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

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 and Neuroscience, 40*(3), 180–189. http://dx.doi.org/10.1177/155005940904000311

Arns, M., Heinrich, H., & Strehl, U. (2014). Evaluation of neurofeedback in ADHD: The long and winding road. *Biological Psychology, 95,* 108–115. http://dx.doi.org/10.1016/j.biopsycho.2013.11.013

Babiloni, C., Brancucci, A., Babiloni, F., Capotosto, P., Carducci, F., Cincotti, F., … Rossini, P. M. (2003). Anticipatory cortical responses during the expectancy of a predictable painful stimulation. A high-resolution electroencephalography study. *European Journal of Neuroscience, 18*(6), 1692–1700. http://dx.doi.org/10.1046/j.1460-9568.2003.02851.x

Babiloni, C., Capotosto, P., Brancucci, A., Del Percio, C., Petrini, L., Buttiglione, M., … Arendt-Nielsen, L. (2008). Cortical alpha rhythms are related to the anticipation of sensorimotor interaction between painful stimuli and movements: A high-resolution EEG study. *The Journal of Pain, 9*(10), 902–911. http://dx.doi.org/10.1016/j.jpain.2008.05.007

Baehr, E., Rosenfeld, J. P., Baehr, R., & Earnest, C. (1999). Clinical use of an alpha asymmetry neurofeedback protocol in the treatment of mood disorders. In J. R. Evans, & A. Arbarbanel (Eds.), *Introduction to Quantitative EEG and Neurofeedback* (pp. 181–203). New York: Academic Press.

Bromm, B., & Lorenz, J. (1998). Neurophysiological evaluation of pain. *Electroencephalography and Clinical Neurophysiology, 107*(4), 227–253. http://dx.doi.org/10.1016/S0013-4694(98)00075-3

Burroughs, R. D. (2011). *Quantitative EEG Analysis of Patients with Chronic Pain: An Exploratory Study*. (Master of Science thesis). Retrieved from University of North Texas (UNT) Digital Library, Denton, Texas. http://digital.library.unt.edu/ark:/67531/metadc103294

Chen, A. C. N. (2001). New perspectives in EEG/MEG brain mapping and PET/fMRI neuroimaging of human pain. *International Journal of Psychophysiology, 42*(2), 147–159. http://dx.doi.org/10.1016/S0167-8760(01)00163-5

Choobforoushzadeh, A., Neshat-Doost, H. T., Molavi, H., & Abedi, M. R. (2015). Effect of neurofeedback training on depression and fatigue in patients with multiple sclerosis. *Applied Psychophysiology and Biofeedback, 40*(1), 1–8. http://dx.doi.org/10.1007/s10484-014-9267-4

Cohen, M. J., McArthur, D. L., & Rickles, W. H. (1980). Comparison of four biofeedback treatments for migraine headache: physiological and headache variables. *Psychosomatic Medicine, 42*(5), 463–480.

Craig, A. D. (2003a). Interoception: The sense of physiological condition of the body. *Current Opinion in Neurobiology, 13*(4), 500–505. http://dx.doi.org/10.1016/S0959-4388(03)00090-4

Craig, A. D. (2003b). Pain mechanisms: Labeled lines versus convergence in central processing. *Annual Review of Neuroscience, 26,* 1–30. http://dx.doi.org/ 10.1146/annurev.neuro.26.041002.131022

Cunningham, M., & Murphy, P. (1981). The effects of bilateral EEG biofeedback on verbal, visuospatial and creative skills in LD male adolescents. *Journal of Learning Disabilities, 14*(4), 204–208.

DeCharmes, R. C., Maeda, F., Glover, G. H., Ludlow, D., Pauly, J. M., Soneji, D., … Mackey, S. C. (2005). Control over brain activation and pain learned by using real-time functional fMRI. *Proceedings of the National Academy of Sciences of the United States of America, 102*(51), 18626–18631. http://dx.doi.org/10.1073/pnas.0505210102

DeLeo, J. A. (2006). Basic science of pain. *The Journal of Bone and Joint Surgery, 88*(Suppl. 2), 58–62. http://dx.doi.org/10.2106/JBJS.E.01286

Dworkin, R. H., Turk, D. C., Farrar, J. T., Haythornthwaite, J. A., Jensen, M. P., Katz, N. P., … Winter, J. (2005). Core outcome measures for chronic pain clinical trials: IMMPACT recommendations. Pain, *113*(1–2), 9–19. http://dx.doi.org/10.1016/j.pain.2004.09.012

Egner, T., & Gruzelier, J. H. (2003). Ecological validity of neurofeedback: Modulation of slow wave EEG enhances musical performance. *NeuroReport, 14*(9), 1221–1224. http://dx.doi.org/10.1097/00001756-200307010-00006

Escolano, C., Navarro-Gil, M., Garcia-Campayo, J., Congedo, M., & Minguez, J. (2014). The effects of individual upper alpha neurofeedback in ADHD: An open-label pilot study. *Applied Psychophysiology and Biofeedback, 39*(3–4), 193–202. http://dx.doi.org/10.1007/s10484-014-9257-6

Fenger, T. N. (1995, September). Visual-motor integration and its relation to EEG neurofeedback brain wave patterns, reading, spelling, and arithmetic achievement in attention deficit disorders and learning disabled students. Paper presented at the meeting of the Society for the Study of Neuronal Regulation, Scottsdale, AZ.

Flor, H. (2003). Cortical reorganisation and chronic pain: Implications for rehabilitation. *Journal of Rehabilitation Medicine, 35*(41), 66–72. http://dx.doi.org/10.1080/16501960310010179

Gannon, L., & Sternbach, R. A. (1971). Alpha enhancement as a treatment for pain: A case study. *Journal of Behavior Therapy and Experimental Psychiatry, 2*, 209–213.

Gruzelier, J., Egner, T., & Vernon, D. (2006). Validating the efficacy of neurofeedback for optimizing performance. *Progress in Brain Research, 159*, 421–431.

Gruzelier, J. (2009). A theory of alpha/theta neurofeedback, creative performance enhancement, long distance functional connectivity and psychological integration. *Cognitive Processing, 10*(Suppl. 1), 101–109. http://dx.doi.org/10.1007/s10339-008-0248-5

Jensen, M. P. (2010). A neuropsychological model of pain: Research and clinical implications. *The Journal of Pain, 11*(1), 2–12. http://dx.doi.org/10.1016/j.jpain.2009.05.001

Jensen, M. P., Grierson, C., Tracy-Smith, V., Bacigalupi, S. C., & Othmer, S. (2007). Neurofeedback treatment for pain associated with complex regional pain syndrome type I. *Journal of Neurotherapy, 11*(1), 45–53. http://dx.doi.org/10.1300/J184v11n01_04

Jensen, M. P., Karoly, P., & Braver, S. (1986). The measurement of clinical pain intensity: A comparison of six methods. *Pain, 27*(1), 117–126. http://dx.doi.org/10.1016/0304-3959(86)90228-9

Jensen, M. P., Sherlin, L. H., Hakimian, S., & Fregni, F. (2009). Neuromodulatory approaches for chronic pain management: Research findings and clinical implications. *Journal of Neurotherapy, 13*(4), 196–213. http://dx.doi.org/10.1080/10874200903334371

Jensen, M. P., Day, M. A., & Miró, J. (2014). Neuromodulatory treatments for chronic pain: Efficacy and mechanisms. Nature Reviews Neurology, 10, 167–178. http://dx.doi.org/10.1038/nrneurol.2014.12

Katz, J., & Melzack, R. (1990). Pain "memories" in phantom limbs: Review and clinical observations. *Pain, 43,* 319–336.

Katz, W. A., & Rothenberg, R. (2005). The nature of pain: Pathophysiology. *Journal of Clinical Rheumatology, 11*(2 Suppl.), S11–S15. http://dx.doi.org/10.1097/01.rhu.0000158686.43637.af

Lansbergen, M. M., van Dongen-Boomsma, M., Buitelaar, J. K., & Slaats-Willemse, D. (2011). ADHD and EEG-neurofeedback: A double-blind randomized placebo-controlled feasibility study. *Journal of Neural Transmission, 118*(2), 275–284. http://dx.doi.org/10.1007/s00702-010-0524-2

Linden, M., Habib, T., & Radojevic, V. (1996). A controlled study of the effects of EEG biofeedback on cognition and behavior of children with attention deficit disorder and learning disabilities. *Biofeedback and Self-Regulation, 21*(1), 35–49.

Lindsay, P., & Wyckoff, M. (1981). The depression-pain syndrome and its response to antidepressants. *Psychosomatics*, *22*(7), 571–577.

Logemann, H. N. A., Lansbergen, M. M., Van Os, T. W. D. P., Böcker, K. B. E., & Kenemans, J. L. (2010). The effectiveness of EEG-feedback on attention, impulsivity and EEG: A sham feedback controlled study. *Neuroscience Letters, 479*(1), 49–53. http://dx.doi.org/10.1016/j.neulet.2010.05.026

Lubar, J. F. (1991). Discourse on the development of EEG diagnostics and biofeedback for attention-deficit/hyperactivity disorders. *Biofeedback and Self-Regulation*, *16*(3), 201–225. http://dx.doi.org/10.1007/BF01000016

McCraty, R., Atkinson, M., & Tomasino, D. (2001). *Science of the Heart: Exploring the Role of the Heart in Human Performance (*Publication No. 01-001). Boulder Creek, CA: HeartMath Research Center, Institute of HeartMath.

Melzack, R., Coderre, T. J., Katz, J., & Vaccarino, A. L. (2001). Central neuroplasticity and pathological pain. *Annals of the New York Academy of Sciences, 933,* 157–174, http://dx.doi.org/10.1111/j.1749-6632.2001.tb05822.x

Melzack, R., & Perry, C. (1975). Self-regulation of pain: The use of alpha-feedback and hypnotic training for the control of chronic pain. *Experimental Neurology, 46,* 452–469.

Miltner, W. H. R., & Weiss, T. (1998). Brain electrical correlates of pain processing. *Zeitschrift für Rheumatologie, 57*(2 Suppl.), S14–S18. http://dx.doi.org/10.1007/s003930050227

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.

Murphy, P. J., Darwin, J., & Murphy, D. (1977). EEG feedback training for cerebral dysfunction: A research program with learning disabled adolescents. *Biofeedback and Self-Regulation, 2,* 288.

Nall, A. (1973). Alpha training and the hyperkinetic child—Is it effective? *Intervention in School and Clinic, 9*(1), 5–19. http://dx.doi.org/10.1177/105345127300900101

National Institute of Neurological Disorders and Stroke, National Institutes of Health. (2015). *Pain in America*. Retrieved from http://www.ninds.nih.gov/disorders/chronic_pain/chronic_pain.htm

Nishigami, T., Nakano, H., Osumi, M., Tsujishita, M., Mibu, A., & Ushida, T. (2014). Central neural mechanisms of interindividual difference in discomfort during sensorimotor incongruence in healthy volunteers: An experimental study. *Rheumatology, 53*(7), 1194–1199. http://dx.doi.org/10.1093/rheumatology/ket494

Othmer, S. F. (2005). Interhemispheric EEG training. *Journal of Neurotherapy, 9*(2), 87–96. http://dx.doi.org/10.1300/J184v09n02_08

Othmer, S., Othmer, S. F., & Kaiser, D. A. (1999). EEG biofeedback: Training for AD/HD and related disruptive behavior disorders. Chapter in J. A. Incorvaia, B. S. Mark-Goldstein, & D. Tessmer (Eds.), *Understanding, Diagnosing, and Treating AD/HD in Children and Adolescents: An Integrative Approach* (235–296). New York: Jason Aronson Press. Peyron, R., Laurent, B., & Garcia-Larrea, L. (2000). Functional imaging of brain responses to pain. A review and meta-analysis. *Clinical Neurophysiology, 30*(5), 263–288.

Pfurtscheller, G. (2003). Induced oscillations in the alpha band: Functional meaning. *Epilepsia*, 44(Suppl. 12), 2–8. http://dx.doi.org/10.1111/j.0013-9580.2003.12001.x

Prinsloo, S., Gabel, S., Lyle, R., & Cohen, L. (2012). Neuromodulation of cancer pain. *Integrative Cancer Therapies,13*(1), 30–37. http://dx.doi.org/10.1177/1534735413477193

Rainville, P., Duncan, G. H., Price, D. D., Carrier, B., & Bushnell, M. C. (1997). Pain affect encoded in human anterior cingulate but not in somatosensory cortex. *Science, 277*(5328), 968–971.

Rasey, H. W., Lubar, J. E., McIntyre, A., Zoffuto, A. C., & Abbott, P. L. (1996). EEG biofeedback for the enhancement of attentional processing in normal college students. *Journal of Neurotherapy, 1(*3), 15–21.

Thompson, L., &, Thompson, M. (1998). Neurofeedback combined with training in metacognitive strategies: Effectiveness in students with ADD. *Applied Psychophysiology and Biofeedback, 23*(4), 243–263. http://dx.doi.org/10.1023/A:1022213731956

Tinazzi, M., Fiaschi, A., Rosso, T., Faccioli, F., Grosslercher, J., & Aglioti, S. M. (2000). Neuroplastic changes related to pain occur at multiple levels of the human somatosensory system: A somatosensory-evoked potentials study in patients with cervical radicular pain. *The Journal of Neuroscience, 20*(24), 9277–9283.

Vernon, D., Egner, T., Cooper, N., Compton, T., Neilands, C., Sheri, A., & Gruzelier, J. (2003). The effect of training distinct neurofeedback protocols on aspects of cognitive performance. *International Journal of Psychophysiology, 47*(1), 75–85.

Whisenant, W. F., & Murphy, P. J. (1977). Bilateral EEG biofeedback and creativity. *Biofeedback and Self-Regulation, 2,* 322.

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NeuroRegulation

Book Review – *Neurofeedback in the Treatment of Developmental Trauma: Calming the Fear-Driven Brain*

by Sebern F. Fisher. W. W. Norton & Company, New York, NY, 2014, 416 pages, ISBN: 978-0-393-70786-1.

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A long-awaited text from the originator of the FPO2 neurofeedback protocol for treating symptoms of psychological trauma, this book is both well crafted and beautifully written. Fisher gives us an insight into her own mind as she prosaically describes her clients from the framework of an experienced and compassionate clinician. The book thoroughly presents the treatment of fearful minds by bringing together psychotherapeutic theory, principles of EEG biofeedback, personal experience, and clear guidance.

The forward by Bessel van der Kolk, MD, sets the stage for the book's integration of developmental and psychological theory, neuroscience, and neurofeedback techniques. The structure of the book takes the reader first through foundational theory and developmental neuroanatomy, detailing the effects of trauma on the childhood brain and its subsequent insidious legacy for adulthood. Secondly, a primer on neurofeedback is woven into the various discussions of development, symptoms, and therapy, making the book a sound starting point for clinicians who are new to the field, as well as offering insights into the nuances of treatment that seasoned clinicians would appreciate. Numerous case studies shed light on the actual process of treatment and illustrate the heart-rending nature of working with this population.

In chapter 1, Fisher makes the case for a diagnostic category that transcends the limitations of two diagnoses commonly applied to persons with the symptoms she describes, namely posttraumatic stress disorder (PTSD) and reactive attachment disorder (RAD). Developmental trauma disorder (DTD) subsumes these two diagnoses into one cohesive syndrome. Fisher details the precursors of DTD, which include attachment rupture, poverty, adverse events, and neglect. Symptomatically, DTD's hallmarks are aberrations in the capacity for empathy, and affective and sensory dysregulation. How these symptoms play out in life and therapy is further described, with special emphasis on the role of transference in the treatment process.

Chapters 2 and 3 comprise a thorough overview of the corpus of conventional neurofeedback concepts, with special attention and application to the treatment of DTD. Fisher goes into some depth on the neuroanatomical and neurophysiological effects of developmental trauma. Laying the groundwork for the reader's grasp of neurofeedback, she describes the significance of the various frequency bands and how EEG biofeedback is used in the amelioration of a dysregulated brain. For the potential client, this description gives a picture of what to expect in a treatment session. For clinicians acquainted with the arousal model as originally developed by the Othmers (Othmer, Othmer, & Kaiser, 1999), this will be familiar territory.

Fisher's discussion of the nuts and bolts of neurofeedback is continued later in the book, but first she delves into the psychodynamics of developmental trauma in chapter 4, which is entitled "Trauma Identity." From the neuronal level to the complexities of the self, she covers a gamut of implications for trauma's effects against the framework of object relations and self-psychological concepts. It is this aspect of Fisher's writing that imparts a special dimension to her approach, which presumes that the treatment is done at the hands of clinicians who are not only skilled in the procedures of neurofeedback, but have a strong operational knowledge of the brain's functions and the mind's

workings, and the developmental perspective to appreciate the encompassing process that therapy with these individuals entails.

Chapter 5 is a clinical guide to introducing clients to neurofeedback and the steps involved in forming the therapeutic relationship and beginning the assessment process. Early in the chapter Fisher emphasizes a symptom-based approach to protocol selection. Quantitative electroencephalogram (QEEG) analysis, in her practice, is reserved for clients with special issues and for cases in which treatment is not progressing as expected. Professional client-therapist issues are also discussed with specific recommendations related to this unique population and treatment approach.

Fisher places great importance in the book on assessment, which she covers in chapter 6. Her approach provides the clinician with a clear path to selecting treatment protocols and a wealth of considerations befitting an encompassing model that bears out the deliberate long-term process of clinical experience, research, and collaboration that produced it. Fisher lays out a very systematic approach to assessing the client's symptoms and indications for specific treatment strategies. The initial assessment covers a variety of symptoms and presentation, including attention, sleep, emotional, behavioral, cognitive, pain, neurological, as well as autoimmune, autonomic nervous system, and endocrine issues—all of which are guiding factors in the arousal (and regulation) model. A plethora of additional considerations are discussed for gaining an even more complete picture of the client's condition. The ongoing assessment proceeds from session to session, and includes attention to the EEG itself and certain measures that may be applicable, as well as dealing with a plateau in progress and indications of when the therapeutic work is to be concluded.

Chapter 7 is a major expansion of the assessment process with in-depth descriptions of protocols. Various electrode placement sites, frequency bands to reward or inhibit, and the customization of protocols to suit the individual needs of each client are described with generous attention to clinical application and the rationale for each respective aspect. In combination with the foundational material presented earlier in the book, the discussion of treatment strategies takes on a particularly profound quality. Included in the protocol descriptions is a section devoted to the FPO2 protocol, which has historically been one of the most well-known features of the "Fisher protocol." Fisher based the protocol on a number of factors: the neurological concepts of Schore (1994) and LeDoux (1996), "as well as on traditions in body and energy work." At every turn, concepts are paired with clinical illustrations.

As the book contains a primer on neurofeedback, so it provides a primer on psychodynamic therapy, which is presented in chapter 8. In so doing, Fisher continues her masterful work in integrating the two approaches into a cogent methodology. Basic psychodynamic concepts are explained and applied to the treatment of DTD, including transference and countertransference, regression, relationship development, shame, projection, the unconscious, dissociation, and finally, the emerging self structure.

The text of the entire book is amply illustrated with clinical examples. As if that isn't enough, Fisher provides a bonus feature at the end. Chapter 9 consists of detailed vignettes of three clients, including analyses of the treatment process for each.

Numerous tables and graphical illustrations are provided in the text, as well as a set of color figures in the book's center. Addenda contain Fisher's extensive Neurofeedback Assessment Questionnaire, a FAQ suitable for sharing with prospective clients, and a FPO2 Protocol Guide. A list of references and thorough index are provided.

In her book, Fisher offers much to readers in many ways. For those who specialize in the treatment of traumatized persons, this book is an indispensable guide even if the reader does not utilize neurofeedback! The discussion of human development and the neuroscience underlying developmental trauma stands on its own. For neurofeedback clinicians at any level of expertise, Fisher's writings are a comprehensive guide. For parents and those suffering from developmental trauma, here is a treasure trove of wisdom. In all, Fisher provides a validation to those who have worked with this population and a hopeful vision for the healing of these profound wounds.

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References

LeDoux, J. E. (1996). *The emotional brain: The mysterious underpinnings of emotional life*. New York: Simon & Schuster.

Othmer, S., Othmer, S. F., & Kaiser, D. (1999). EEG Biofeedback: An Emerging Model for Its Global Efficacy. In J. R. Evans & A. Abarbanel (Eds.), *Introduction to Quantitative EEG and Neurofeedback* (pp. 243-304). San Diego, CA: Academic Press.

Schore, A. N. (1994). *Affect regulation and the origin of the self: The neurobiology of emotional development*. Hillsdale, NJ: Lawrence Erlbaum Associates.

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KEYNOTE PRESENTATIONS

QEEG and Neurofeedback in the Assessment and Treatment of Psychological Disorders

C. Richard Clark, PhD
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Professor of Psychology, Flinders University, Adelaide, South Australia

Neurofeedback therapy works by harnessing the natural plasticity of the brain—its ability to learn and adapt to the world, from childhood through to older adult age. There are many brain networks critical to neuroplasticity, working beneath the mind to shape the content of conscious awareness. Dysregulation of these networks can result from a myriad of factors—such as genetic predisposition, negative life experience, and hindered brain development. Such dysregulation will detrimentally affect how the brain processes our experiences, in ways the conscious mind cannot control.

This talk will discuss the functional architecture and electrophysiological underpinnings of neurofeedback therapy, together with a review of assessment and treatment methods. There will be (a) an examination of quantitative EEG as a parametric tool for assessing the regulation of brain systems function, (b) the role of neuropsychological assessment, and (c) coverage of theoretical viewpoints, methods, and applications of neurofeedback therapy, including its relationship to operant learning theory. The presentation will also discuss the relationship between neurotherapy and psychotherapy, emphasizing their complementary, respective roles in redressing dysregulation in the physical world of neurons and networks (neurotherapy) and the dysfunctional thoughts and feelings that can emerge as a result (psychotherapy). Examples of the application of neurofeedback therapy will be presented with an emphasis on Attention-deficit/Hyperactivity Disorder (ADHD).

Neurobiology of Attention-deficit/Hyperactivity Disorder

Mark Bellgrove, PhD
Professor in Cognitive Neuroscience and Larkin's Fellow, Monash University, Melbourne, Australia

Attention-deficit/Hyperactivity Disorder (ADHD) is a prevalent mental health condition of childhood that frequently persists into adulthood. In this presentation I will overview our current knowledge regarding the neurobiology of ADHD, surveying the genetic, pharmacological, brain imaging, and neuropsychological literatures. I will argue that despite significant advances in our understanding of the pathophysiological mechanisms of ADHD, research studies persist in studying ADHD as though it is a unitary construct. Advances in our understanding of the disorder and strategies for the appropriate targeting of medications to individuals with specific phenotypes must tackle the issue of clinical and underlying neurobiological heterogeneity. Some potential research methods to gain traction on this issue will be discussed.

QEEG and ERPs in ADHD Assessment, Diagnosis and Treatment

Yuri Kropotov, PhD
Director, Institute of the Human Brain, Russian Academy of Sciences, St. Petersburg, Russia
Professor II of the Norwegian University of Science and Technology, Trondheim, Norway

Modern neuroscience demonstrates that there are many reasons why people experience behavioral symptoms of Attention-deficit/Hyperactivity Disorder (ADHD), including existence of a focus near the Rolandic fissure, maturation lag, disruption of the basal ganglia thalamo-cortical circuits, etc. Resting-state spontaneous EEG and event-related potentials (ERPs) in different behavioral paradigms are among functional neuromarkers in neuroscience. It has been consistently shown that the ERP waves such as CNV and P300 fit the criteria for biomarkers: 1) have high test-retest reliability; 2) consistently reflect experimental manipulations in sensory and cognitive domains; and 3) discriminate ADHD from healthy population with quite large effect sizes. It's also a common view that ERP waves are the sum of activities from widely distributed cortical areas and must be decomposed into separate latent components with distinct localizations and different functional meanings.

This lecture presents 10 years of the author's experience of applying ERPs in clinical practice of ADHD. The experience includes studies on: 1) test-retest reliability of ERP latent components; 2) ERP neuromarkers of ADHD; 3) ERP indexes of neuropsychological domains such as energization, monitoring, task switching, etc.; 4) predicting effects and side-effects of Ritalin in ADHD population; 5) creating neurofeedback protocols for ADHD on the basis of ERP assessment; 6) creating tDCS protocols for ADHD on the basis of ERP assessment; 7) monitoring the effects of treatments by ERPs.

EEG Anomalies in ADHD: Linking EEG Activity with Mechanisms and Behavior

Adam R. Clarke, PhD[1], Robert J. Barry, PhD[1], Rory McCarthy, MD[2], and Mark Selikowitz, MD[2]
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Attention-deficit/Hyperactivity Disorder (ADHD) is one of the most common psychiatric disorders of childhood, affecting approximately 5% of primary school children. Almost all models of the disorder accept that the behavioral cluster which is ADHD results from an underlying central nervous system (CNS) dysfunction. However, the exact nature of this dysfunction is poorly understood. Several electrophysiological-based models of ADHD have been proposed and recent research has suggested that most are too simplistic in nature, and the underlying CNS dysfunctions are inaccurately labelled. Part of the problem results from the use of multiple bands in the analysis of the EEG, as this approach does not allow an understanding of the role of any discrete band on functioning. In a different approach, our group has been decomposing the EEG into single bands and relating anomalies in these bands to specific brain states (such as arousal) and to behavior. Results from a number of studies, and their implications for understanding the link between brain and behavior, will be discussed.

Neurofeedback, ADHD and Sleep (Part I) The NIMH-funded ADHD Research: 'The Definitive Trial' into the Efficacy of Neurofeedback in ADHD? (Part II)

Martijn Arns, PhD
Research Institute Brainclinics, Nijmegen, The Netherlands
Dept. of Experimental Psychology, Utrecht University, Utrecht, The Netherlands

Recent insights suggest an etiological contribution of sleep disorders in sub-groups of ADHD patients, specifically sleep-onset insomnia (Arns & Kenemans, 2012). Chronobiological treatments, such as melatonin and morning bright light, have demonstrated clinical effects in ADHD and we recently demonstrated an association between the worldwide prevalence of ADHD and solar intensity (Arns, van der Heijden, Arnold, & Kenemans, 2013), as a further indication for the role of circadian dysregulation and sleep in the etiology of ADHD. In relation to neurofeedback, it has been demonstrated that Sensori-Motor Rhythm (SMR) neurofeedback impacts on the sleep spindle circuitry (SSC) resulting in increased sleep spindle density (see Arns & Kenemans, 2012, for review). Overlap between the reticulo-thalamo-cortical SSC and the circadian network has been reported, suggesting overlap between neurofeedback and chronobiological treatments. The treatment effects on ADHD symptoms such as inattention, hyperactivity, and impulsivity thus arise as a result of normalized sleep, as will be demonstrated based on a recent study, where only for SMR neurofeedback it was found that improvement in sleep-onset latency mediated the improvements on inattention. For Theta/Beta

neurofeedback this was not found, suggesting specificity of SMR and Theta/Beta neurofeedback protocols in the treatment of ADHD.

In the second part of this presentation the current evidence level for various neurofeedback protocols in the treatment of ADHD and different efficacy designs will be reviewed (Arns, Heinrich, & Strehl, 2014), as well as the need, rationale, and strategy of the NIMH funded double-blind placebo controlled iCAN study (international Collaborative ADHD Neurofeedback study) that is currently recruiting 140 children with ADHD at Ohio State University and the University of North Carolina (The Collaborative Neurofeedback Group, 2013).

References

Arns, M., & Kenemans, J. L. (2012). Neurofeedback in ADHD and insomnia: Vigilance stabilization through sleep spindles and circadian networks. *Neuroscience and Biobehavioral Reviews, 44,* 183–194. http://dx.doi.org/10.1016/j.neubiorev.2012.10.006

Arns, M., van der Heijden, K. B., Arnold, L. E., & Kenemans, J. L. (2013). Geographic variation in the prevalence of attention-deficit/hyperactivity disorder: The sunny perspective. *Biological Psychiatry, 74*(8), 585–590. http://dx.doi.org/10.1016/j.biopsych.2013.02.010

Arns, M., Heinrich, H., & Strehl, U. (2014). Evaluation of neurofeedback in ADHD: The long and winding road. *Biological Psychology*, *95*, 108–115. http://dx.doi.org/10.1016/j.biopsycho.2013.11.013

The Collaborative Neurofeedback Group, Arnold, L. E., Arns, M., Conners, K., deBeus, R., Hirshberg, L., ... Monastra, V. (2013). A proposed multisite double-blind randomized clinical trial of neurofeedback for ADHD: Need, rationale, and strategy. *Journal of Attention Disorders*, *17*(5), 420–436. http://dx.doi.org/10.1177/1087054713482580

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