

Journal of Neurotherapy: Investigations in Neuromodulation, Neurofeedback and Applied Neuroscience

Selected Abstracts of Conference Presentations at the 2010 International Society for Neurofeedback and Research (ISNR) 18th Annual Conference, Denver, Colorado

Published online: 25 Nov 2010.

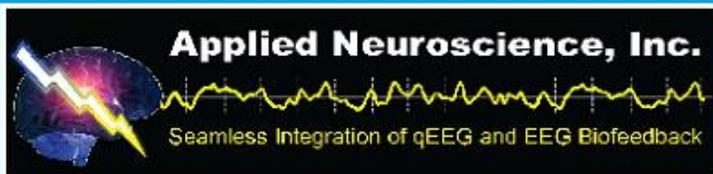
To cite this article: (2010) Selected Abstracts of Conference Presentations at the 2010 International Society for Neurofeedback and Research (ISNR) 18th Annual Conference, Denver, Colorado, *Journal of Neurotherapy: Investigations in Neuromodulation, Neurofeedback and Applied Neuroscience*, 14:4, 321-371, DOI: [10.1080/10874208.2010.523353](https://doi.org/10.1080/10874208.2010.523353)

To link to this article: <http://dx.doi.org/10.1080/10874208.2010.523353>

PLEASE SCROLL DOWN FOR ARTICLE

© International Society for Neurofeedback and Research (ISNR), all rights reserved. This article (the “Article”) may be accessed online from ISNR at no charge. The Article may be viewed online, stored in electronic or physical form, or archived for research, teaching, and private study purposes. The Article may be archived in public libraries or university libraries at the direction of said public library or university library. Any other reproduction of the Article for redistribution, sale, resale, loan, sublicensing, systematic supply, or other distribution, including both physical and electronic reproduction for such purposes, is expressly forbidden. Preparing or reproducing derivative works of this article is expressly forbidden. ISNR makes no representation or warranty as to the accuracy or completeness of any content in the Article. From 1995 to 2013 the *Journal of Neurotherapy* was the official publication of ISNR (www.isnr.org); on April 27, 2016 ISNR acquired the journal from Taylor & Francis Group, LLC. In 2014, ISNR established its official open-access journal *NeuroRegulation* (ISSN: 2373-0587; www.neuroregulation.org).

THIS OPEN-ACCESS CONTENT MADE POSSIBLE BY THESE GENEROUS SPONSORS



PROCEEDINGS OF THE 2010 ISNR CONFERENCE

Selected Abstracts of Conference Presentations at the 2010 International Society for Neurofeedback and Research (ISNR) 18th Annual Conference, Denver, Colorado

ORAL PRESENTATIONS

Low-Frequency Repetitive Transcranial Magnetic Stimulation (rTMS) Modulates Evoked-Gamma Frequency Oscillations in Autism Spectrum Disorder (ASD)

Joshua M. Baruth, MS

University of Louisville School of Medicine
<jmbaru01@louisville.edu>

It has been reported that individuals with Autism Spectrum Disorder (ASD) have abnormal reactions to the sensory environment and visuo-perceptual abnormalities. Electrophysiological research has provided evidence that gamma band activity (30–80 Hz) is a physiological indicator of the coactivation of cortical cells engaged in processing visual stimuli and integrating different features of a stimulus. A number of studies have found augmented and indiscriminative gamma band power at both early (i.e., evoked gamma) and late (i.e., induced gamma) stages of visual processing in ASD; this may be related to decreased inhibitory processing and an increase in the ratio of cortical excitation to inhibition. Low frequency or “slow” (=1 Hz) repetitive transcranial magnetic stimulation (rTMS) has been shown to increase inhibition of stimulated cortex by the activation of inhibitory circuits. We wanted

to test the hypothesis of gamma band abnormalities at early stages of visual processing in ASD by investigating relative evoked (i.e., ~100 ms) gamma power in a visual oddball task using Kanizsa illusory figures. Our results indicate that in individuals with ASD-evoked gamma activity is not discriminative of stimulus type, whereas in controls early gamma power differences between target and nontarget stimuli are highly significant. Following 12 sessions of bilateral “slow” rTMS treatment to the dorsolateral prefrontal cortex individuals with ASD showed significant improvement in discriminatory gamma activity between relevant and irrelevant visual stimuli with few, if any, side effects reported. We propose that slow rTMS may have increased cortical inhibitory tone and decreased the ratio of cortical excitation to inhibition, which improved discriminatory gamma activity at early stages of visual processing. We also found significant improvement in behavioral questionnaires (i.e., irritability, repetitive behavior) as a result of rTMS. Contrary to available pharmacological interventions, rTMS has shown significant benefits in treating core symptoms of ASD with few, if any, side effects.

REFERENCES

- Borojerd, B., Prager, A., Muellbacher, W., Cohen, L. G. (2000). Reduction of human visual cortex

- excitability using 1-Hz transcranial magnetic stimulation. *Neurology*, *54*, 1529–1531.
- Brown, C., Gruber, T., Boucher, J., Rippon, G., & Brock, J. (2005). Gamma abnormalities during perception of illusory figures in autism. *Cortex*, *41*, 364–376.
- Charman, T. (2008). Autism spectrum disorders. *Psychiatry*, *7*, 331–334.
- Grice, S. J., Spratling, M. W., Karmiloff-Smith, A., Halit, H., Csibra, G., De Haan, M., et al. (2001). Disordered visual processing and oscillatory brain activity in autism and Williams syndrome. *NeuroReport*, *12*, 2697–2700.
- Pascual-Leone, A., Walsh, V., & Rothwell, J. (2000). Transcranial magnetic stimulation in cognitive neuroscience—virtual lesion, chronometry, and functional connectivity. *Current Opinion in Neurobiology*, *10*, 232–237.
- Tallon-Baudry, C., Bertrand, O., Delpuech, C., & Pernier, J. (1996). Stimulus specificity of phase-locked and non-phase-locked 40 Hz visual responses in human. *Journal of Neuroscience*, *16*, 4240–4249.

The Effects of Neurofeedback in Children with Autism: Results of a Randomized Single Blind Attention Placebo-Controlled Study

Mirjam Kouijzer, MSc
Radboud University Nijmegen
<m.kouijzer@pwo.ru.nl>

While writing this conference abstract in March 2010, a study investigating the effects of neurofeedback in autism is running in the Netherlands. After accomplishing two smaller studies with promising results (Kouijzer, de Moor, Gerrits, Congedo, & van Schie, 2009; Kouijzer, van Schie, de Moor, Gerrits, & Buitelaar, in press), we now try to prevent our results from attention and expectancy biases. In addition to the EEG feedback group and the waiting list control group, we included a Skin Conductance (SC) feedback group. All participants of the present study ($n = 41$) were pretested with EEG and executive function tasks and parents and teachers filled out behavior questionnaires. Then, the EEG and SC feedback groups had identical sessions of EEG or SC feedback without knowing which type of feedback they received. EEG and SC feedback sessions were identical with electrodes attached to the scalp (measuring EEG) and to the fingers (measuring SC). After 40 sessions of EEG or SC feedback, all participants were retested with EEG

and executive function tasks, and parents and teachers filled out behavior questionnaires again. Data collection ends in July 2010. We hope that the results of this study can be presented for the first time at the ISNR conference in Denver.

REFERENCES

- Kouijzer, M. E. J., De Moor, J. M. H., Gerrits, B. J. L., Buitelaar, J. K., & Van Schie, H. T. (2009). Long-term effects of neurofeedback treatment in autism. *Research in Autism Spectrum Disorders*, *3*, 496–501.
- Kouijzer, M. E. J., De Moor, J. M. H., Gerrits, B. J. L., Congedo, M., & Van Schie, H. T. (2009). Neurofeedback improves executive functioning in children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, *3*, 145–162.
- Kouijzer, M. E. J., Van Schie, H. T., De Moor, J. M. H., Gerrits, B. J. L., & Buitelaar, J. K. (in press). Neurofeedback treatment in autism. Preliminary findings in behavioral, cognitive, and neurophysiological functioning. *Research in Autism Spectrum Disorders*. Advance online publication. doi:10.1016/j.rasd.2009.10.007

EEG Connectivity Assessment and Training: A Multichannel Directed Information Flow Perspective

David Joffe, BA
EEG Dynamics
<dj2242@aol.com>

Classical coherence analysis methods provide insufficient information to explicitly characterize the direction of information flow between two or more EEG scalp electrode locations, as a function of frequency. In addition, it is impossible to determine the extent to which the coherence measured between any two particular scalp electrode sites may be due to the influence of one or more additional scalp electrode sites, using coherence analysis alone. Additional knowledge in both of these areas may improve a neurotherapist's ability to assess QEEG dynamics more completely, and also improve the efficacy of treatment.

One class of methods that may be employed to address both of these concerns

in the context of multichannel QEEG assessment and neurofeedback training, involves what are known as multivariate autoregressive (MVAR) estimators. However, direct measures of EEG information flow direction and influence based on MVAR methods are not currently utilized in either QEEG assessment or neurofeedback due to the lack of available turnkey research and/or clinical tools, as well as a lack of familiarity regarding the potential clinical efficacy of these tools, on the part of neurotherapists.

Building on his 2008 *Journal of Neurotherapy* article “Connectivity Assessment and Training: A Partial Directed Coherence Approach,” the author focuses on three MVAR-derived measures known as Granger Causality, Partial Directed Coherence, and the Directed Transfer Function, using intuitive graphical displays to convey the potential power of these methods for both QEEG assessment and neurofeedback training. Also included are examples based on multichannel EEG data sets for the purposes of comparing and contrasting the unique perspectives afforded by each of these three methods, as well as highlighting the strengths and weaknesses of the three methods with respect to classical coherence analysis.

REFERENCE

Joffe, D. (2008). Connectivity assessment and training: A partial directed coherence approach. *Journal of Neurotherapy*, 12, 111–122.

Single-Case Experimental Designs: A Valuable Method for Evaluating Neurofeedback in Clinical Practice

Matthew Nock, PhD
Harvard University
<nock@wjh.harvard.edu>

This presentation will introduce single-case experimental designs, distinguish them from case studies, describe the conceptual basis for such designs, outline in detail three different types of single-case designs that are likely to be valuable for use in neurofeedback research, and provide examples of such

designs from the literature in order to illustrate their use and value as an approach to testing the causal relations between treatment and outcomes.

Alcohol Addiction: A Clinical Pathophysiological Approach

Dirk De Ridder, MD, PhD
University Hospital Antwerp
<dirk.de.ridder@neurosurgery.be>

It has recently become clear that alcohol addiction might be related to a brain dysfunction, in which a genetic background and environmental factors shape brain mechanisms involved with alcohol consumption. Craving, a major component determining relapses in alcohol abuse, has been linked to abnormal activity in the orbitofrontal cortex, dorsal anterior cingulate cortex (dACC), and amygdala.

rTMS targeting the dACC using a double cone coil in an attempt to suppress very severe intractable alcohol craving can be applied. Functional imaging studies consisting of fMRI and resting state EEG can be performed before rTMS, after successful rTMS, and after unsuccessful rTMS.

Craving was associated with beta activity and connectivity between the dACC and PCC, which disappeared after successful rTMS. Cue induced worsening of craving activated the vmPFC and PCC on fMRI as well as the nucleus accumbens area, DMPFC and inferior parietal area, with associated suppression of the VLPFC.

Relapse was associated with recurrence of ACC and PCC activity, but in gamma band and nucleus accumbens and DMPFC activity on fMRI.

Linking functional imaging changes to craving intensity permits to build a pathophysiological model of alcohol craving that can be applied clinically using neuromodulation in the broad sense, whether by neurofeedback, rTMS, tDCS, or implants.

REFERENCES

Degenhardt, L., Chiu, W. T., Sampson, N., Kessler, R. C., Anthony, J. C., Angermeyer, M., et al. (2008).

- Toward a global view of alcohol, tobacco, cannabis, and cocaine use: Findings from the WHO World Mental Health Surveys. *PLoS Med.*, 5, e141.
- Grant, B. F., Dawson, D. A., Stinson, F. S., Chou, S. P., Dufour, M. C., & Pickering, R. P. (2004). The 12-month prevalence and trends in *DSM-IV* alcohol abuse and dependence: United States, 1991–1992 and 2001–2002. *Drug and Alcohol Dependency*, 74, 223–234.
- Hayward, G., Mehta, M. A., Harmer, C., Spinks, T. J., Grasby, P. M., & Goodwin, G. M. (2007). Exploring the physiological effects of double-cone coil TMS over the medial frontal cortex on the anterior cingulate cortex: An H₂(15)O PET study. *European Journal of Neuroscience*, 25, 2224–2233.
- Koob, G. F. (2006). The neurobiology of addiction: A neuroadaptational view relevant for diagnosis. *Addiction*, 101(Suppl. 1), 23–30.
- Koob, G. F., & le Moal, M. (2006). *Neurobiology of addiction*. Amsterdam: Academic Press-Elsevier.

Multimodal Brain Imaging: Combining Brain Stimulation and Functional Neuroimaging to Understand a Changing Brain

Alvaro Pascual-Leone, MD, PhD

Berenson-Allen Center for Noninvasive Brain Stimulation, Harvard Medical School
<apleone@bidmc.harvard.edu>

The human brain is intrinsically plastic, changing across the lifespan. Such changes may prove adaptive and lead to functional benefits, or may be the very cause of disease and disability. The challenge is to learn enough about the mechanisms of brain plasticity to guide them, enhancing some and suppressing others, to promote the best functional outcome for a given individual. This requires insights about causal relations between brain activity and behavior.

Functional brain imaging provides correlational information about brain activity and behavior. Establishing causal links requires intervention and brain stimulation techniques enable this, thus offering the potential of adding another dimension to functional brain imaging. Multimodal brain imaging, combining brain imaging and neurophysiologic measuring and noninvasive stimulation methodologies, allows the establishment of a causal relationship and a precise chronometry between regional brain

activation and behavior. Application of similar methods in animal models enables true translational mouse-to-human approaches, bridging mechanistic and clinical investigation.

Noninvasive brain stimulation with Transcranial Magnetic Stimulation (TMS) or Transcranial Direct Current Stimulation (tDCS) can interfere with activity in a specific cortical brain region and modulate brain network dynamics. These techniques can both be combined with brain mapping methods. For example, PET or fMRI can identify information about brain areas associated with behavior and TMS can transiently deactivate a region of the brain, thus creating a “virtual patient” and explore causal relations. EEG, MEG, and ERPs can provide further chronometric information. Repetitive TMS or tDCS allows the noninvasive modulation of activity in a specified cortical target in the brain convexity and its functionally connected cortico-subcortical neural network. MRI and EEG can guide such application of rTMS. Depending on stimulation parameters cortical excitability of the directly targeted brain region can be increased or decreased beyond the duration of the rTMS train. Network effects can result in behavioral benefits through paradoxical functional facilitation, induction of desirable plastic changes, or release of specific neurotransmitters. Such combinations of noninvasive brain stimulation and brain mapping methods can lead to clinically relevant therapeutic effects in neuropsychiatry and neurorehabilitation and provide unique insights into brain plasticity mechanisms in health and disease across the lifespan.

Cognitive Improvement Following Z-Score Neurofeedback Therapy of 20 Moderate to Severe Brain Injury Patients: Preliminary Results of a Pilot Study

Victor Zelek, PhD

Northeast Center for Special Care
<victorzelek@msn.com>

Although neurofeedback (EEG Biofeedback) has been shown to be an effective treatment

modality for a variety of psycho-cognitive disorders, its application for brain injury patients has been slow and mostly limited to mild TBI. Lengthy treatments, poor compliance, and inconsistent results have been cited among the reasons. The current prospective pilot study examined Z-score neurofeedback treatment efficacy in improving cognitive functioning of 20 adult patients with a history of moderate to severe brain injury (defined as duration of unconsciousness more than 30 min and 24 hr, respectively). All patients were residing at a subacute inpatient brain injury rehabilitation facility (Northeast Center for Special Care). They were 3 months to several years post-injury. The etiology of brain injury included TBI, CVA, Infectious Encephalopathy, and Anoxic Encephalopathy. Their cognitive abilities were evaluated using Repeatable Battery for the Assessment of Neuro-psychological Status (RBANS) before and after Neurofeedback treatment course. All had QEEG analyses done before and after treatment. Neurofeedback was given 2 to 3 times a week for the total of 20 to 30 sessions. Cognitive improvement was measured using RBANS. Electrophysiological improvement was reflected by the normalization of Z-score Amplitude and Coherence values that were abnormal at the outset of treatment.

For most patients in the study RBANS Total Scale Score (TSS) improved following the course of neurofeedback, but only one third showed statistically significant cognitive improvement. It is estimated that with a greater number of neurofeedback sessions the patients are likely to continue making cognitive gains. The cognitive improvement correlated with the normalization of brain-wave Amplitude and Coherence but not always at the injury site. TBI patients showed more improvement and EEG normalization than other diagnostic categories in the study. Brainwave Z-score normalization patterns were examined from session to session as well as within each session. The study also focused and provided recommendations on practical ways to overcome many challenges of using neurofeedback with moderate and severe brain injury patients.

REFERENCES

- Byers, A. P. (1995). Neurofeedback therapy for a mild head injury. *Journal of Neurotherapy*, 1(1), 22–37.
- Doppelmayr, M., Nosko, H., Pecherstorfer, T., & Fink, A. (2007). An attempt to increase cognitive performance after stroke with neurofeedback. *Biofeedback*, 35, 126–130.
- Duff, J. (2004). The usefulness of quantitative EEG (QEEG) and neurotherapy in the assessment and treatment of post-concussion syndrome. *Clinical EEG & Neuroscience*, 35, 198–209.
- Ham, L. P., & Packard, R. C. (1996). A retrospective, follow-up study of biofeedback-assisted relaxation therapy in patients with posttraumatic headache. *Biofeedback & Self-Regulation*, 21, 93–104.
- Hoffman, D. A., Stockdale, S., & Van Egren, L. (1996a). EEG neurofeedback in the treatment of mild traumatic brain injury [Abstract]. *Clinical Electroencephalography*, 27(2), 6.
- Hoffman, D. A., Stockdale, S., & Van Egren, L. (1996b). Symptom changes in the treatment of mild traumatic brain injury using EEG neurofeedback [Abstract]. *Clinical Electroencephalography*, 27(3), 164.
- Keller, I. (2001). Neurofeedback therapy of attention deficits in patients with traumatic brain injury. *Journal of Neurotherapy*, 5(1,2), 19–32.
- Laibow, R. E., Stubblebine, A. N., Sandground, H., & Bounias, M. (2001). EEG Neurobiofeedback treatment of patients with brain injury: Part 2: Changes in EEG parameters versus rehabilitation. *Journal of Neurotherapy*, 5(4), 45–71.
- Thatcher, R. W. (2000). EEG operant conditioning (biofeedback) and traumatic brain injury. *Clinical Electroencephalography*, 31(1), 38–44.
- Thornton, K. E., & Carmody, D. P. (2005). Electroencephalogram biofeedback for reading disability and traumatic brain injury. *Child & Adolescent Psychiatric Clinics of North America*, 14(1), 137–162.
- Thornton, K. E., & Carmody, D. P. (2008). Efficacy of traumatic brain injury rehabilitation: Interventions of QEEG-guided biofeedback, computers, strategies, and medications. *Applied Psychophysiology & Biofeedback*, 33(2), 101–124.
- Tinius, T. P., & Tinius, K. A. (2001). Changes after EEG biofeedback and cognitive retraining in adults with mild traumatic brain injury and attention deficit disorder. *Journal of Neurotherapy*, 4(2), 27–44.
- Walker, J. E. (2007). A neurologist's experience with QEEG-guided neurofeedback following brain injury. In J. R. Evans (Ed.), *Handbook of neurofeedback*, (pp. 353–361). Binghamton, NY: Haworth Medical.

Laplacian Z-Score Neurofeedback: A Unique Option in The Realm of Multi-Channel Z-Score Neurofeedback

Nancy Wigton, MA
Applied Neurotherapy Center
<nwig@cox.net>

This presentation reviews a newly available Z-Score Neurofeedback technique whereby the real-time Laplacian montage Z-score values are able to be directly trained. Various case studies are presented, complete with pre- and post-QEEG data as well as clinical outcome measures when available.

Until 2006 the main Neurofeedback approach was limited to 2-channel amplitude training. In 2006 a new 4-channel Neurofeedback technique, called Z-Score Neurofeedback (ZNF), became available that uses real-time Z-scores from an age-matched normative database. Since its introduction many clinicians report that the ZNF approach provides for faster clinical outcomes. However, until recently, the maximum number of channels that could be trained at one time was 4 and training was limited to the linked-ears normative database.

The use of Multi-Channel ZNF greatly expands the number of scalp locations and measures and includes the ability to train real-time Z-scores using not only linked-ears montage data (as well as coherence and phase measures) but also the Laplacian montage data. In cases where the Laplacian montage data reveals more relevant clinical issues to address, it is now possible to directly train these values. Although results are preliminary, and more study is needed to replicate results, this new approach may turn out to give the clinician a great advantage in more efficiently addressing clinical issues within the realm of Multi-Channel ZNF.

REFERENCES

- Collura, T. F. (2008a, April). Whole-head normalization using live Z-scores for connectivity training, Part 1. *NeuroConnections*, 12–18.
- Collura, T. F. (2008b, July). Whole-head normalization using live Z-scores for connectivity training, Part 2. *NeuroConnections*, 9–12.
- Collura, T. F., Guan, J. G., Tarrant, J., Bailey, J., & Starr, F. (2010). EEG Biofeedback Case Studies Using Live Z-Score Training and a Normative Database. *Journal of Neurotherapy*, 14, 22–46.
- Collura, T. F., Thatcher, R. W., Smith, M. L., Lambos, W. A., & Stark, C. R. (2009). EEG biofeedback training using live Z-scores and a normative database. In T. Budzynski, H. Budzynski, J. Evans, & A. Abarbanel (Eds.), *Introduction to QEEG and neurofeedback: Advanced theory and applications* (pp. 103–141). New York, NY: Academic Press.
- Saab, M. (2008, April). Z-score biofeedback with thought technology's infinity system. *NeuroConnections*, 26–30.
- Stark, C. R. (2008, April). Consistent dynamic Z-score patterns observed during Z-score training sessions. *NeuroConnections*, 37–38.
- Tegan, E. (2008, July). Z-score training case review of severe mood instabilities. *NeuroConnections*, 33–34.
- Thatcher, R. W. (2008). Z-Score EEG Biofeedback: Conceptual Foundations. *NeuroConnections*, April, 9–11.
- Thatcher, R. W. (2009). *Multi-channel Z-score EEG biofeedback: Laplacian, average reference, phase reset and discriminant functions*. Paper presented at the 17th Annual ISNR Conference, Indianapolis, IN.
- Wigton, N. (2008). *Does Z-score NF work better than non Z-score NF?* Poster presented at the 16th Annual ISNR Conference, San Antonio, TX.
- Wigton, N. (2009). First impressions of neuroguide real-time Z-score training. In J. Demos (Ed.), *Getting started with dynamic Z-score training* (pp. 81–89). Neurofeedback of S.VT LLC.

What Are We Training When We Train SMR?

Michael O'Bannon, PhD
Private practice
<mob@mindspring.com>

Neurofeedback training of the sensory-motor rhythm (SMR; 12–15 Hz along the sensorimotor strip) has a long and well-established history. It is one of the most commonly used types of training in clinical practice. Current software and hardware allow wide variation in the actual implementation of SMR training protocols in both clinical practice and the laboratory, however. These variations may produce unintended consequences for outcomes of treatment and research.

This presentation reviews SMR protocol variations that arise from differences in bandpass filter characteristics, choices of feedback signal “inhibit” bands, and use of autothresholding. In addition, it provides an analysis of the vulnerability of traditional SMR protocols to high amplitude out-of-band signals that are often present in the typical EEG records of clients.

First, responses of several commercial software/hardware systems to identical EEG records will be examined and their differences compared. Second, high-resolution contingency analyses of several client sessions will be presented to differentiate EEG events that trigger feedback signals over the course of SMR training. These results illustrate some of the unanticipated responses of the EEG to traditional SMR protocols designs. Finally, several recommendations will be offered to assist selection of appropriate protocols in the future.

REFERENCES

- Collura, T. F. (2000). *Filter comparison—BrainMaster and EEG Spectrum systems*. Bedford, PH: BrainMaster Technologies.
- Serman, M. B., Macdonald, L. R., & Stone, R. K. (1974). Biofeedback training of the sensorimotor EEG rhythm in man: Effects on epilepsy. *Epilepsia*, *15*, 395–416.
- Vernon, D., Egnor, T., Cooper, N., Compton, T., Neilands, C., Sheri, A., et al. (2003). The effect of training distinct neurofeedback protocols on aspects of cognitive performance. *International Journal of Psychophysiology*, *47*, 75–85.

Clinical Red Flags of Undiagnosed Mild Traumatic Brain Injuries

Kay Sheehan, EdD
ADD Center of Colorado
<drkms1@gmail.com>

Introduction

Many psychotherapists and neurofeedback providers have clients who are unsuspecting of having sustained a mild traumatic brain injury (TBI). Some of these

clients, at some point in their lives, may even have gone to an emergency room because an incident occurred in which a mild TBI needed to be ruled out, and were told their MRI was fine and so were they. Other clients, as well as any previous medical and mental health providers, may be completely unsuspecting of a possible mild TBI. Uncovering probable mild TBI(s) is important in indicating the need for further assessment, properly diagnosing the client (correcting misdiagnoses when appropriate), and in providing the proper treatment. It is also frequently a relief for the client in understanding the reason for his or her symptoms.

Method

Over many years of doing psychotherapy, biofeedback and neurofeedback evaluations and treatment, red flags indicating probable mild TBIs have become apparent in the initial evaluation of clients with various presenting problems. A brief overview of mild TBIs is presented, the red flags to look for in children/adolescents and adults are discussed, and cases illustrating the initial red flags and subsequent assessments are presented.

Results and Conclusions

There are some patterns in presenting problems/history of children/adolescents and adults that are red flags of possible mild TBIs indicating the need for further assessment.

REFERENCES

- Bounias, M., Laibow, R. E., Stubbelbine, A. N., Sandground, H., & Bonaly, A. (2002). EEG-neurobiofeedback treatment of patients with brain injury Part 4: Duration of treatments as a function of both the initial load of clinical symptoms and the rate of rehabilitation. *Journal of Neurotherapy*, *6*, 23–38.
- Duff, J. (2004). The usefulness of quantitative EEG (QEEG) and neurotherapy in the assessment and treatment of post-concussion syndrome. *Clinical EEG & Neuroscience*, *35*, 198–209.
- Gasquoine, P. G. (1997). Post concussion symptoms. *Neuropsychology Review*, *7*, 77–85.

- Prigatano, G. P. (1992). Personality disturbances associated with traumatic brain injury. *Journal of Consulting and Clinical Psychology, 60*, 360–368.
- Reitan, R. M., & Wolfson, D. (1999). Two faces of mild head injury. *Archives of Clinical Neuropsychology, 14*, 191–202.
- Thatcher, R. W., Walker, R. A., Gerson, I., & Geisler, F. H. (1989). EEG discriminant analysis of mild head trauma. *Electroencephalography and Clinical Neurophysiology, 73*, 94–106.
- Tinius, T. P., & Tinius, K. A. (2000). Changes after EEG biofeedback and cognitive retraining in adults with mild traumatic brain injury and attention deficit hyperactivity disorder. *Journal of Neurotherapy, 4*, 27–44.

Atypical Migraine Aura: Clinical Presentation and Clinical Implications

Jeffrey Carmen, PhD
Private practice
<carmen5272@aol.com>

Introduction

Migraine headaches are very common, with estimates as high as 20% of the normal population. They are sometimes preceded by an aura that is obvious to the migraine sufferer. However, the aura can present itself in uncommon ways that, although still important, are more difficult to detect. This oral presentation covers these less typical aura presentations.

Method

There will be a 15-min lecture with PowerPoint slides, followed by a 5-min Q/A period.

Results

Participants will acquire a greater understanding of the subtle variables of migraine brain events.

Conclusion

The migraine aura is a significant part of the migraine mechanism. It is critical to understand the aura variables in order to

track migraine frequency and intensity as well as intervention progress. This presentation provides enough information to allow the participant to pursue further information within the headache literature.

REFERENCES

- International Headache Society Classification System. (n.d.). Available from <http://ihs-classification.org/en/>
- Sacks, O. (1992). *Migraine*. Berkeley: University of California Press.

Gorak Video Game in the Resocialization of Infants in Situations of Social Risk

Dirce Maria Navas Perissinotti, DSc, and Yusaku Soussumi, MD
São Paulo Federal University
<dircelko@uol.com.br>

Introduction

This study examines the humanistic video game Gorak, created by the Center of Study and Investigation in Neuro-psychoanalysis to Virada Project of Rukha Institute, based on Soussumi's Theory, applied as adjuvant approach to reintegrate children in social risk. Prior studies indicate that the performance in tasks mediated by affective functions increase cognitive tasks. Gorak was created to reflect the affective relations' training in the adjustment of social abilities, socialization and social adjustment, empathy, social assertiveness coping, self-control, and better social participation. The game would allow better attentional processing and verbal cognition related to the prefrontal cortex.

Objective

The purpose is to test the effectiveness of the Gorak applied in children under social risk, particularly to exam cognitive activity (WISC-III), social abilities (IHS-Del Prete & Del Prete), and EEG signals (bipolar Fp1-Fp2).

Method

Thirty children (6–9 years of age) were evaluated pre- and postplaying, in a period of 10 weeks, being applied the instruments cited. Gorak was presented to the children twice weekly across 20 sessions of 1 hr each in a controlled environment, each child utilizing one computer individually.

Results

The results showed strong statistical positive correlation and significance for the subtests Picture Completion, Similarities, Picture Arrangement, and Comprehension. In the HIS, and also was obtained for items involving negotiation, persuasion, and acceptance, not meaning passivity, but better capacity to choose the adequate social coping. Discrete cognitive difficulties related to the attentional processing (Theta in Fp1-Fp2) arose. Inhibition of attention was associated with the increase on Theta and electric activity slowed. In the posttest there was discrete improvement of the standards of Theta.

Discussion

We conclude that Gorak enabled the children to improve the capacity to establish logical relations and to form verbal concepts and provided improvement of the capacity to synthesize and integrate knowledge. Improved acknowledgment of social relationship rules and facility of argument was also noticed.

REFERENCES

- Arroyo-Palacio, J., & Romano, D. M. (2008). Towards a standardization in the use of physiological signals for affective recognition systems. In A. J. Spink, M. R. Ballintijn, N. D. Bogers, F. Grieco, L. W. S. Loijens, L. P. J. J. Noldus, et al. (Eds.), *Proceedings of measuring behavior 2008* (pp. 121–124). Maastricht, The Netherlands.
- Beeli, G., Casutt, G., Baumgartner, T., & Jäncke, L. (2008). Modulating presence and impulsiveness by external stimulation of the brain. *Behavioral and Brain Functions*. Retrieved from <http://www.behavioralandbrainfunctions.com/content/4/1/33>
- Fonagy, P. (1999). Guest editorial: Memory and therapeutic action. *International Journal of Psycho-Analysis*, *80*, 215–223.
- Gilleade, K. M., Dix, A., & Allanson, J. (2006). Affective videogames and modes of affective gaming: Assist me, challenge me, emote me. *Proceedings of DiGRA 2005 Conference: Changing Views – Worlds in Play*. Vancouver: University of Vancouver.
- Soussumi, Y. (2006). Tentativa de Integração entre algumas Concepções Básicas da Psicanálise e da Neurociência. *Psic. Clin., Rio De Janeiro*, *18*(1), 63–82.
- Wickramasekera, I. (1991). The unconscious, somatization, psychophysiological psychotherapy and threat perception: Footnotes to cartography of the unconscious mind. *Biofeedback*, *19*, 18–23.

Effectiveness of Neurofeedback in Youth with ADHD Problems and Comorbid Disorders

Marleen Bink, MSc, and Chijs van Nieuwenhuizen, PhD
Tilburg University
<m.bink@ggze.nl>

Background

Youngsters in (forensic) mental health care often display ADHD-problems with inattention and/or hyperactivity-impulsivity. A majority of them also experience problems on other domains. Co-occurrence of externalizing disorders and/or internalizing disorders is the rule rather than the exception. At this moment, best practices for treatment of ADHD consist of behavioural intervention and medication. But this best practice appears to be less effective for youngsters with comorbid disorders. In the majority of the youngsters with ADHD an underactivity of the frontal and central brain region can be observed. Neurofeedback is a training method which intends to (partially) correct this brain activity by giving direct feedback to the brain.

Methods

The objective is to investigate whether neurofeedback is an effective intervention

for youngsters with AD(H)D-problems and comorbid disorders. This is done by looking at an ongoing study with a randomized controlled design. The aim is to include 100 youngsters in the experimental condition and 50 youngsters in the control condition. In this study, the experimental condition consists of treatment as usual in combination with 40 neurofeedback sessions. These sessions aim to inhibit theta (4–7 Hz) activity and reward beta (12–15 Hz) activity. The control condition consists of treatment as usual.

Results

Measurements are taken pretreatment (t1), direct posttreatment (t2), 6 months posttreatment (t3), and 1 year posttreatment (t4). At t1 thru t4, EEG-measurements, neuropsychological tests, clinical interviews, and/or behavioural questionnaires are administered. In this presentation, preliminary pre- and direct postmeasurements of the first inclusion group are presented.

Conclusion

The hypothesis is that neurofeedback will improve the capability of the brain to process information and will reduce attention, hyperactivity, and impulsivity symptoms.

REFERENCES

- Angold, A., Costello, E. J., & Erkanli, A. (1999). Comorbidity. *Journal of Child Psychology Psychiatry and Allied Disciplines*, *40*, 57–87.
- Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. *Psychological Bulletin*, *121*(1), 65–94.
- Barkley, R. A. (2006). Comorbid disorders, social and family adjustment, and subtyping. In R. A. Barkley (Ed.), *Attention-deficit hyperactivity disorder: a handbook for diagnosis and treatment*, (pp. 184–218). New York: Guilford.
- Barkley, R. A., Fischer, M., Smallish, L., & Fletcher, K. (2004). Young adult follow-up of hyperactive children: Antisocial activities and drug use. *Journal of Child Psychology and Psychiatry*, *45*, 195–211.
- Barry, R. J., Clarke, A. R., & Johnstone, S. J. (2003). A review of electrophysiology in attention deficit/hyperactivity disorder: I. Qualitative and quantitative electroencephalography. *Clinical Neurophysiology*, *114*, 171–183.
- Clarke, A. R., Barry, R. J., McCarthy, R., & Selikowitz, M. (2001a). EEG-defined subtypes of children with attention-deficit/hyperactivity disorder. *Clinical Neurophysiology*, *112*, 2098–2105.
- Clarke, A. R., Barry, R. J., McCarthy, R., & Selikowitz, M. (2001b). Excess beta activity in children with attention-deficit/hyperactivity disorder: An atypical electrophysiological group. *Psychiatry Research*, *103*, 205–218.
- Lubar, J. F. (1997). Neocortical dynamics: Implications for understanding the role of neurofeedback and related techniques for the enhancement of attention. *Applied Psychophysiology and Biofeedback*, *22*, 111–126.

Adult ADHD: Physiological Arousal During Resting State and Task Conditions

Marie Gonzales, BS, Sarah Wyckoff, MA, and Ute Strehl, PD, PhD, MSc
University of Tübingen
<mvg03@cox.net>

Objective

Recent research on Attention-Deficit/Hyperactivity Disorder (ADHD) has focused on central nervous system (CNS) arousal, QEEG Phenotypes, and EEG vigilance models. Distinct and stable patterns of EEG activity have emerged using these models and various subtypes of ADHD identified. However, the assessment and profile of peripheral physiological measures of arousal in ADHD has been less consistent and requires further investigation. Mangina and Beuzeron-Mangina (1992) reported that children and adolescents with learning disabilities/ADHD have an impaired regulation and asymmetries in electrodermal response during cognitive tasks compared to controls subjects. In a recent study Barry, Clarke, Johnstone, McCarthy, and Selikowitz (2009) reported no significant correlation between theta/beta ratios and skin conductance

level (SCL) in an adolescent ADHD population. However, reduced SCL, alpha, and beta power was observed in the ADHD group compared with control subjects. Finally, analysis of EEG Vigilance in a childhood ADHD population indicated that individuals with ADHD have more frequent vigilance state shifts and tend to spend more time in lower vigilance stages (Sander, Arns, Olbrich, & Hegerl, in press). EEG Vigilance and heart rate was accessed in a control population and the average heart rate decreased as participants entered the lower arousal/vigilance stages (Olbrich et al., 2009). Additional research is needed to identify the baseline and task specific physiological response in an adult ADHD population. We hypothesize that adults ADHD will exhibit reduced physiological arousal (reduced heart rate and parasympathetic dominance of HRV and EDR variables) compared to controls during resting state and cognitive/vigilance tasks.

Methods

Heart rate, heart rate variability (HRV), respiration rate, and electrodermal response (EDR) activity was investigated as a function of CNS arousal/vigilance during resting state, CNV task, and auditory oddball task in two groups of adults (18+ years old), with and without ADHD. Adult ADHD participants met *DSM-IV* criteria for combined, hyperactive, or attention type ADHD. Participants in both groups reported no additional serious physical, neurological, or psychiatric disorders, had a Full-Scale IQ greater than 80, and were right hand dominant. Arousal was defined in terms sympathetic and parasympathetic dominance in relation to heart rate, respiration, HRV, and GSR. Analysis of physiological measures was conducted for both groups and conditions.

Results

This investigation is part of a long-term treatment study currently in progress. The most current results in relation to the

pretreatment physiological measure of clinical and control participants will be presented at the time of the presentation.

Conclusion

Specific findings will be discussed and implication in the current treatment study and future research will be explored.

REFERENCES

- Barry, R. J., Clarke, A. R., Johnstone, S. J., McCarthy, R., & Selikowitz, M. (2009). Electroencephalogram theta/beta ratio and arousal in attention deficit/hyperactivity disorder: Evidence of independent processes. *Biological Psychiatry*, *66*, 398–401.
- Mangina, C. A., & Beuzeron-Mangina, J. H. (1992). Psychophysiological treatment for learning disabilities: Controlled research and evidence. *International Journal of Psychophysiology*, *12*, 243–250.
- Olbrich, S., Mulert, C., Karch, S., Trenner, M., Leicht, G., Pogarell, O., et al. (2009). EEG-vigilance and BOLD effect during simultaneous EEG/fMRI measurement. *NeuroImage*, *45*, 319–332.
- Sander, C., Arns, M., Olbrich, S., & Hegerl, U. (in press). EEG-vigilance and response to stimulants in pediatric patients with attention deficit/hyperactivity disorder.

Theta/Beta and SCP Training in Children with Attention-Deficit/Hyperactivity Disorder: Behavioral and Neurophysiological Results from a Randomized Controlled Trial

Hartmut Heinrich, PhD
University of Erlangen
<hheinri@arcor.de>

Neurofeedback (NF) could help to improve attentional and self-management capabilities in children with ADHD. In a randomized controlled trial, we evaluated the clinical efficacy of neurofeedback training using an attention skills training as control condition. We also compared slow cortical potential (SCP) training, which addresses phasic regulation of cortical excitability, to theta/beta training both at the behavioral and the neurophysiological level.

Ninety-four children with ADHD, aged 8 to 12 years, either completed 36 sessions of NF training ($n = 59$) or a computerized attention skills training ($n = 35$). NF training consisted of one block of theta/beta training and one block of SCP training, each block comprising 18 units of 50 min (balanced order).

At the behavioral level, NF was superior to the control training concerning core ADHD symptomatology as well as associated domains. For the primary outcome measure (improvement in the FBB-HKS total score, parent ratings), the effect size was .60. For theta/beta and SCP training, comparable improvements were observed.

At the neurophysiological level (resting EEG, event-related potentials during the attention network test), specific effects for the two NF protocols could be demonstrated. For theta/beta training, for example, decrease of theta activity in the EEG was associated with a reduction of ADHD symptomatology. SCP training was accompanied, for example, by an increase of the contingent negative variation in the attention network test, that is, children were able to allocate more resources for preparation. EEG- and ERP-based predictors were found.

Future studies should address inter alia how to optimize (individualize) neurofeedback training, that is, which training protocol (or combination of protocols) should be used for a particular child.

Acknowledgments

This study was funded by the German Research Foundation (HE4536/2, MO 726/2, RO 698/4).

REFERENCES

- Gevensleben, H., Holl, B., Albrecht, B., Vogel, C., Schlamp, D., Kratz, O., et al. (2009). Is neurofeedback an efficacious treatment for ADHD? A randomized controlled clinical trial. *Journal of Child Psychology and Psychiatry*, 50, 780–789.
- Heinrich, H., Gevensleben, H., & Strehl, U. (2007). Annotation: Neurofeedback—Train your brain to train behaviour. *Journal of Child Psychology and Psychiatry*, 48, 3–16.

The Brain That Changes Itself: The Neuroplasticity Revolution & Film Clips of People Undergoing Plastic Change

Norman Doidge, MD
Columbia University
<normand@uww.edu>

The discovery that the human brain can change its own structure and function with thought and experience, turning on its own genes to change its circuitry, reorganize itself, and change its operation is the most important change in our understanding of the brain in 400 years. We explore how, given that the human brain has been plastic, we have missed this core feature, and how this misunderstanding led scientists to doubt the claims made by the pioneers of neurofeedback. Many new cures for neurological and psychiatric conditions are described. Using film clips of patients from his book *The Brain That Changes Itself*, Dr. Doidge demonstrates some of the key principles of neuroplasticity.

Learning Objectives and Outline

The participants will define neuroplasticity, review the current understanding of it, and the history of the concept.

The participants will learn the ways in which the human brain is not “hardwired” and the clinical implications of this.

The participants will learn why, if the brain has always been plastic, it wasn’t detected and early manifestations of it were dismissed.

Core innovations, using sensory substitution as an example, will be described, using film clips.

Neuroplastic principles, and a new approach to treatment of neurological and psychiatric problems that reorganizes the brain, will be described.

The concept of the plastic paradox will be introduced and discussed to demonstrate how neuroplasticity gives rise to both flexible and rigid behaviors and outcomes. Participants will be able to identify and describe the plastic paradox.

REFERENCE

Doidge, N. (2007). *The brain that changes itself*. New York: Viking/Penguin.

QEEG-Guided Neurofeedback for Anger Control

Jonathan Walker, MD
 Neurotherapy Center of Dallas
 <admin@neurotherapydallas.com>

Recent changes in QEEG databases have revealed most anger control problems are associated with excess high-frequency beta activity in one or several brain areas. After downtraining such activity, the anger is usually decreased and anger control is improved. QEEG findings and the posttreatment findings in 67 patients shows that this approach was significant in reducing anger in all but 8% of patients. None were worse.

REFERENCES

Walker, J. E. (2007). A neurologist's clinical experience with QEEG neurofeedback in rehabilitation following brain injury. In J. R. Evans (Eds.), *Contemporary topics in neurofeedback. Handbook of neurofeedback*, (pp. 353–361). Binghamton, NY: Haworth.

Walker, J. E., & Kozlowski, G. P. (2005). Neurofeedback treatment of epilepsy. *Child and Adolescent Psychiatric Clinics of North America*, 14, 163–176.

Walker, J. E., Kozlowski, G., & Lawson, R. (2007). A modular activation/coherence approach to evaluating clinical/QEEG correlations and for guiding neurofeedback training: Modular insufficiencies, modular excesses, disconnections, and hyperconnections. *Journal of Neurotherapy*, 11, 25–44.

Walker, J. E., Lawson, R., & Kozlowski, G. (2007). Current status of QEEG and neurofeedback in the treatment of clinical depression. In J. R. Evans (Ed.), *The Handbook of neurofeedback. Dynamics and clinical applications* (pp. 341–352). Binghamton, NY: Haworth.

Walker, J. E., & Norman, C. (2006). The neurophysiology of dyslexia: A selective review with implications for neurofeedback remediation and results of treatment in 12 consecutive patients. *Journal of Neurotherapy*, 10, 45–55.

Osteopathic Treatment of the Encephalon: A QEEG Study

David Bergstein, DO
 Stillpoint Therapies, Inc.
 <osteodave@earthlink.net>

Debora Elliott, MA
 Interactive Brain Analysis

The purpose of this study was to explore the objective changes in brainwave activity resulting from direct osteopathic treatment of the encephalon. Quantitative electroencephalography (QEEG) was employed to generate electrocortical “brainmaps” for analysis of these dependent variables: Absolute Power, Coherence, Phase Lag.

A mixed gender group of 20 healthy participants between 20 and 50 years of age were studied in a crossover experimental design. Each participant underwent a prestudy treatment to clear nonphysiologic osteopathic lesions 30 days prior to the control phase of the experiment. First, individuals participated in a 60-min supine rest period with a QEEG measurement performed prior to and following the intervening rest period (each with an eyes-open and eyes-closed component). This was the control condition. The experimental condition was identical in all aspects, except that the intervening rest period was replaced with a single session of osteopathic endocranial treatment, exactly 7 days following the control session, again with QEEG measurement performed before and after the intervening treatment. A six technique protocol was designed and employed for the experimentation session, with techniques chosen to emphasize the mobilization of the encephalon globally and to reestablish the thalamus as its primary motion fulcrum. We hypothesize that such treatment of the endocranium should lead to systematic and quantifiable changes in the brain's electrocortical activity. The results of all four QEEG measurements were recorded for each participant; the signals were processed and analyzed statistically.

Significant changes in group electrocortical activity resulted from the endocranial treatment protocol. A large increase in

Absolute Power in the Alpha frequency band ($p = .028$), increased Coherence in the Beta frequency band ($p = .02$), decreased Coherence in the Theta frequency band ($p = .016$), and decreased Phase Lag in the Beta frequency band ($p = .012$) were found in the eyes-closed condition for the Treatment group. A small decrease in Absolute Power across the Delta ($p = .017$) and Theta ($p = .034$) frequency bands, substantially increased Coherence in the Beta frequency band ($p = .018$), and increased Phase Lag across the Theta ($p = .025$) and Alpha ($p = .024$) frequency bands were found in the eyes-open condition for the Treatment group. In addition to the significant changes in magnitude for these dependent variables, a consistent pattern of nonrandom, centralized, and orderly activity was found in most of the Pre/Post results for the treatment group. In contrast, although several dependent variables did change as a result of the Rest period, all control measures lacked either statistical significance (Absolute Power) or central organization (Coherence and Phase Lag).

In all, these findings provide strong evidence that electrocortical activity was directly affected by the endocranial treatment protocol. The results of this osteopathic study provide justification for further osteopathic research in the endocranial field.

EnListen and Learn: Auditory Processing Training Impacts Neural Substrates of Reading Readiness in Dyslexic Readers

Roger Riss, PhD, and Paula Ray, PsyD
Madonna Rehabilitation Hospital
<riss@madonna.org>

Objective

Convergent neuropsychological, neuroimaging, and electrophysiological evidence has implicated a central role for phonological-processing dysfunction in dyslexia. In the present pilot study, we examine the impact of an auditory processing intervention on the

neural substrates of reading readiness in young dyslexic readers.

Participants and Methods

As a component of a larger pilot study, we analyzed surface qEEG and low resolution brain electromagnetic tomography (LORETA) activation patterns during reading in 2 children (ages 6 and 8 years old) with documented reading disability. The children received 60 hr of training, consisting of exposure to gated and filtered sound, tailored to each child's specific auditory processing pattern, embedded in a classical musical recording (EnListen method). We identified pre-post training changes in qEEG and voxel-level neuro-electric source localization (LORETA) patterns during reading, and measured the impact of training on psychometric indices of reading readiness.

Results

We observed increased activation during reading in left temporo-parietal cortex and left inferior frontal regions, similar to patterns observed in normal readers. We noted a shift from right frontal to bilateral frontal activation consistent with patterns reported for well-compensated dyslexic readers. Increased regional activation in the anterior cingulate gyrus was interpreted to reflect enhanced activation of an attention circuit during task. Although overall reading gains were modest, gains of up to 1 *SD* on measures of phoneme discrimination, working memory and reading fluency, suggested enhanced reading readiness.

Conclusions

Preliminary findings suggest that auditory-processing training may have potential to positively impact neural correlates of reading readiness in dyslexic readers, providing an enhanced foundation for subsequent remedial educational interventions.

REFERENCES

- Aylward, E. H., Richards, J., Beringer, V. W., Nagy, W. E., Field, B. A., et al. (2003). Instructional treatment associated with changes in brain activation in children with dyslexia. *Neurology*, *61*, 212–219.
- Breteler, M. H., Arns, M., Peters, S., Giepmans, I., & Verhoeven, L. (2010). Erratum to “Improvements in Spelling after QEEG-based Neurofeedback in Dyslexia: A Randomized Controlled Treatment Study. *Applied Psychophysiology and Biofeedback*, *35*, 182.
- Breteler, M. H., Arns, M., Peters, S., Giepmans, I., & Verhoeven, L. (2010). Improvements in spelling after QEEG-based neurofeedback in dyslexia: A randomized controlled treatment study. *Applied Psychophysiology and Biofeedback*, *35*, 5–11.
- Eden, G. F., & Moats, L. (2002). The role of neuroscience in the remediation of students with dyslexia. *Nature Neuroscience*, *5*(Suppl.), 1080–1084.
- Gaab, N., Gabrieli, J. D., et al. (2007). Neural correlates of rapid auditory processing are disrupted in children with developmental dyslexia and ameliorated with training: An fMRI study. *Restorative Neurology and Neuroscience*, *25*, 295–310.
- Gabrieli, J. D. (2009). Dyslexia: A new synergy between education and cognitive neuroscience. *Science*, *325*, 280–283.
- Horwitz, B., Rumsey, J. M., et al. (1998). Functional connectivity of the angular gyrus in normal reading and dyslexia. *Proceedings of the National Academy of Sciences USA*, *95*, 8939–8344.
- Kohler, S., Paus, T., et al. (2004). Effects of left inferior prefrontal stimulation on episodic memory formation: A two-stage fMRI-rTMS study. *Journal of Cognitive Neuroscience*, *16*, 178–188.
- Liddle, E., Jackson, G., et al. (2005). An evaluation of a visual biofeedback intervention in dyslexic adults. *Dyslexia*, *11*, 61–77.
- Rumsey, J. M., Horwitz, B., et al. (1999). A functional lesion in developmental dyslexia: left angular gyral blood flow predicts severity. *Brain and Language*, *70*, 187–204.
- Shaywitz, S. E., & Shaywitz, B. A. (2008). Paying attention to reading: The neurobiology of reading and dyslexia. *Developmental Psychopathology*, *20*, 1329–1349.
- Shaywitz, S. E., Shaywitz, B. A., et al. (2003). Neural systems for compensation and persistence: Young adult outcome of childhood reading disability. *Biological Psychiatry*, *54*, 25–33.
- Tansey, M. A., & Bruner, R. L. (1983). EMG and EEG biofeedback training in the treatment of a 10-year-old hyperactive boy with a developmental reading disorder. *Biofeedback & Self-Regulation*, *8*, 25–37.
- Temple, E., Deutsch, G. K., et al. (2003). Neural deficits in children with dyslexia ameliorated by behavioral remediation: Evidence from functional MRI. *Proceedings of the National Academy of Sciences USA*, *100*, 2860–2865.
- Thornton, K. E., & Carmody, D. P. (2005). Electroencephalogram biofeedback for reading disability and traumatic brain injury. *Child and Adolescent Psychiatric Clinics of North America*, *14*, 137–162, vii.

Marketing the Neurofeedback Practice

Jeffrey Hunter, DBA
Assumption College
<hunterjg@cox.net>

The presentation involves a brief introduction to a strategic marketing assessment and planning tool that can be used to develop a comprehensive marketing plan for the neurofeedback practice (please see below). The concept of USP, development of a Unique Selling Point for the neurofeedback practice, is discussed.

USP, or unique selling point (and sometimes referred to as unique selling proposition or unique selling position), is a fundamental concept in marketing. It refers to some element of the marketer’s offering which makes it more attractive to potential clients or customers than the offering of competitors who are vying for the same clients or customers. To be effective, a USP should provide some type of differentiation that is of value to customers. An example from the automotive world would be an automobile which is unusually efficient and environment friendly, much more so than other competing automobiles. Furthermore, a good USP should be one that is not easily imitated. It should be long-lasting and should also be able to be communicated effectively to potential clients or customers. The “U” of USP equals that which makes the product or service different. The “S” is that which makes it attractive. The “P” represents its ability to make an impression on the mind of a consumer.

In the domain of neurofeedback practice, a potential USP might be “specialization.” A practice is the only one in the state of Rhode Island that provides comprehensive

therapeutic approaches to ADD/ADHD, including qEEG diagnosis, Z-score neurofeedback training, whole-family counseling, behavior therapy for the patient, and nutritional analysis with dietary recommendations for the child. (This example is intended simply to illustrate the point, not serve as a recommendation.)

The neurofeedback practice that develops, maintains, and promotes an effective USP when compared to its competitors will gain and retain market share.

The oral presentation also makes brief reference to the following material, which forms the basis of the proposed workshop presentation on marketing the neurofeedback practice.

REFERENCES

- Blakeman, R. (2007). *Integrated marketing communication*. Lanham, MD: Rowman & Littlefield.
- Fortenberry, J. L. (2009). *Health care marketing: Tools and techniques*. Sudbury, MA: Jones & Bartlett.
- Levinson, J. C. (2007). *Guerilla marketing*, 4th ed. New York, NY: Mariner.

Neurofeedback: A Critical Treatment Component to Behavioral Modification and Parent Education for Individuals Diagnosed with Fetal Alcohol Syndrome (FAS)

James Kowal, PhD
Center for Traumatic Stress
<jkowal@traumaticstress.org>

Ajeet Charate
Trinity Services, Inc.

Fetal alcohol syndrome (FAS) is a condition that is very prevalent and extremely difficult to treat. It is on the spectrum of the Fetal Alcohol Spectrum Disorders (FASD). Prevalence of FASD is estimated to be at least 10 per 1,000, or 1% of all births. FASD is an umbrella term used to describe the range of effects that occur in an individual who is exposed to alcohol prenatally. These effects may include physical, mental, behavioral, and/or learning disabilities with lifelong

implications. This pilot study describes the development and implementation of treatment protocols that includes pre- and postintervention QEEG, neurofeedback training, consultation for behavioral modification, and parental education on FAS. It also discusses the variations and complexities of using various neurofeedback treatment protocols and the overall scope of treatment for children diagnosed on the spectrum of FASD.

REFERENCE

This is a pilot study, and on literature review no other research studies were found in which neurofeedback has been used as an intervention for individuals diagnosed with FAS.

Gamma Induction/Beta Attunement (GI/BA) Intervention Protocol & Neurodegenerative

Jaclyn Gisburne, PhD, and Jana Harr, BA
Rocky Mountain NeuroAdvantage
<jaclyn@svquietly.com>

The GI/BA protocol is an intervention protocol developed over the past 4 years. Formerly known as “beta-reset,” the GI/BA protocol has shown promise with cognitive, affective, and physical pathologies. Its full potential, limitations, and the understanding of its neurological potentials are still being explored. However, the purpose of this presentation is to look at this and other protocols and their utility as interventions that interrupt and/or reverse the systemic activities associated with chronic and neurodegenerative disorders. We discuss how evoking gamma wave potentials, which naturally emanate from the occipital and parietal regions, can facilitate restoration of more normal frequency distribution throughout the brain. We surmise that the gamma-wave potentials are instrumental in the “resetting” of the frequency distributions at these areas and that they have global implications in the remission of symptoms. We discuss several case studies that reflect the resetting activities as evidenced by the often-instantaneous recovery of the clients. We also discuss

briefly the role of stress/trauma in the development of pathologies and several adjunct modalities that help the clients resolve these entrenched and often encapsulated experiences.

REFERENCES

- Bauer, M., Oostenveld, R., Peeters, M., & Fries, P. (2006). Tactile spacial attention enhances gamma-band activity in somatosensory cortex and reduces low-frequency activity in parieto-occipital areas. *Journal of Neuroscience*, 26, 490–501.
- Canolty, R. T., Edwards, E., Dalal, S. S., Soltani, M., Nagarajan, S. S., Kirsch, H. E., et al. (2006, September 15). High gamma power is phase-locked to theta oscillation in human neocortex. *Science*, 1626–1628.
- Fields, R. D. (2009, November). New culprits in chronic pain. *Scientific American*, 50–57.
- Levy, R., Hutchinson, W. D., Lozano, A. M., & Dostrovsky, J. O. (2000). High-frequency synchronization of neuronal activity in the subthalamic nucleus of Parkinsonian patients with limb tremor. *Journal of Neuroscience*, 20, 7766–7775.
- Porreca, F., & Price, T. (2009, November). When pain lingers. *Scientific American*, 34–41.
- Schiller, D., Levy, I., Niv, Y., LeDoux, J. E., & Phelps, E. A. (2008). From fear to safety and back: Reversal of fear in the human brain. *Journal of Neuroscience*, 28, 11517–11525.

Classification of ADHD Patients on the Basis of Independent ERP Components Using a Machine Learning System—Crossvalidation With New Data

Andreas Mueller, PhD

Brain and Trauma Foundation
<andreas.mueller@psychologie.ch>

Background

In the context of sensory and cognitive-processing deficits in ADHD patients, there is considerable evidence of altered event related potentials (ERP). Most of the studies, however, were done on ADHD children. Using the independent component analysis (ICA) method, ERPs can be decomposed into functionally different components. Using the classification method of support vector machine, this study investigated whether features of independent ERP

components can be used for discrimination of ADHD adults from healthy participants.

Methods

Two groups of age- and sex-matched adults (74 ADHD, 74 controls) performed a visual two stimulus GO/NOGO task. ERP responses were decomposed into independent components by means of ICA. A feature selection algorithm defined a set of independent component features, which was entered into a support vector machine.

Results

The feature set consisted of five latency measures in specific time windows, which were collected from four different independent components. The independent components involved were a novelty component, a sensory related and two executive function related components. Using a 10-fold cross-validation approach, classification accuracy was 92%.

Conclusions

A crossvalidation study by means of support vector machine with new data of Norwegian research group to classify ADHD adults which indicates that classification by means of linear and nonlinear methods is feasible in the context of clinical groups. Further, independent ERP components have been shown to provide features that can be used for characterizing clinical populations. The transformation from research into clinical praxis will be shown.

REFERENCES

- Barkley, R. A., Murphy, K. R., & Fischer, M. (2008). *ADHD in adults. What the science says*. New York: Guilford.
- Barry, R. J., Clarke, A. R., Johnstone, S. J. (2003). A review of electrophysiology in attention deficit/hyperactivity disorder: I. Qualitative and quantitative electroencephalography. *Clinical Neurophysiology*, 114, 171–183.

- Brown, T. E. (2009). *ADHD comorbidities. Handbook for ADHD complications in children and adults*. Washington, DC: American Psychiatric Publishing.
- Fallgatter, A. J., Ehlis, A. C., Rosler, M., Strik, W. K., Blocher, D., & Herrmann, M. J. (2005). Diminished prefrontal brain function in adults with psychopathology in childhood related to attention deficit hyperactivity disorder. *Psychiatry Research*, *138*, 157–169.
- Kropotov, J. D. (2008). Executive system. In J. D. Kropotov (Ed.), *Quantitative EEG, event-related potentials and neurotherapy*, (pp. 253–291). Amsterdam: Academic Press.

Using EEG to Predict Neurotherapy Treatment Outcome in PTSD

Estate Sokhadze, PhD, Eric Toolson, PhD, Beth Perry, PhD, and Michael Hollifield, MD
University of Louisville
<tato.sokhadze@louisville.edu>

Introduction

Posttraumatic stress disorder (PTSD) is a debilitating disease characterized by hyperarousal, avoidance, and numbing, and/or reexperiencing aspects of the original trauma. Lifetime prevalence of PTSD in community samples is around 6.8% and as high as 30% among specific populations such as Vietnam veterans and female victims of rape. Treatment efficacy in PTSD ranges from 50% to 75% but is highly variable among sufferers, which results in high treatment costs, significant drop-out rates, and low treatment efficiency. Published efforts to quantify biological differences between PTSD and non-PTSD subjects have failed to yield consistent findings (Costa et al., 2002). More important for predicting treatment outcomes, there is a paucity of research to identify biomarkers early in treatment that signal an eventual therapeutic response to an intervention. Our long-term goal is to produce an algorithm that allows discrimination of responders from nonresponders in the early stages of a therapeutic intervention. To that end, we believe that the best predictors/monitors of response to therapy will be based on appropriate analysis of data from physiological measures that directly reflect

activity of the brain regions that have been shown to play an important role in PTSD (Lanius et al., 2006). Of the various available methodologies for differentiating between PTSD and non-PTSD subjects, we have chosen to focus on analysis of EEG data, using an array of theoretical and algorithmic approaches drawn from the recent literature.

Materials and Methods

Data were obtained from a sample of 10 individuals with comorbid PTSD and cocaine addiction and 9 control participants from a larger study on attentional bias to pictorial cues (Sokhadze et al., 2008). The 10 participants underwent 12 sessions of neurofeedback as a therapeutic intervention, with the goal of increasing the sensorimotor rhythm with either a decrease or no change in theta waves at C3 (motor strip) referenced to the left mastoid. After preprocessing (60 Hz filtering removal of artifacts), data were characterized by FFT, and the largest Lyapunov exponents (L1), correlation dimensions (D2), and autocorrelation functions (ACF) were computed using proprietary Matlab[®]-based software, based on a windowed variant (Toolson & Perry, 2010, in preparation) of the Rosenstein et al. (1993) algorithm.

Results

Two measures of the complexity of the EEG tracings—L1 and the rate of decay of the ACF—were significantly lower in PTSD-cocaine addicted patients than in controls. After neurofeedback, the EEG complexity as measured by both L1 and ACF rate of decay were significantly increased, and this correlated with clinical improvement. However, in contrast with results reported by Chae and colleagues (2004), among others, we did not find a statistically significant difference in the correlation dimension (D2) of EEG recordings comparing PTSD-cocaine addicted patients with controls, nor did neurofeedback result in any change in the value of D2.

Conclusions

L1 and ACF rate of decay complexity in EEG tracings may be markers of improvement with neurofeedback in PTSD cocaine-addicted participants. Further research is needed to determine if these biomarkers will distinguish between responders and nonresponders early in treatment, if this biomarker effect is similar in “pure PTSD” participants versus comorbid participants, and if this biomarker effect is similar or different across types of therapeutic interventions.

REFERENCES

- Chae, J.-H., Jeong, J., Peterson, B. S., Kim, D.-J., Bahk, W.-M., Jun, T.-Y., et al. (2004). Dimensional complexity of the EEG in patients with posttraumatic stress disorder. *Psychiatry Res-Neuroimaging*, *131*, 79–89.
- Costa, M., Goldberger, A. L., & Peng, C. K. (2002). Multiscale entropy analysis of complex physiologic time series. *Physical Review Letters*, *89*, 68–102.
- Lanius, R. A., Bluhm, R., Lanius, U., & Pain, C. (2006). A review of neuroimaging studies in PTSD: Heterogeneity of response to symptom provocation. *Journal of Psychiatric Research*, *40*, 709–729.
- Rosenstein, M. T., Collins, J. J., & De Luca, C. J. (1993). A practical method for calculating Lyapunov exponents from small data sets. *Physica D*, *65*, 117–134.
- Sokhadze, E., Singh, S., Stewart, C., Hollifield, M., El-Baz, A., & Tasman, A. (2008). Attentional bias to drug-and stress-related pictorial cues in cocaine addiction comorbid with PTSD. *Journal of Neurotherapy*, *12*, 205–225.

Nutrient Modifiers of Neuroplasticity and Performance, and the Exploration of Novel QEEG Assessment Metrics

Michael Schmidt, PhD
NASA Ames Research Center
<wisemedicine@aol.com>

The human brain is nearly 60% lipid and it is structurally dependent upon specific molecular lipid forms.

At almost all stages of development and aging, provision of specific lipid molecules in specific ratios shapes neural architecture and function. These molecular demands are amplified in an array of clinical conditions as well as being vital in efforts to develop elite performers. Although lipids shape neural architecture and are at the foundation of neuroplasticity, a range of essential nutrient inputs drive the metabolic networks that define the efficiency of neuron communication. This session explores the role of specific lipids in shaping neural architecture, function, and neuroplasticity, along with selected essential nutrient inputs that are fundamental to nervous system function and performance. It also briefly introduces methodologies with the potential for characterizing the impact of subtle variance in metabolic networks by using conventional QEEG assessment metrics, such as coherence, as well as some novel techniques characterizing directional information flow.

REFERENCES

- Amara, C. E., et al. (2007). Mild mitochondrial uncoupling impacts cellular aging in human muscles in vivo. *PNAS*, *104*, 1057–1062.
- Bouwens, M., et al. (2009). Fish-oil supplementation induces anti-inflammatory gene expression profiles in human blood mononuclear cells. *American Journal of Clinical Nutrition*, *90*, 415–424.
- Çakır, T., et al. (2007). Reconstruction and flux analysis of coupling between metabolic pathways of astrocytes and neurons: Application to cerebral hypoxia. *Theoretical Biology and Medical Modeling*, *4*, 48.
- Lukiw, W. J., et al. Docosahexaenoic acid and the aging brain. *Journal of Nutrition*, *138*, 2510–2514.
- Secher, N. H., et al. (2008). Cerebral blood flow and metabolism during exercise: Implications for fatigue. *Journal of Applied Physiology*, *104*, 306–314.

Beyond Neurotherapy: The Ethics of National Security Neuroscience

Jonathan Marks, MA, BCL
Harvard University
<Jonathan_Marks@hks.harvard.edu>

Neuroscience and associated neuro-technologies have transformed diagnosis and

treatment for thousands of patients—from those with severe depression that is not responsive to drugs to those with severely impaired consciousness. Some ethicists and science studies scholars have expressed concerns about the manner in which therapies have changed as a result of recent neuroscience developments. This lecture focuses on ethical concerns raised by various nontherapeutic applications involving both drugs and medical devices—among them, fMRI, EEG, transcutaneous magnetic stimulation, and oxytocin—in the national security context. How are such drugs and devices transforming the national security mission? How and why have these drugs and devices been developed? And how have they been received and implemented in the national security world? What are the ethical implications of these translations? Are the implications different depending upon whether the technologies live up to the claims made by their proponents? This lecture addresses these questions and explores the complex interactions between the national security and neuroscience communities—as well as the implications of recent research on public understanding of brain images and neuroscientific explanations.

REFERENCES

- Marks, J. H. (2007). Interrogational neuroimaging in counterterrorism: A “no-brainer” or a human rights hazard? *American Journal of Law & Medicine*, 33, 483–500.
- Marks, J. H. (2010a). Neuroconcerns: Some responses to my critics. *American Journal of Bioethics: Neuroscience*, 1(2), W1–W3.
- Marks, J. H. (2010b). A neuroskeptic’s guide to neuroethics and national security. *American Journal of Bioethics: Neuroscience*, 1(2), 4–12.

The Immediate Effect of NeuroField as Measured by Pre–Post QEEG

Nicholas Dogris, PhD
 NeuroField, Inc.
 <nicholasdogris@verizon.net>

Twenty research subjects participated in an experiment to measure the immediate impact

of NeuroField electromagnetic stimulation on the brain. Seven-min QEEG recordings were obtained in an eyes open and eyes closed condition. The NeuroField cap was placed over the QEEG cap and a brief electromagnetic stimulation was administered. Immediately following the NeuroField stimulation the QEEG eyes open and eyes closed condition was repeated. The data were analyzed via the NeuroGuide statistics program using a multivariate analysis of variance and descriptive methods. Significant differences were observed from pre- to posttest conditions for each test subject. NeuroField appears to lift cortical suppression as evidenced by significant absolute power, asymmetry, coherence, and phase changes in the brain. The clinical relevance of NeuroField appears to be in its ability to lift suppression so that other methods of EEG neurofeedback can be used more effectively.

REFERENCES

- Berman, R. M., Narasimhan, M., Sanacora, G., et al. (2000). A randomized clinical trial of repetitive transcranial magnetic stimulation in the treatment of major depression. *Biological Psychiatry*, 47, 332–337.
- Grunhaus, L., Dannon, P. N., Schreiber, S., et al. (2000). Repetitive transcranial magnetic stimulation is as effective as electroconvulsive therapy in the treatment of nondelusional major depressive disorder: An open study. *Biological Psychiatry*, 47, 314–324.
- McTaggart, L. (2008). *The field*. New York: Harper.
- Martis, B., & Janicak, P. G. (2000, July). Transcranial magnetic stimulation for major depression: Therapeutic possibilities. *International Drug Therapy Newsletter*, 1–10.
- Oschman, J. L. (2000). *Energy medicine*. New York: Churchill Livingstone.
- Post, R. M., Kimbrell, T. A., McCann, U. D., et al. (1999). Repetitive transcranial magnetic stimulation as a neuropsychiatric tool: Present status and future potential. *The Journal of ECT*, 15, 39–59.
- Pridmore, S., Bruno, R., Turnier-Shea, Y., et al. (2000). Comparison of unlimited numbers of rapid transcranial magnetic stimulation (rTMS) and ECT treatment sessions in major depressive episode. *International Journal of Neuropsychopharmacology*, 3, 129–134.
- Pridmore, S., & Belmaker, R. (1999). Transcranial magnetic stimulation in the treatment of psychiatric disorders. *Psychiatry and Clinical Neuroscience*, 53, 541–548.

An International Study of the BAUD Applications for Emotional and Chronic Pain Issues

G. Frank Lawlis, PhD

Lawlis Peavey PNP Center
<LawlisF@aol.com>

The purpose of this study was to determine whether the Bioacoustical Utilization Device (BAUD) could be effective in alleviating symptoms related to emotional issues, impulsive behavioral issues, and/or chronic pain. Eighty-six patients treated by 19 therapists in the United States, Switzerland, Portugal, and Denmark underwent treatment with the BAUD for one or more sessions. A Likert rating of symptomatology was recorded before and after treatment for all patients and 3 weeks after treatment in a subset of patients. Analysis of immediate posttreatment data using McNemar's test demonstrated clinically and statistically significant improvement in all three symptom groupings. Data recorded 3 weeks posttreatment demonstrated stability of posttreatment results in most patients in the emotional issues and chronic pain categories (insufficient 3-week posttreatment data were available in the impulsive behavioral issues category for analysis). These data suggest that the BAUD is effective in alleviating symptoms from a variety of psychological sources.

REFERENCES

- Bittman, B., Berk, L., Shannon, M., Sharaf, M., Westergard, J., Guegler, K. J., et al. (2005). Recreational music-making modulates the human stress response. *Medical Science Monitor*, *11*, BR31–40.
- Doidge, N. (2007). *The brain that changes itself*. New York: Penguin.
- Labbe, E., Schmidt, Babun, J., & Pharr, M. (2007). Coping with stress: The effectiveness of different types of music. *Applied Psychophysiology and Biofeedback*, *32*, 163–168.
- Lawlis, F. (2004). *The ADD answer*. New York: Viking.
- Lawlis, F. (2009). *Retraining the brain*. New York: Plume.
- Lutz, A., Greischar, L., Rawlings, N. B., Ricard, M., & Davidson, R. J. (2004). Long-term mediators

self-induce high-amplitude gamma synchrony during mental practice. *Proceedings of the National Academy of Sciences USA*, *101*, 16369–16373.

Pilot Investigation: QEEG Phenotypes and EEG Vigilance of Adult ADHD

Sarah Wyckoff, MA

University of Tübingen
wyckoffsarah@yahoo.com

Martijn Arns, MSc

Brainclinics Diagnostics

Christian Sander and Ute Strehl

University of Tübingen

Objective

EEG/QEEG analysis of adults with ADHD compared to healthy controls and/or normative database populations have produced a variety of patterns of activity. Adults with ADHD show the typical increase in Theta/Beta ratios, with varying Theta elevations and Beta reductions (Bresnahan, Anderson, & Barry, 1999; Bresnahan & Barry, 2002; Clarke et al., 2008a). Some exhibit increased absolute Theta and Alpha power without differences in Beta activity (Koehler et al., 2009). Others exhibit increased low-Alpha (8–10 Hz) and/or high-Alpha (10–12 Hz) activity, depending on task condition (Loo et al., 2009; White, 2001, 2003), whereas coherence data indicates a reduction in hemispheric differences of Delta and Alpha bands (Clarke et al., 2008b) and increased right-hemisphere alpha dominance is correlated with a greater number of ADHD symptoms (Hale et al., 2009). Distinct EEG and behavior patterns or subtypes in an adult ADHD population have also been observed. Thompson and Thompson (2005) reported elevated Theta/Beta ratios, low Alpha, elevated Hi Beta/SMR, and combined elevated Theta/Beta and Hi Beta/SMR ratios correspond to specific symptom presentation in a clinical adult population. In an attempt to develop theory-driven models for QEEG interpretation, neurofeedback protocol selection, and medication response prediction the EEG Phenotype (Johnstone, Gunkelman, & Lunt, 2005) and EEG Vigilance (Bente,

1964; Hegerl, 2008) models have emerged to explain state and trait differences in clinical populations. These models have been utilized in the evaluation of childhood ADHD and control populations and stimulant medication response in childhood ADHD (Arns, Gunkelman, Breteler, & Spronk, 2008; Sander, Arns, Olbrich, & Hegerl, in press). Limited research is available on the application of these models in an adult ADHD population for assessment or determination of neurofeedback treatment protocols.

Methods

Continuous 19-channel EEG was recorded during eye closed and eyes open resting state, CNV task, and auditory oddball task in two groups of adults (18+ years old), with and without ADHD. Adult ADHD participants met *DSM-IV* criteria for combined, hyperactive, or attention type ADHD. Participants in both groups reported no additional serious physical, neurological, or psychiatric disorders; had a full scale IQ greater than 80; and were right hand dominant. For each participant, the EOG corrected raw EEG for the EO and EC condition was obtained and a comparison of the individual data to a matched normative database controls was conducted. Participant data were then rated to possess one or more of the following EEG phenotypes outlined by Johnstone et al. (2005): (a) Frontal Slow, (b) Slowed Alpha Peak Frequency, (c) Frontal Beta Spindles, or (d) Paroxysmal EEG. EEG Vigilance clusters and state changes will also be assessed in accordance with the Vigilance Classification Algorithm presented by Hegerl and colleagues (2008).

Results

This investigation is part of a long-term treatment study currently in progress. The most current results related to the distribution of EEG Phenotypes and EEG Vigilance states in the ADHD and control populations will be presented at the time of the presentation.

Conclusion

Specific findings are discussed and implications for protocol selection in the current treatment study and future research projects are explored.

REFERENCES

- Arns, M., Gunkelman, J., Breteler, M., & Spronk, D. (2008). EEG phenotypes predict treatment outcome to stimulants in children with ADHD. *Journal of Integrative Neuroscience*, 7(3), 421–438.
- Bente, D. (1964). Vigilanz, dissoziative Vigilanzverschiebung und Insuffizienz des Vigilanztonus [Vigilance, dissociative vigilance shifting and insufficiency of vigilance stages]. In H. Kranz & K. Heinrich (Eds.), *Begleitwirkung und Mißerfolge der psychiatrischen Pharmakotherapie [Accompanying effects and failures of psychiatric pharmacotherapy]*. Stuttgart, Germany: Thieme.
- Bresnahan, S. M., Anderson, J. W., & Barry, R. J. (1999). Age-related changes in quantitative EEG in attention-deficit/hyperactivity disorder. *Biological Psychiatry*, 46, 1690–1697.
- Bresnahan, S. M., & Barry, R. J. (2002). Specificity of Quantitative EEG analysis in adults with attention deficit hyperactivity disorder. *Psychiatry Research*, 112, 133–144.
- Clarke, A. R., Barry, R. J., Heaven, P. C., McCarthy, R., Selikowitz, M., & Bryne, M. K. (2008a). EEG coherence in adults with attention-deficit/hyperactivity disorder. *International Journal of Psychophysiology*, 76, 35–40.
- Clarke, A. R., Barry, R. J., Heaven, P. C., McCarthy, R., Selikowitz, M., & Bryne, M. K. (2008b). EEG in adults with attention-deficit/hyperactivity disorder. *International Journal of Psychophysiology*, 70, 176–183.
- Hale, T. S., Smalley, S. L., Hanada, G., Macion, J., McCracken, J. T., McGough, J. J., et al. (2009). Atypical alpha asymmetry in adults with ADHD. *Neuropsychologia*, 47, 2082–2088.
- Hegerl, U., Stein, M., Mulert, C., Mergl, R., Olbrich, S., Dichgans, E., et al. (2008). EEG-vigilance differences between patients with borderline personality disorder, patients with obsessive-compulsive disorder and healthy controls. *European Archives of Psychiatry and Clinical Neuroscience*, 258, 137–143.
- Johnstone, J., Gunkelman, J., & Lunt, J. (2005). Clinical database development: Characterization of EEG phenotypes. *Clinical EEG and Neuroscience*, 36, 99–107.

- Koehler, S., Lauer, P., Schreppel, T., Jacob, C., Heine, M., Boreatti-Hummer, A., et al. (2009). Increased EEG power density in alpha and theta bands in adult ADHD patients. *Journal of Neural Transmission*, 116, 97–104.
- Loo, S. K., Hale, T. S., Macion, J., Hanada, G., McGough, J. J., McCracken, J. T., et al. (2009). Cortical activity patterns in ADHD during arousal, activation, and sustained attention. *Neuropsychologia*, 47, 2114–2119.
- Sander, C., Arns, M., Olbrich, S., & Hegerl, U. (in press). EEG-vigilance and response to stimulants in pediatric patients with attention deficit/hyperactivity disorder. *Journal of Clinical Neurophysiology*.
- Thompson, L., & Thompson, M. (2005). Neurofeedback intervention for adults with ADHD. *Journal of Adult Development*, 12, 123–130.
- White, J. N., Jr. (2001). *Neuropsychological and electrophysiological assessment of adults with attention deficit hyperactivity disorder*. Unpublished doctoral dissertation, The University of Tennessee, Knoxville.
- White, J. N., Jr. (2003). Comparison of QEEG reference databases in basic signal analysis and in the evaluation of adult ADHD. *Journal of Neurotherapy*, 7.

Effects of Gamma Neurofeedback Training on Perceived Positive Emotional State and Cognitive Functions

Estate Sokhadze, PhD
University of Louisville
<tato.sokhadze@louisville.edu>

Jonathan Cowan, PhD, Timothy Horrell, BS, Allan Tasman, MD, Guela Sokhadze, and Christopher Stewart, MD

Introduction

It has been shown that emotional abnormalities are typical for addicts. Alexithymia (i.e., state of deficiency in understanding, processing, or describing emotions; Fukunishi, 1996) and dysphoria (i.e., state of inability to experience positive emotions, mood lability; Cowan et al., 1980), and decreased emotional reactivity to natural positive reinforcers (Gerra et al., 2003) are highly prevalent among substance abusers and in those at risk for development of substance use disorders. Therefore, in addition to well-known attentional and cognitive

impairment, there are disruptions in processing emotion in individuals with substance dependence and in those predisposed to drug abuse. According to the “allostasis” theory (Koob et al., 2004; Koob & Le Moal, 2001) sensitization to drugs and counteradaptation are hypothesized to contribute to dysregulation of hedonic homeostasis and to observed brain reward system abnormalities in already-addicted individuals. However, in some cases hedonic dysfunctions and lower sensitivity to positive affect might be an inherited trait predisposing afflicted individuals to drug-seeking and drug-taking behaviors that may result in substance abuse and ultimately in drug dependence.

Neurofeedback training-based neurotherapy is one of the potentially efficacious nonpharmacological treatment options for substance use disorders (Sokhadze et al., 2007, 2008, 2009). There have been an increasing number of neurofeedback protocols that report success in treating a variety of addictive behaviors. There are practically no studies on the use of neurofeedback in adolescents and young adults with occasional drug use when individuals have drug use history but did not yet developed substance dependence (Trudeau, 2005). One of the most promising direction of neurofeedback research is development of protocols that might be used to prevent drug abuse through self-regulation training aimed to enhance of EEG measures of positive emotional states. In a previous study it was show an association of prefrontal gamma oscillations with positive emotional states (Cowan & Rubik, 2009). One of the specific aims of this pilot study was to determine the dynamics of self-reported perceived positive emotional state rating before, during and after twelve 25-min-long neurofeedback training course in 2 groups of participants. One group had documented drug use history ($N=6$; most of them referred from Louisville Adolescent Network for Substance Abuse Treatment—a community mental health system of care for adolescents with substance use/abuse issues), and the other was a group of drug-naïve participants ($N=6$; recruited mostly from students and residents). Our hypothesis was that the prefrontal high-frequency power

increase over 12 neurofeedback training sessions is possible and will be accompanied by increased rating scores of positive emotional states. Our prediction was that successful completers of the neurofeedback training in the groups of adolescents and young adults both with and without drug/alcohol abuse history will improve subsequent performance on cognitive tests and will increase positive affect.

Method

As a preferred neurofeedback protocol, we used enhancement of gamma range (centered around 40 Hz) activity (so-called Neureka! parameter; Peak Achievement Trainer [PAT]) and inhibition/suppression of other frequencies (i.e., "Focus" parameter) at the prefrontal site (FPz). Training of high-frequency activity in 40 Hz centered gamma band at the midline prefrontal site after 12 sessions resulted, as it was predicted in better performance on neurocognitive (MicroCog) and attention (IVA + Plus) tasks and improved scores on emotional self-reports (happiness and self-satisfaction, Siahpush et al., 2008) and clinical (Beck Depression Inventory-II; Beck et al., 1996) status. This protocol used in the study is based on the PAT application "Brain Happiness and Focus" and is intended to train focus, concentration and emotional state (Cowan, 2008). Individual reports of self-received happiness scores were assessed during each neurofeedback session using the Continuous Response Digital Interface dial (CRDI; Geringer et al., 2004) and recorded on a per-minute basis. The CRDI reading showed significant positive correlation with relative gamma power during individual training sessions and exhibited the tendency to increase with the number of conducted neurofeedback sessions.

Conclusion

Neurofeedback training aimed at enhancement of focus and Neureka! measures was accompanied by positively correlated subjective self-reports of positive emotional feelings during self-regulation sessions and resulted in

improved performance on IVA + Plus and MicroCog tests at the post-12-session neurofeedback course. Posttraining evaluations showed a decrease in depression scores and increased happiness and self-satisfaction rating in both participant groups in this study. We discuss potential utility of the Focus and Neureka! protocol for self-regulation of attention and emotional state in individuals predisposed for substance abuse.

REFERENCES

- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the Beck Depression Inventory-II*. San Antonio, TX: Psychological Corporation.
- Cowan, J. D., Kay, D. C., Neidert, G. L., Ross, F. E., & Belmore, S. M. (1980). Defeated and joyless: Potential measures of change in drug abuser characteristics. *Journal of Nervous and Mental Disease*, 168, 391-399.
- Cowan, J. D. (2008). *The Neureka! protocols manual*. Goshen, KY: Peak Achievement Training.
- Fukunishi, I. (1996). Alexithymia in substance abuse: Relationship to depression. *Psychological Reports*, 78, 641-642.
- Geringer, J. M., Madsen, C. K., & Gregory, D. (2004). A fifteen-year history of the Continuous Response Digital Interface: Issues relating to validity and reliability. *Bulletin of the Council for Research in Music Education*, 160, 1-15.
- Gerra, G., Baldaro, B., Zaimovic, A., Moi, G., Bussandri, M., et al. (2003). Neuroendocrine responses to experimentally-induced emotions among abstinent opioid-dependent subjects. *Drug & Alcohol Dependence*, 71, 25-35.
- Koob, G. F., & Le Moal, M. (2001). Drug addiction, dysregulation of reward, and allostasis. *Neuropsychopharmacology*, 24, 97-129.
- Koob, G. F., Ahmed, S. H., Boutrel, B., Chen, S., et al. (2004). Neurobiological mechanisms in the transition from drug use to drug dependence. *Neuroscience and Biobehavioral Reviews*, 27, 739-749.
- Siahpush, M., Spittal, M., & Singh, G. K. (2008). Happiness and life satisfaction prospectively predict self-rated health, physical health, and the presence of limiting, long-term health conditions. *American Journal of Health Promotion*, 23(1), 18-26.
- Sokhadze, E., Stewart, C., & Hollifield, M. (2007). Integrating cognitive neuroscience methods with neurofeedback therapy in treatment of substance use disorder comorbid with PTSD. *Journal of Neurotherapy*, 11(2), 13-44.

- Sokhadze, E., Stewart, C., Sokhadze, G., Hollifield, M., & Tasman, A. (2009, April 3). *EEG biofeedback and reactivity to pictorial and verbal drug-and stress-related cues in cocaine addiction comorbid with PTSD*. Paper presented at the AAPB annual meeting, Albuquerque, NM.
- Sokhadze, T. M., Cannon, R. & Trudeau, D. L. (2008). EEG biofeedback as a treatment for substance use disorders: Review, rating of efficacy and recommendations for future research. *Applied Psychophysiology & Biofeedback*, 33, 1–28.
- Trudeau, D. L. (2005). Applicability of brain wave biofeedback to substance use disorder in adolescents. *Child and Adolescent Clinics of North America*, 14, 125–136.

Using Neurofeedback to Treat OCD Symptoms in a Low Functioning Down's Syndrome Client

Kay Sheehan, EdD, and Rodney Mers, MA
ADD Center of Colorado
<drkms1@gmail.com>

Introduction

The unique aspects of this case are the extent of the client's low functioning and the severity of his OCD symptoms. This 24-year-old young adult man's OCD symptoms significantly improved by Session 12. Neurofeedback training was continued to see how further training could improve his overall functioning.

The goal of training was to reduce the severity of the OCD (compulsive type) symptoms to improve his daily life and to see if medications for OCD (Zoloft and supplements) could be reduced or eliminated. The primary OCD symptoms his parents found hampering his daily life were his focus on one thing (e.g., turning the light switch on and off until he was redirected and constantly sorting mail until redirected), his inability to shift from one task to another, and his inability to do things in a timely manner consistent with his overall functioning (e.g., taking 2 hr to fold a stack of towels). The client's compulsive behavior resulted in his inability to move from task to task in a sheltered workshop and consequent removal from the workshop.

Method

Training sites were chosen based on QEEG information and symptoms. Using Neurocybernetics EEGer software, the initial sites trained were CZ-A1 and F4-A2. CZ was chosen to impact neural activity associated with OCD. F4 was selected to impact executive functions. During the initial training session, 0–7 Hz activity at both sites was two to three times higher in amplitude than SMR. Therefore, it was decided to inhibit 0–7 Hz amplitudes and increase SMR (12–15 Hz) amplitudes. Additional training sites included C4-A2, FZ-A1, T3-F7 and T3-P3. Every training session consisted of training each of two sites for 15 min, respectively. The client was seen for a total of 51 sessions.

Results

Due to the client's low-functioning condition, he was unable to do any pre- and post-testing, such as the TOVA. His parents' finances precluded a posttraining QEEG. Therefore, the positive change assessment was based on reports from the parents of the client's behaviors at home, check lists, and in-session observations of the client's behaviors and interactions by the neurofeedback therapist. In addition, the training data were recorded in a file on the EEGer software enabling within and between session comparisons.

At 12 sessions the parents had seen very positive changes in the client's ability to stop an activity and move on to another activity. He was making eye contact more often and delaying less when asked to do something. Neurofeedback was continued to see if further improvement could be made in these behaviors, to address language issues, and to eliminate medications for OCD.

The parents continued to note improvement in the client's ability to move from task to task as well as to complete tasks more quickly. In addition, the client's Zoloft and all supplements were discontinued, and the improvements made during the training were maintained.

The therapist who did the neurofeedback training noted increasing eye contact as training progressed to the point of consistent

appropriate eye contact toward the end of the neurofeedback training. At the 45th session, for the first time, the client picked a neurofeedback game, which he had not done previously. His language also improved—for example, at the 47th session, the client began using phrases as opposed to making one-word responses as he had been doing. When asked to pay attention to the screen so he could get more beeps, he always had just said, “Sorry.” At this session he said, “Sorry about that” while making eye contact. In the remaining neurofeedback sessions, he progressed from using a one-word response, “Sorry,” when asked a question to using a simple sentence: “I don’t know.”

Conclusion

This case suggests that neurofeedback training can be helpful in treating OCD symptoms in a low-functioning Down’s Syndrome client. The positive changes in his behavior helped the client and his parents have a more positive daily routine and may lead to sheltered workshop employment. The client’s parents were so pleased with his progress they decided to continue neurofeedback training but opted for home training due to finances.

REFERENCES

- Brownback, T., & Mason, L. (2003). *The Brownback, Mason and Associates Neurofeedback System: Manual 3*. Allentown, PA.
- Cheng, Z., Liu, S., & Gong, Y. (1993). Stability of intellectual structure of learning disabled and normal children. *Psychological Science (China)*, *16*, 158–161.
- Fleischman, M. J., & Othmer, S. (2006). Case study: Improvements in IQ score and maintenance of gains following EEG biofeedback with mildly developmentally delayed twins. *Journal of Neurotherapy*, *9*, 35–46.
- Hammond, D. C. (2003). QEEG guided neurofeedback in the treatment of obsessive compulsive disorder. *Journal of Neurotherapy*, *7*, 25–52.
- Thompson, M., & Thompson, L. (2003). *The neurofeedback book: An introduction to basic concepts in applied psychophysiology*. Wheat Ridge, CO: The Association for Applied Psychophysiology and Biofeedback.

Single Cell Memory: How Individual Neurons Route and Store Temporary Information to Maintain Attention

Donald Cooper, PhD

University of Colorado, Boulder

<d.cooper@colorado.edu>

Our brain’s short-term memory system has been likened to the rewritable RAM memory of a computer. To perform normal functions, we need the ability to transiently store, quickly and reliably, large amounts of data, but only a small amount of this needs to be retained in the longer term. Scientists have spent decades working out which parts of the brain are responsible for this memory buffer system and how neural networks manage this feat. Original theories suggested the memories were retained by multiple cells forming “circuits” around which electrical impulses were fired for the necessary period. More recent ideas have centered around the concept that even an individual neurons in the cortex could somehow hold information. To test this we probed individual prefrontal neurons from mice using tiny electrodes to measure their function. We found that a particular component of the cells in question tells the cell to start an internal signaling system that holds the “memory” in place. Gene deletion of a protein that initiates this signaling cascade eliminated the single-cell memory buffer. Details and updates can be obtained from the Cooper Laboratory website at <http://www.Neurocloud.org>

REFERENCES

- (Available at <http://www.Neurocloud.org>)
- Fowler, M. A., Sidiropoulou, K., Phillips, C., Ozkan, E., & Cooper, D. C. (2007). Corticolimbic expression of TRPC4 and TRPC5 channels in the rodent brain. *PLoS One*, *2*(6), e57.
- Sidiropoulou, K., Lu, F. M., Fowler, M., Xiao, R., Ozkan, E., Phillips, C., et al. (2009). Dopamine modulation of prefrontal cortical mGluR5-mediated intrinsic regenerative activity. *Nature Neuroscience*, *12*, 287–300.
- Switching signals in the brain. *PLoS Biology*, *3*(6), e210.

Could Neurofeedback Reconstruct Synchronous Networks Lost Following Traumatic Brain Injury?

Paul Rapp, PhD

Department of Military and Emergency Medicine Uniformed Services, University of the Health Sciences
<prapp@usuhs.mil>

In some instances, individuals receiving mild closed skull head injuries are asymptomatic in the immediate postinjury period but subsequently present significant impairment in cognitive and affective processing. The objective of our research program is to identify individuals at risk of delayed-onset dysfunction and to provide preemptive treatment. The analysis begins with a physiological model of progressive diffuse axonal injury. This model predicts alterations in transient, stimulus-dependent synchronous behavior. Preliminary results are consistent with this prediction. The model suggests that procedures which facilitate the reconstruction of synchronous networks may prevent postinjury deterioration in asymptomatic patients and may provide an effective treatment for patients who present postinjury neuropsychiatric disorders. It is hypothesized that neurofeedback protocols using recently developed time-dependent measures of multivariate central nervous system synchronous behavior may accomplish this.

REFERENCES

- Allefeld, C., Frisch, S., & Schlesewsky, M. (2005). Detection of early cognitive processing by event-related phase synchronization analysis. *NeuroReport*, *16*, 13–16.
- Allefeld, C., & Kurths, J. (2004). An approach to multivariate phase synchronization analysis and its application to event related potentials. *International Journal of Bifurcation and Chaos*, *14*, 417–426.
- Delorme, A., & Makeig, S. (2004). EEGLAB: An open source toolbox for analysis of single trial EEG dynamics including independent component analysis. *Journal of Neuroscience Methods*, *134*, 9–21.
- Hernandez, R. S., & Rapp, P. E. (2007). Network synchronization/desynchronization defects in the pathogenesis of neuropsychiatric disorders. In R.

Wang, F. Gu, & E. Shen (eds.), *Advances in cognitive neurodynamics—Proceedings of the International Conference on Cognitive Neurodynamics*. New York: Springer Verlag.

A Pilot Study: Positive Emotion Monitoring with Hemoencephalography Among a Group of Taiwanese Elders

Huey-Tzy Justina Chen, PhD

Fu Jen Catholic University
<046509@mail.fju.edu.tw>

Paul Kwong, DSc

Hong Kong University

Meng-Twin Wu

Catholic University Sai-Hung Tang, Cardinal Tien Hospital

The objective of the intervention is to improve the positive emotion by eliciting joy from a protocol of “story-telling.” In this 2009 pilot study, 7 participants in Taipei ranging from 60 to 85 years of age were selected in a snowball sample. Each participant chatted with the principle investigator for a couple of minutes, with Toomim’s hemoencephalography (HEG) headband attached, before and after the participant told his or her stories. The single sessions of about 20 min per person were conducted on site. Each participant’s oxygen levels as measured by HEG signals at fp1 and fp2 were measured before intervention as baseline and during the storytelling episode.

The new modality of “neurofeedback” intervention consists of (a) letting the participant learn to observe his or her colorful HEG signals online, (b) encouraging with the activity facilitators to elicit life stories with uplifting episodes, (c) telling the participants to relate their immanent positive feelings and “somatic markers” to HEG signal as displayed dynamically on a portable screen, and (d) intentionally reproducing joyful feelings.

It was found that the signals during the ordinary conversational period, “before” and “after” the storytelling, are lower. There are clear “net gains” of HEG signals in the story-telling period. The results show that when the participants exhibit joyful and positive narrations, emotional expressions or

linguistic affects of the HEG signals are obviously strong and sustained longer. A pilot study resulting from the new method of using HEG to augment an intervention of an activity program for elderly persons is presented.

Storytelling as augmented by HEG neurofeedback might be an effective modality of intervention to improve the positive emotion of elderly people. This dynamically facilitated approach is probably more efficient than a passive approach whereby an elderly participant plays neurofeedback games by himself or herself. Further studies are called for with a sufficient sample size and video recording in order to design an evidence-based intervention protocol.

Neuroscience and Depth Psychology: A First Step in Connecting Neurofeedback with Jung's Theory of Complexes

David Drapes, PhD
Private practice
<ddrapes@yahoo.com>

This will be a sharing, seminarlike offering to examine the effects of brain wave modulation and control beyond symptom reduction toward personality change. Clinical case material will be presented that support the work of C. G. Jung, one of the first clinical professionals to use biofeedback 100 years ago. The question of neurofeedback as a treatment for personality complexes will be addressed from theoretical, neurological, and clinical perspectives.

Empirical research based on a patient's subjective reports appears to indicate a greater and more pervasive change in outlook toward self and life with neurofeedback-assisted depth psychotherapy. This exposes the neurological underpinnings of Jung's ideas about complexes, psychic energy, and archetypal patterns, perhaps rhythmic patterns within the collective universe of the patient's mind/brain or neuro-oscillation gone awry.

Happiness, benevolence, resilience, and an open embrace of life have been outcomes observed with patients in neurofeedback-assisted depth psychotherapy. Neurofeedback

training/treatment can evolve beyond techniques to neurofeedback psychology and psychotherapy for those seeking more and professionals wanting to take the field further.

In addition to case vignettes and pilot study research results, discussion, musings, idealizing, and a critical eye will be employed to assess the potential of synthesizing the best of the past with the brightest of the present toward a creative neuropsychological hybrid for the future.

REFERENCES

- Buzsaki, G. (2006). *Rhythms of the brain*. New York: Oxford University Press.
- Horstman, J. (2009). *The scientific American: Day in the life of your brain*. New York: Wiley.
- Jung, C. G. (1960). *The structure and dynamics of the psyche*. Princeton, NJ: Princeton University Press.
- Lindorff, D. (2004). *Jung and Pauli*. Wheaton, IL: Quest Books.
- Schwartz, J. (2002). *The mind and the brain*. New York: Regan Books.
- Stein, M. (1998). *Jung's map of the soul*. Chicago: Open Court.

Effectiveness of an Advanced Form of Transcranial Electrical Stimulation in Cases of Persistent Anxiety and Depression

Nancy White, PhD
The Enhancement Institute
<nancy@enhancementinstitute.com>

In an average year in the United States some 40 million people suffer from anxiety and another 20 million become clinically depressed. The symptoms of these disorders cause substantial distress for the sufferers and their families and cost society dearly each year in lost time and suboptimal job performance. Moreover, the number of people exhibiting symptoms of depression and anxiety continues to grow and is projected to continue growing until 2020. Research continues to demonstrate that the available pharmaceutical treatments are not much more effective than placebo; in fact, recent articles point out that placebo appears to be as effective as most of

the currently popular psychotropics. Persons who cannot find an effective solution to their depression or anxiety tend to get worse and develop bad habits as they seek to compensate, making treatment even more complex. Although certain neurofeedback protocols have proven effective for depression and anxiety, they still take months to mediate the symptoms of these disorders. This presentation reviews the results of a pilot study involving anxious and/or depressed patients who were treated in a clinical setting using an advanced Transcranial Electrical Stimulation (TES) system approved by the Food and Drug Administration for the treatment of anxiety, depression, and insomnia. In clinical trials this system mediated the symptoms of anxiety, depression, and insomnia in a lasting way in most patients within 2 to 4 weeks by providing stimulation that appears to affect the hypothalamus and associated brain structures to adapt and alter the levels of neurochemicals critical to maintaining normal mood. The purpose of the pilot study was to confirm the effectiveness of advanced TES in the everyday clinical setting using as measurement standards the quantitative EEG and a multifaceted battery of pre- and posttests and scans common to the clinical setting. Case studies demonstrate the nature and progress of treatment and the specific outcomes achieved.

REFERENCES

- Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry*, 4, 53–63.
- Demotes-Mainard, P., Bourgeois, M., & Vincent, J. D. (2004). Efficiency of transcranial electrostimulation on anxiety and insomnia symptoms during a wash-out period in depressed patients: A double blind study. *Biological Psychiatry*, 29, 451–456.
- Krupitsky, E., Woody, G., Katsnelson, Y., Rice, D., Bushkova, N., Palkin, Y., et al. (2002). *Transcranial Electronic Stimulation (TES) using Nexalin[®] for mild/moderate major depression in adults: A Russian randomized trial*. Unpublished manuscript.
- Kirsch, D. L. (2002). *The science behind cranial electrotherapy stimulation*. Edmonton, Canada: Medical Scope.
- Kirsch, D. L., & Smith, D. B. (2004). Cranial electrotherapy stimulation for anxiety, depression, insomnia, cognitive dysfunction, and pain: A review and meta-analysis. In P. J. Rosch & M. S. Markov (Eds.), *Bioelectromagnetic medicine* (pp. 687–699). New York: Dekker.
- Palkin, J. R., Bushkova, N. V., Demjanenko, A. M., Sderzhikova, E. I., Janushko, G. L., Bushov, K. G., et al. *Evaluation of effectiveness of use of noninvasive transcranial electrostimulation for treatment of border line depression*. SPB Psychiatric Hospital, S. Petersburg Centre of Neuroses, Leningrad Regional Addiction Center, Psycho-neurological Research Institute, and Kalaco Scientific, Inc.
- Pizinger, R., Katsnelson, Y., Krupitsky, E. M., & Woody, G. (2009) *Presentation of the Efficacy of Transcranial Electrostimulation to the Under-Secretary of Health for the Department of Veterans Affairs*. Personal communication.
- The World Health Organization. (2004a). *The global burden of disease: 2004 update*. Geneva, Switzerland: Author.
- The World Health Organization. (2004b). *The world health report 2004: Changing history, Annex Table 3: Burden of disease in DALYS by cause, sex, and mortality stratum in WHO regions, estimates for 2002*. Geneva, Switzerland: Author.

Practitioner Perspectives of Neurofeedback Therapy

Jonathon Larson, EdD, Catherine Ryan, and Mogens Baerentzen, MS
 Illinois Institute of Technology
 <larsonjon@iit.edu>

Introduction

Research continues to increase empirical support for neurofeedback therapy (NFT) efficacy and effectiveness. Hammond (2007) compiled an extensive bibliography of neurofeedback research, and Yucha and Gilbert (2008) published *Evidence-Based Practice in Biofeedback and Neurofeedback*. Kaiser (2010) reported 5,565 biofeedback and neurofeedback papers indexed within PubMed, and Arns (2010) summarized 31 applied neuroscience papers published between August and December of 2009. Despite advances in the quantity and quality of NFT research, a comprehensive literature review found a handful of investigations into practitioner

variables related to NFT process and outcome variables. Hammond and Kirk (2008) emphasized the importance of establishing formal NFT practice standards. An investigation reported demographic variables for practitioners from around the world (Rubi, 2006). A staff training program highlighted age as a potential practitioner variable for specific client types (Thompson & Thompson, 2008). Research reported the importance of exploring client and practitioner relationships (Aguilar-Prinsloo & Lyle, 2010). With limited investigation on NFT practitioner variables and recommendations from current research, we proposed to explore practitioner variables through qualitative methods. The goal and research significance of this study were to identify a wide range of NFT themes for utilization in future research on practitioner process and outcome variables.

Methods

We utilized Community Based Participatory Research (CBPR) to engage practitioners in the process of identifying NFT factors. Through online surveys, we collected demographic variables and perspectives about NFT from 70 consented participants. We utilized SPSS descriptive statistics for our demographic and NFT experience information. We utilized Loftland and Loftland's (1984) systematic filing system and Berg's (2004) themes to concepts to analyze our data set, which allowed us to categorize similar themes into conceptual frameworks.

Results

Our sample demonstrated various levels of age, education, licensures, certifications, and experience; however, we found an equal gender distribution. For the advantage conceptual framework, we found 84 concepts fitting into 6 categories. The disadvantage framework included 53 concepts within 5 categories. Practitioner characteristics were divided into three separate conceptual frameworks: skills (35 concepts in 3 categories), knowledge (29 concepts in 4 categories), and traits (36 concepts in 5 categories).

Conclusions

Our results provided 237 themes sorted into 23 categories within 5 conceptual frameworks for future research on NFT practitioner process and outcome variables. The disadvantage framework highlighted utilization and dissemination problems while the advantage framework identified variables possibly impacting NFT. The skill, knowledge, and trait conceptual frameworks offered direction for exploring practitioner variables influencing NFT outcomes. Future factor analysis research may include developing and testing a measurement tool for practitioner variables. We do not offer these findings as a comprehensive list of NFT issues or practitioner factors; rather, we offer this as a potential starting point for investigating practitioner variables related to NFT. In addition, we may have missed additional themes due to our sample size and method of data collection. Overall, we utilized CBPR and systematic filing methods to categorize practitioner perspectives to guide components of future NFT research.

REFERENCES

- Aguilar, S., & Lyle, R. (2010). Client perception of the neurofeedback experience: The untold perspective. *Journal of Neurotherapy, 14*, 55–60.
- Arns, M. (2010). News from other journals and websites. *Journal of Neurotherapy, 14*, 61–64.
- Barrett-Lennard, G. T. (1962). Dimensions of therapist response as causal factors in therapeutic change. *Psychological Monographs: General and Applied, 76*, 1–33.
- Berg, B. L. (2004). *Qualitative research methods*, 5th ed. New York: Pearson Education.
- Cattell, H. E. P., & Mead, A. D. (2008). The sixteen personality factor questionnaire (16PF). *The Sage Handbook of Personality Theory and Assessment, 2*, 135–159.
- Gaston, L., & Marmar, C. R. (1991). *Manual for the California Psychotherapy Alliance Scales—CALPAS*. Unpublished manuscript, Department of Psychiatry McGill University, Montreal, Canada.
- Goldberg, L. R. (1993). The structure of phenotypic personality traits. *American Psychologist, 48*, 26–34.
- Hammond, D. C. (2007). Comprehensive neurofeedback bibliography. *Journal of Neurotherapy, 11*, 45–60.

- Hammond, D. C., & Kirk, L. (2008). First, do no harm: Adverse effects and the need for practice standards in neurofeedback. *Journal of Neurotherapy, 12*, 79–88.
- Horvath, A. O., & Greenberg, L. (1986). The development of the Working Alliance Inventory: A research handbook. In L. Greenberg & W. Pinsoff (Eds.), *The psychotherapeutic process. A research handbook* (pp. 529–556). New York: Guilford.
- Israel, B. A., Eng, E., Schultz, A. J., & Parker, E. A. (Eds.). (2005). *Methods in community-based participatory research in health*. San Francisco, CA: Wiley.
- Kaiser, D. (2010, Spring). Letter from AAPB president: Neurocosmology and the law. *NeuroConnections*, pp. 4 & 6.
- Loftland, J. A., & Loftland, L. H. (1984). *Analyzing social settings: A guide to qualitative observation and analysis*. Belmont, CA: Wadsworth.
- McGuire-Snieckus, R., McCabe, R., Catty, J., Hansson, L., & Priebe, S. (2007). A new scale to assess the therapeutic relationship in community mental health care: STAR. *Psychological Medicine, 37*, 85–95.
- Rubi, M. C. M. (2006). Neurofeedback around the world. *Journal of Neurotherapy, 10*, 63–73.
- Yucha, C., & Montgomery, D. (2008). *Evidence-based practice in biofeedback and neurofeedback*. Wheat Ridge, CO: Association for Psychophysiology and Biofeedback.

PANEL ABSTRACTS

Neurofeedback: The Past, Present and Future

Eugenia Bodenhamer-Davis
University of North Texas
<genie@unt.edu>

Judith Lubar, LCSW
Southeastern Biofeedback Institute
Lynda Thompson, PhD
ADD Centre
Abstract by *Leslie Sherlin, PhD*

As any previous ISNR attendee will tell you much of the learning experience occurs in the hallways and around dinner tables with experienced providers. In my early career and still today I find catching conversation with the brightest minds of our field to be the most satisfying and educational experiences and the one that I always remember most from the conferences. If you're lucky enough to find an empty seat at a table with one of our elder pioneers or an up-and-coming innovator you should take advantage of this

opportunity to just capture the stories of their experience. In the meantime, ISNR has hosted a special panel that has quickly become a favorite for bringing this same idea into a room for all to enjoy.

Our first special panel took place in 2008 with contributions from Tom Budzynski, Joel Lubar, and Barry Sterman. It was truly one of the most entertaining and educational events of the conference as we all came together to share experiences and speculate about our common future. The panel participants are given a very loose goal of making a 30-min presentation each sharing their perspective of neurofeedback's past, present, and future. It ranged from a photo slideshow of other pioneers and colleagues highlighting landmark achievements, presentation of data and studies, to simple storytelling about our field and accomplishments. In 2009 we were very honored to hear from Joe Kamiya, Juri Kropotov, and Nancy White. This year I have decided to turn our focus to a few of the pioneering women of our field. The invited speakers will be Eugenia Bodenhamer-Davis, Judith Lubar, and Lynda Thompson. Be sure to arrive early on Wednesday to participate in the welcome reception and the special panel where we hear these pioneering women give us their perceptions on "Neurofeedback: the Past, Present and Future."

Neurofeedback for Epilepsy: A Review and Update

Gabriel Tan, PhD
VA Medical Center
<tan.gabriel@va.gov>

D. Corydon Hammond, PhD
University of Utah Medical Center
Jonathan Walker, MD
Neurotherapy Center of Dallas
Robert Coben, PhD
Neurorehabilitation & Neuropsychological Services

This symposium is intended to provide both a review and an update on the use of neurofeedback (NF) to treat individuals suffering from epilepsy, particularly those who appear

to be resistant to traditional pharmacotherapy. The prevalence and types of epilepsy along with the respective treatment outcome data are briefly described. The symposium then describes NF approaches and protocols for the treatment of epilepsy (including SMR and SCP training). The discussion includes a presentation of a recently published meta-analysis on NF and epilepsy (Tan et al., 2009). In addition, the low energy NF system is also briefly presented (Hammond, 2007; Larsen, 2006; Ochs, 2006). The symposium also discusses an approach that focuses on individualizing clinical protocol based on QEEG findings (Walker, 2008; Walker & Kozlowski, 2005) to improve on the outcome. Last but not least, data supporting the use of genotype/EEG connectivity analyses in the treatment of epilepsy in autistic children are presented. Q & A session at the end will hopefully solicit input from the audience for further discussion.

REFERENCES

- Ayers, M. E. (1988). Long-term clinical treatment follow-up of EEG neurofeedback for epilepsy. *Epilepsy Support Program Newsletter*, 3(2), 8–9.
- Ayers, M. E. (1995). Long-term follow-up of EEG neurofeedback with absence seizures. *Biofeedback & Self-Regulation*, 20, 309–310.
- Collura, T. F. (2010). *Advanced BrainMaster EEG Neurofeedback Practicum*. Future Health.
- Collura, T. F., Guan, J., Tarrant, J., Bailey, J., & Starr, F. (2010). EEG biofeedback case studies using live Z-scores and a normative database. *Journal of Neurotherapy*, 14, 22–46.
- Gehring, A., & Blaser, A. (1993). *MMPI – Minnesota Multiphasic Personality Inventory*. Deutsche Kurzform für Handauswertung. 2. Aufl. Bern, Switzerland: Huber.
- Hammond, D. C. (2007). *LENS: The Low Energy Neurofeedback System*. New York: Haworth.
- Hammond, D. C., & Kirk, L. (2008). First, do no harm: Adverse effects and the need for practice standards in neurofeedback. *Journal of Neurotherapy*, 12, 79–88.
- Hammond, D. C. (2010). The need for individualization in neurofeedback. *Applied Psychophysiology & Biofeedback*, 35, 31–36.
- Hautzinger, M., Bailer, M., Worall, H., & Keller, F. (1994). *Beck-Depressions-Inventar* (Bearbeitung der deutschen Ausgabe). Bern, Switzerland: Hans Huber.
- John, E. R., et al. (1987). Normative databanks and neurometrics: Basic concepts, methods and results of norm construction. In A. Remond (Ed.), *Handbook of electroencephalography and clinical neurophysiology* (Vol. 3, pp. 449–495). Amsterdam: Elsevier.
- Kraak, B., & Nord-Rüdiger, D. (1989). *Fragebogen zu Lebenszielen und zur Lebenszufriedenheit*. Göttingen, Germany: Hogrefe.
- Krampe, G. (1991). *Fragebogen zu Kompetenz- und Kontrollüberzeugungen*. Göttingen, Germany: Hogrefe.
- Larsen, S. (2006). *The healing power of neurofeedback: The revolutionary LENS technique for restoring optimal brain function*. Rochester, VT: Healing Arts Press.
- Nelson, H. E. (1976). A modified card sorting test sensitive to frontal lobe defects. *Cortex*, 12, 313–324.
- Nuwer, M. R. (1988). Frequency analysis and topographic mapping of EEG and evoked potentials in epilepsy. *EEG*, 69, 18–126.
- Ochs, L. (2006). The Low Energy Neurofeedback System (LENS): Theory, background, and introduction. *Journal of Neurotherapy*, 10(2–3), 5–39.
- Reichert, M., & Perrez, M. (1993). *Fragebogen zum Umgang mit Belastung im Verlauf-UBV*. Bern, Switzerland: Hans Huber.
- Sterman, M. B. (2000). Basic concepts and clinical findings in the treatment of seizure disorders with EEG operant conditioning. *Clinical Electroencephalography*, 32, 45–55.
- Tan, G., Thornby, J., Hammond, D. C., Strehl, U., Canady, B., Amemann, K., et al. (2009). Meta-analysis of EEG biofeedback in treating epilepsy. *Clinical EEG & Neuroscience*, 40, 173–179.
- Walker, J. E. (2008). Power spectral frequency and coherence abnormalities in patients with intractable epilepsy and their usefulness in long-term remediation of seizures using neurofeedback. *Clinical EEG & Neuroscience*, 39, 203–204.
- Walker, J. E., & Kozlowski, G. P. (2005). Neurofeedback treatment of epilepsy. *Child & Adolescent Psychiatric Clinics of North America*, 14, 163–176.

Applications of QEEG and Neurofeedback in Sport Psychology

Michael Linden, PhD
ADD Treatment Centers
<drmike49@aol.com>

Jeffrey Fannin, PhD
Center for Cognitive Enhancement
Wes Sime, PhD
First Step Recovery & Wellness Center

The science of sport psychophysiology has been developing gradually over the past 20

to 30 years. Initially most theoretical models and intervention techniques were cognitive and behaviorally based. In recent years the principles of the Zone of Optimal Performance were developed, which features statistical models to calculate the ideal range of arousal and confidence associated with best outcomes. More recently, brain wave patterns and ratios of various frequencies and states of consciousness have been assessed during performance in preparation to conduct training protocols designed to enhance the qualities of focus, concentration, and more relevant terms such as “being fully engaged or absorbed in the task at hand.”

The panel brings together the expertise of three professionals approaching sport performance from different perspectives. These perspectives include (a) QEEG profiles of successful athletes and QEEG based Neurofeedback, (b) QEEG Assessment and Neurofeedback with athletes with ADD and Asperger’s, and (c) General Neurofeedback combined with other physiological techniques with athletes.

The outcome of this panel session should be to advance the science of sport psychophysiology to utilize more accurate neurological (QEEG) assessment and training (Neurofeedback) strategies with athletes.

REFERENCES

- Blumenstein, B. (2002). Biofeedback applications in sport and exercise: Research findings. In B. Blumenstein, M. Bar-Eli, & G. Tenenbaum (Eds.), *Brain and body in sport and exercise: Biofeedback applications in performance enhancement* (pp. 37–54). New York: Wiley & Sons.
- Blumenstein, B., Bar-Eli, N., & Tenenbaum, G. (2002). *Biofeedback applications in performance enhancement: Brain and body in sport and exercise*. New York: Wiley & Sons.
- Hatfield, B. D., Haufler, A. J., Hung, T. M., & Spalding, T. W. (2004). Electroencephalographic studies of skilled psychomotor performance. *Journal of Clinical Neurophysiology*, 21, 144–156.
- Hatfield, B. D., Haufler, A. J., & Spalding, T. W. (2006). A cognitive neuroscience perspective on sport performance. In E. Acevedo & Ekkekakis (Eds.), *Psychobiology of physical activity* (pp. 221–240). Champaign, IL: Human Kinetics Press.
- Fannin, J. (2009a). Is brain mapping the future of leadership? *BNet Insight*.
- Fannin, J. (2009b). *Mapping the complex mind*. Tempe: Arizona State University Research.
- Sime, W. E. (2000, October). *The use of biofeedback and neurofeedback in applied psychophysiology to obtain objective assessment of progress in reducing stress and tension in performance and increasing attentional focus*. Paper presented at the meeting of the American Association for Applied Sport Psychology, Nashville, TN.
- Sime, W., & Silverman, S. (2008, January). Many roads to Rome: Becoming fully engaged in the performance. *NeuroConnections*.
- Strack, L., & Wilson (Eds.). (in press). *Athletes with Asperger’s and Attention Deficit Disorder in Applications of Biofeedback and Neurofeedback in Sport Psychology*. AAPB Publishing.

POSTER ABSTRACTS

A Study Comparing the Brain Function of Healthy and ADHD Adults During Rest and Go/NoGo Task in EEG/ERP and fMRI

Cynthia Kerson, PhD

ISNR

<office@isnr.org>

Leslie Sherlin, PhD

NovaTech EEG

Estate Sokhadze, PhD

University of Louisville

Rex Cannon, PhD

University of Tennessee

David Hubbard, MD

Hubbard Foundation

Discussion

The prevalence of Attention Deficit Hyperactivity Disorder (ADHD) is an estimated 4.1% in adults, second only to depression. Recently, several quantitative electroencephalographic (QEEG), event-related potential (ERP), and functional magnetic resonance imaging (fMRI) studies have been completed to examine electrophysiological and blood flow behaviors in adults with Attention Deficit Hyperactive Disorder (ADHD). This study utilizes concomitant neuroimaging methodologies to examine the default mode network (DMN) in healthy and ADHD adults to ascertain differences during rest and Go/NoGo

task. The DMN consists of 12 functionally related regions that are consistently shown increased in activity in an eyes-closed resting condition as compared to functionally specific cognitive tasks or eyes-opened resting condition. Recent data indicate dysfunction in right parietal areas in ADHD as compared to control. However, the strength of the temporal connections in EEG frequency domains has not been investigated in this population.

Objectives

This EEG/ERP/fMRI study proposes to correlate brain behavior from each neuroimaging method and elucidate functional connectivity patterns in the ADHD group during resting state (eyes open and eyes closed) and an active cognitive task. Recruitment of neural resources involving temporal correlations may provide important information about both attentional and self-regulatory processes in ADHD individuals as compared to healthy controls. These data may provide important information relating to potential biomarkers for ADHD as well as to increase the specificity of methods for neurotherapy treatment of ADHD. The data may also confirm that EEG is an adequate methodology to evaluate ADHD. Given the regional deficits shown in ADHD research we examine the default network regions and their specific relationship with the bilateral anterior insular cortices. Numerous regions within the default network, especially left medial prefrontal and anterior medial regions are shown (assuming sources at or near the surface electrode F3, Fz, F7 contribute to the ERP average) to contribute many of the putative mechanisms found in ERP research (e.g., frontal NoGo-N2 and P3, Error-related Negativity, etc.).

Methods

In this study, we recruit 16 participants (8 healthy and 8 ADHD adults). We attempt to recruit an equal number of age-similar male and female individuals. The ADHD adults are recruited through local clinicians and CHADD chapters. They have been initially interviewed by phone and administered the

Connors Rating Scale and the Mini International Neuropsychiatric Interview to determine accuracy of symptom reporting and to rule out psychological comorbidities. Exclusion criteria consist of previous head trauma, recent drug or alcohol abuse (14 days), or neurological syndromes. We record sequential 19-channel EEG, ERP, and fMRI during the eyes open and closed states and while performing the TOVA Go/NoGo continuous attention test. Eyes open and eyes closed states are recorded for approximately 5 min. The TOVA test takes approximately 20 min to administer. The QEEG results are evaluated with comparison to a normative database and with the standardized low-resolution electromagnetic tomography (eLORETA) analysis. Functional connectivity is assessed using the seed-based approach in eLORETA. The fMRI results will be evaluated using Brain VoyagerTM and other neuroimaging software packages.

REFERENCES

- Beauregard, M., & Levesque, 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 & Biofeedback, 31*, 3–20.
- Bregadze, N., & Lavric, A. (2006). ERP differences with vs. without concurrent fMRI. *International Journal of Psychophysiology, 62*, 54–59.
- Karakas, H. M., Karakas, S., Özkan Ceylan, A., & Tali, E. T. (2009). Recording event-related activity under hostile magnetic resonance environment: Is multimodal EEG/ERP-MRI recording possible? *International Journal of Psychophysiology, 3*, 6.
- Matsuda, T., Matsuura, M., Ohkubo, T., Ohkubo, H., Takahashi, K., Tamaki, M., et al. (2002). Simultaneous recording of EEG and functional MRI. *International Congress Series, 1232*, 351–355.
- Otswald, D., Porcara, C., & Bagshaw, A. P. (2010). An informative theoretic approach to EEG-fMRI integration of visually evoked responses. *NeuroImage, 49*, 498–516.
- Uddin, L. Q., Kelly, A. M., Bharat, B., Margulies, D. S., Shehzad, Z., Shaw, D., et al. (2008). Network homogeneity reveals decreased integrity of default-mode network in ADHD. *Journal of Neuroscience Methods, 169*, 249–254.
- Verkhlyutov, V. M., Gapienko, G. V., Ushakov, V. L., Portnova, G. V., Verkhlyutova, I. A., Anisimov,

N. V., et al. (2010). MRI morphometry of the cerebral ventricles in patients with attention deficit hyperactivity disorder. *Neuroscience and Behavioral Physiology*, 40, 295–303.

Factors Influencing Neuroscience Grades of Medical Students

Liris Benjamin, MBBS, MPH, PhD, Phillip Cooles, MD, Alexander Martin, PhD, Laura Welke, PhD, and Griffin Benjamin, MD
Ross University School of Medicine
<lbenjamin@rossmed.edu.dm>

Introduction

Neuroscience has rapidly become an integral part of living and society. This growth necessitates a greater understanding and simplification of subject. New approaches to medical education need to be incorporated into the curriculum to enable students to meet its evolving dimensions. These approaches must target not only the physical constructs of the material but also the bio-psychosocial components of both the learner and the educator.

As described earlier by Bloom, but still fundamental to education today, learning should involve knowledge (cognition), attitude (affective), and skills (psychomotor). In other words, not only the head but also the heart and hand of our students need to be considered. The aim of this study was therefore to determine whether students' prior knowledge, attitude or study practice influenced their Neuroscience grades.

Methods

A cross-sectional survey was conducted using an electronic self-administered questionnaire via Survey Monkey. The instrument was designed to gain information from third-semester medical students that had previously done the Neuroscience course. Self-reported information on students' prior knowledge (MCAT scores), attitude, and study practices toward Neuroscience and their final grades was collected.

The study was conducted in collaboration with members from the Anatomy and

Introduction to Clinical Medicine departments. Anonymity and confidentiality was maintained because names and identification were not requested and responses were not directly linked to addresses. Data were analyzed using correlation where $p < .05$ was determined as significant.

Results

The study indicated that students entering with higher MCAT scores were more likely to have higher Neuroscience grades ($p = .03$). It also showed that students who spent more hours reviewing lectures on media site were more likely to have higher grades ($p = .04$). There was no relationship between attitude and grade ($p = .29$).

Discussion and Conclusion

Media site is an effective learning resource for Neuroscience students. This method of delivery of the curriculum is apt for medical students who normally have arduous schedules. Furthermore, MCAT score is a predictor of Neuroscience grades.

REFERENCES

- Bloom, B. S. (Ed.). (1956). *Taxonomy of educational objectives. The classification of educational goals* (pp. 201–207). Susan Fauer Company.
- Howe, A., Campion, P., Searle, J., & Smith, H. (2004). Learning in practice. New perspectives—approaches to medical education at four new UK medical schools. *British Medical Journal*, 329, 327–331.

EEG Correlates of Improved Outcomes from SDOC Using a Novel Treatment Protocol

Philip DeFina, PhD
International Brain Research Foundation
<pdefina@ibrfinc.org>

James Thompson, PhD
Comprehensive Neuroscience Center
<jameswgthompson@gmail.com>

Jonathan Fellus, MD, Rosemarie Moser, PhD, Philip Schatz, PhD, Monika Eller,

OTR, Maria McNish, MS, Pasquale Frisina, PhD, and Charles Prestigiaco, MD

Objective

The purpose of this research is to validate clinical progression and emergence in patients with Severe Disorders of Consciousness (SDOC), following a novel Multi-modal Care Protocol (MCP), using electroencephalogram (EEG) data.

Methods

Participants were 21 SDOC patients from the following etiologies: Vegetative State (VS) traumatic (VS-TBI; $n = 6$), VS nontraumatic (VS-NTBI; $n = 10$), Minimally Conscious State (MCS-TBI; $n = 3$), MCS nontraumatic (MCS-NTBI; $n = 2$). Patients in the study received the MCP protocol, a 12-week innovative coma recovery protocol involving polypharmacy, nutraceuticals, median nerve stimulation, and traditional therapies. The MCP is designed to maximally normalize electrochemical balance, through multimodal neuromodulation, optimizing the brain's ability to heal and repair the injured cells and networks. Patients were assigned to independent groups based on emergence from VS/MCS ($n = 13$), clinical improvement ($n = 4$), or no change ($n = 4$). Design was a within-subjects retrospective case series measuring pre-post MCP intervention EEG data. Main Outcome Measures were: Coma Recovery Scale-Revised (CRS-R), Disability Rating Scale (DRS), Delta-Alpha Ratio (DAR), Theta-Beta ratio (TBR), Power Ratio Index (PRI), Total Asymmetry (TotAsym), and Peak Frequency (Peak Hz).

Results

Outcome groups showed no significant differences on EEG measures at baseline. Patients emerging from SDOC showed significant changes from baseline on CRS-R and DRS scores, as well as DAR, TBR, PRI, and changes in Peak Hz in posterior

regions. No significant changes were noted on EEG measures for patients in the "clinical improvement" or "no change" groups.

Conclusions

Results provide objective validation of the efficacy of the MCP in improving patient outcomes, and the validity of EEG measures in quantifying patient recovery status.

REFERENCES

- American Academy of Neurology. (1995). Practice parameter: Assessment and management of persons in the persistent vegetative state. *Neurology*, *45*, 1015–1018.
- American Medical Association. (2009). *Current procedural terminology*. Washington, DC: Author.
- Andrews, K., et al. (1996). Misdiagnosis of the vegetative state: Retrospective study in a rehabilitation unit. *British Medical Journal*, *313*, 13–16.
- Childs, N. L., Mercer, W. N., & Childs, H. W. (1993). Accuracy of diagnosis of persistent vegetative state. *Neurology*, *43*, 1465–1467.
- Coleman, M. R., et al. (2009). A multimodal approach to the assessment of patients with disorders of consciousness. *Progress in Brain Research*, *177*, 231–248.
- Finnigan, S. P., et al. (2007). Quantitative EEG indices of sub-acute ischaemic stroke correlate with clinical outcomes. *Clinical Neurophysiology*, *118*, 2525–2532.
- Giacino, J., & Kalmar, K. (1997). The vegetative and minimally conscious states: A comparison of clinical features and functional outcome. *Journal of Head Trauma Rehabilitation*, *12*(4), 36–51.
- Giacino, J. T., et al. (2002). The minimally conscious state: Definition and diagnostic criteria. *Neurology*, *58*, 349–353.
- Leon-Carrion, J., et al. (2009). Delta-alpha ratio correlates with level of recovery after neurorehabilitation in patients with acquired brain injury. *Clinical Neurophysiology*, *120*, 1039–1045.
- Mohonk Report. (2006). *A report to congress improving outcomes for individuals with disorders of consciousness, assessment, treatment, and research needs*. Mohonk, NY.
- The Multi-Society Task Force on PVS. (1994). Medical aspects of the persistent vegetative state. *New England Journal of Medicine*, *330*, 1499–1508.
- Shepperd, S., et al. (2009). Can we systematically review studies that evaluate complex interventions? *PLoS Medicine*, *6*(8), e1000086.

- Whyte, J., Laborde, A., & DiPasquale, M. (1999). Assessment and treatment of the vegetative and minimally conscious patient. In M. Rosenthal, E. R. Griffith, J. S. Kreutzer, & B. Pentland (Eds.), *Rehabilitation of the adult and child with traumatic brain injury* (3rd ed., pp. 435–452). Philadelphia: FA Davis.
- Zumsteg, D., Hungerbuhle, H., & Wieser, H. (2004). *Atlas of adult electroencephalography*. Bad Honnef, Germany: Hippocampus Verlag.

Whole-Head EEG Effects of Lateralized EEG Biofeedback Training

Andrew Hill, MA, Cphil, and Eran Zaidel, PhD
UCLA
<andrewhill@ucla.edu>

Clinical EG Biofeedback is typically performed with one or two electrode channels. Selection of training sites is based on clinical education and experience and rationalization of scalp location related to underlying neuroanatomical structures. For example, left anterior sites may be trained for language, temporal sites for emotion regulation, parietal sites for sensory integration, and so on. It is unclear however to what extent the choice of training site affects the underlying EEG produced at that training site compared to EEG at other sites throughout the scalp.

We are performing a short but intense course of EEG Biofeedback, with concomitant lateralized behavioral testing. Dense array (64-channel) EEG is recorded during eyes open and eyes closed non-task baselines, under lateralized continuous and transient attention tasks, and in response to single-channel EEG feedback conditions. Four groups (double-blind, random assignment) with different EEG Biofeedback protocols are being trained with contrasting EEGBF protocols including C4-A2 SMR, C3-A1 SMR, C3-A1 Beta, and a Sham EEGBF condition.

With this data we will be able to correlate changes in whole-head EEG with EEGBF training on discrete channels, contrast different EEGBF protocols to determine if they produce different EEG effects, and

behaviorally examine attention processes as they occur in each hemisphere separately. This approach will allow clinical selection of training protocols to be informed by models of lateralized attention as well as possibly elucidating the methods of action of EEGBF that are related to electrode placement.

REFERENCES

- Fallgatter, A. J., & Herrmann, M. J. (2001). Electrophysiological assessment of impulsive behavior in healthy subjects. *Neuropsychologia*, *39*, 328–333.
- Gruzelier, J. G., & Egner, T. (2005). Critical validation studies of neurofeedback. *Child and Adolescent Psychiatric Clinics of North America*, *14*, 83–104.
- Hill, A., Barnea, A., Herzberg, K., Rassis-Ariel, A., Rotem, S., Meltzer, Y., et al. (2008). *Measuring and modulating hemispheric attention*. In F. Aboitiz & D. Cosmelli (Eds.), *From attention to goal-directed behavior* (pp. 125–144). New York, NY: Springer.
- Raz, A. (2004). Attentional mechanisms and networks. In C. Spielberger (Ed.), *Encyclopedia of applied psychology* (pp. 203–208). San Diego, CA: Elsevier Science.
- Riccio, C. A., Reynolds, C. R., Low, P., & Moore, J. J. (2002). The continuous performance test: A window on the neural substrates for attention? *Archives of Clinical Neuropsychology*, *17*(3), 235–272.
- Zaidel, E., Clarke, J. M., & Suyenobu, B. (1990). Hemispheric independence: A paradigm case for cognitive neuroscience. In A. B. Scheibel & A. F. Wechsler (Eds.), *Neurobiology of higher cognitive function*, (pp. 297–355). New York: Guilford.

Neurofeedback, Cranial Electrotherapy Stimulation, and Microcurrent Electrical Therapy to Treat Tinnitus: A Case Series

Mark Lawrence Johnson, MS, and Eugenia Bodenhamer-Davis, PhD
University of North Texas
<markjohnson2@my.unt.edu>

Background

Tinnitus is a vexing disorder characterized by phantom sound perceptions that have no external source. Individuals with tinnitus who seek treatment are often told they must learn to live with it. There has been some

initial evidence that neurofeedback and electrical stimulation therapy modalities may suppress tinnitus in some individuals. The purpose of this study was to retroactively examine a case series of clients who were treated for tinnitus with varied neuromodulatory interventions to extend the current literature featuring these modalities and also to more closely consider etiological and other clinical considerations.

Measures

Five client records were obtained from a university-based neurotherapy clinic. These records were used to examine the efficacy of several different treatment modalities used to treat tinnitus, which included neurofeedback, cranial electrotherapy stimulation, and microcurrent electrical therapy. These records were then assessed for pre- and post-changes on assessment measures related to treatment outcomes.

Results

Statistical analyses are in progress. It is expected that independent *t*-test results will reveal significant improvements in tinnitus severity in some participants.

Conclusions

Depending on the etiology of the tinnitus, pre- and post-test measures (EMG baseline, BAI, BDI, BHS, PSQI, and MCMI) are also expected to yield some etiological considerations that might inform treatment strategies.

REFERENCES

- Dohrmann, K., Weisz, N., Schlee, W., Hartmann, T., & Elbert, T. (2007). Neurofeedback for treating tinnitus. *Progress in Brain Research*, *166*, 473–485.
- Engelberg, M., & Bauer, W. (1985). Transcutaneous electrical stimulation for tinnitus. *Laryngoscope*, *95*, 1167–1172.
- Saunders, J. C. (2007). The role of central nervous system plasticity in tinnitus. *Journal of Communication Disorders*, *40*, 313–334.
- Vernon, J. (1987). Use of electricity to suppress tinnitus. *Seminars in Hearing*, *8*(1), 29–48.
- Weiler, E. W. J., Brill, K., Tachiki, K., & Schneider, D. (2002). Neurofeedback and quantitative electroencephalography. *International Tinnitus Journal*, *8*, 87–93.
- Weisz, N., Moratti, S., Meinzer, M., Dohrmann, K., & Elbert, T. (2005). Tinnitus perception and distress is related to abnormal spontaneous brain activity as measured by magnetoencephalography. *PLoS Medicine*, *2*(6), 0546-0553.

EEG Predictors of Treatment Response to Cognitive-Behavioral Therapy in Patients with Obsessive-Compulsive Disorder

Jana Koprivova, MA, Michal Raszka, MD, Jan Prasko, MD, PhD, Martin Brunovsky, MD, PhD, and Jiri Horacek, MD, PhD
Prague Psychiatric Center, and Charles University, Prague
<koprivova@pcp.lf3.cuni.cz>

Background

Despite medical progress, a high percentage of obsessive-compulsive patients are still not responding at all or not responding sufficiently to current treatment. There is a need to seek predictors of treatment outcome that would help in the choice of the best treatment option in particular cases. EEG studies previously reported that patients not responding to selective serotonin reuptake inhibitors (SSRIs) show abnormalities in the theta (Prichep et al., 1993) or beta (Fontenelle et al., 2006) frequency band. The aim of our study was to look for EEG predictors of treatment response to cognitive behavioral therapy. Based on previous findings on EEG abnormalities in anterior cingulate cortex (ACC; Koprivova et al., submitted; Sherlin & Congedo, 2005) and its predictive value for treatment response to SSRIs (Fontenelle et al., 2006) we focused on ACC and its ventral and dorsal subdivision.

Methods

We analyzed EEG of 15 OCD patients (age = 31.4 ± 8.05; 6 men and 9 women; 5

drug-free and 10 medicated with SSRIs) who were suffering from marked obsessive-compulsive symptoms (the mean score at the Yale–Brown Obsessive-Compulsive Scale [Y-BOCS] was 22.9 ± 7.61) and took part in a 6-week intensive cognitive behavioral therapy program. Clinical obsessive-compulsive symptoms as well as symptoms of general anxiety (according to the Beck Anxiety Inventory [BAI]) were assessed twice, before and after the therapy. EEG was recorded pretreatment. EEG data were analyzed in 8 frequency bands (delta 2–6 Hz, theta 6.5–8 Hz, alpha1 8.5–10 Hz, alpha2 10.5–12 Hz, beta1 12.5–18 Hz, beta2 18.5–21 Hz, beta3 21.5–30 Hz, gamma 30.5–40 Hz). Based on the priori hypothesis, absolute and relative current density power was computed in ACC and its ventral and dorsal subdivision using the standardized Low-Resolution Electromagnetic Tomography software (Pascual-Marqui, 2002). Spearman correlations were computed between absolute and relative current density power and relative change in psychopathology (Y-BOCS score, obsession and compulsion subscore, Beck Depression Inventory and BAI scores).

Results

The improvement of clinical symptoms after cognitive behavioral therapy was negatively related to the amount of absolute and relative theta pretreatment ($p = .05$). Higher absolute and relative theta in ventral ACC and total ACC before treatment was related with smaller relative improvement of Y-BOCS score and obsession subscore. The improvement of obsession subscore was also negatively related with relative theta in dorsal ACC. Subjectively rated improvement of general anxiety as measured with BAI was negatively related with relative theta in total ACC as well as in its both subdivisions and with relative alpha1 current density power in dorsal ACC. There was no correlation between subjectively rated improvement of depressive symptoms (Beck Depression Inventory score) and EEG before treatment.

Discussion

Our results are consistent with previous findings of higher theta in SSRIs nonresponders (Prichep et al., 2003). Theta in OCD generated in medial frontal cortex has been linked with an enhanced performance monitoring and obsessive thoughts (Fitzgerald et al., 2005; Maltby et al., 2005). It is possible that elevated theta activity may be a general predictor of worse treatment outcome in OCD. This is a challenge for neurofeedback aimed at reducing the theta activity that might help to overcome the resistance.

Acknowledgments

This work was supported by the grant IGA NS9751-3/2008 provided by the Ministry of Health of the Czech Republic and CNS MSMT CR 1M0517 provided by the Ministry of Education, Youth and Sports of the Czech Republic.

REFERENCES

- Fitzgerald, K. D., Welsh, R. C., Gehring, W. J., Abelson, J. L., Himle, J. A., Liberzon, I., et al. (2005). Error-related hyperactivity of the anterior cingulate cortex in obsessive-compulsive disorder. *Biological Psychiatry*, *57*, 287–294.
- Fontenelle, L. F., Mendlowicz, M. V., Ribeiro, P., Piedade, R. A., & Versiani, M. (2006). Low-resolution electromagnetic tomography and treatment response in obsessive-compulsive disorder. *International Journal of Neuropsychopharmacology*, *9*(1), 89–94.
- Koprivova, J., Congedo, M., Horacek, J., Prasko, J., Raszka, M., Brunovsky, M., et al. (submitted). *EEG source analysis in obsessive-compulsive disorder using standardized low-resolution electromagnetic tomography and independent component analysis*. Manuscript submitted for publication.
- Maltby, N., Tolin, D. F., Worhunsky, P., O'Keefe, T. M., & Kiehl, K. A. (2005). Dysfunctional action monitoring hyperactivates frontal-striatal circuits in obsessive-compulsive disorder: an event-related fMRI study. *Neuroimage*, *24*, 495–503.
- Pascual-Marqui, R. D. (2002). Standardized low-resolution brain electromagnetic tomography (sLORETA): Technical details. *Methods & Findings in Experimental & Clinical Pharmacology*, *24*(Suppl. D), 5–12.

Prichep, L. S., Mas, F., Hollander, E., Liebowitz, M., John, E. R., Almas, M., et al. (1993). Quantitative electroencephalographic subtyping of obsessive-compulsive disorder. *Psychiatry Research*, *50*, 25–32.

Sherlin, L., & Congedo, M. (2005). Obsessive-compulsive dimension localized using low-resolution brain electromagnetic tomography (LORETA). *Neuroscience Letters*, *387*, 72–74.

Investigating the P900 Component with 5-Button CTP Protocol for Assessing Memory Deficit and Malingering

Elena Labkovsky, PhD, and J. Peter Rosenfeld, PhD

Northwestern University
<e-labkovsky@northwestern.edu>

Introduction

For our studies on investigation of deception, memory deficit and malingering we developed an ERP-based Complex Trial Protocol (CTP; Rosenfeld et al., 2004). The modified version of the original CTP was later developed and 5-button random response was introduced to the protocol for increasing the CTP accuracy. To demonstrate that the modified CTP is resistant to countermeasure use, we taught our participants to implement mental “countermeasures” (CMs). Adding the 5-button random response seems to cause emergence of a novel long latency ERP component -P900.

The current study is an attempt to further investigate the P900 component.

Methods

In the CTP with random button responses (Rosenfeld et al., 2008) a rare Probe (P) or frequent Irrelevant (I) stimulus appears on screen first and is followed by either a target (T) or nontarget (NT) stimulus in the second part of the same trial. There were 4 different Irrelevants and 1 Probe included. A participant was instructed to randomly press 1 of 5 buttons on one response box to the first stimulus (P or I). Then the participant pressed 1 of 2 buttons on another response box for T or NT.

In our previous studies the biggest P900 amplitude for the relevant stimulus (P) was found in the group with 2 (of 4) countered I stimuli. Based on that it was decided to include the following groups in the present study:

1. 2CM—one of the stimuli was a participant’s birth date (P) and 2 (of 4) I stimuli were countered.
2. 2CMPrC—one of the stimuli was a participant’s birth date (P) and 2 (of 5) stimuli were countered. One countered stimulus was the P and another was one of the 4 Is.
3. 2CM IN PrC—there was no P (all 5 stimuli in the protocol were irrelevant to the subject). Two (of 5) stimuli were countered.
4. NoCM IN—there was no P (all 5 stimuli in the protocol were irrelevant to the participant). There were no countermeasures involved.

The total number of participants was 49.

Results

The group amplitudes for P900 (peak-peak), uV (SD in parentheses), were as follows:

1. 2CM group ($n = 13$)—P: 4.130 (3.004), Iall (all 4 Is combined): 3.320 (2.441).
2. 2CM IN PrC group ($n = 11$)—P: 1.337 (0.739), Iall: 0.860 (0.694).
3. 2CM PrC group ($n = 12$)—P: 2.727 (2.308), Iall: 1.643 (1.076).
4. NoCM IN group ($n = 13$)—P: 1.448 (1.861), Iall: 0.595 (0.703).

A mixed analysis of variance (ANOVA; 4 group \times 2 stimulus type) yielded $F(3, 45) = 7.007$, $p < .001$ for groups and $F(1, 45) = 14.172$, $p < .001$ for stimulus type (P vs. Iall). There was no interaction found, $F(3, 45) = 0.319$, $p = .812$.

The follow-up ANOVAs (4 groups) separately for P and Iall (peak-to-peak amplitudes, uV) with a post hoc Bonferroni adjustment showed no difference between the two “innocent” groups (NoCM IN and 2CM IN), $p = .111$ (P) and $p = 1.0$ (Iall).

There was a significant difference between 2CM group and each of the two innocent groups for both P and Iall: $p = .19$ (P, 2CM IN PrC), $p = .001$ (Iall, 2CM IN PrC); and $p = .18$ (P, NoCM IN), $p = .036$ (Iall, NoCM IN). In the group with Probe and 2 countermeasures (one of the countered stimuli was the P), no significant difference was found for the P when comparing with either 2CM group ($p = .689$) or each of the innocent groups ($p = .803$ – 2CM In PrC; $p = .9$ – NoCM IN).

Iall in 2CM group significantly differed from each of the 3 other groups ($p = .001$ – 2CM IN PrC; $p = .036$ – 2CM PrC; and $p = .000$ – NoCM IN). There was no difference in Iall between the 3 later groups.

Conclusions

The results demonstrate that the presence of a relevant to the subject item (P) is crucial in eliciting the P900 component with the 5-button CTP protocol and countermeasures to 2 (of 4) irrelevants. When P and an I share the same characteristic (as it is in 2CM PrC where one I and the P are countered, thus making the P similar to the I) the P900 amplitude to the P becomes smaller.

P900 may represent an “action monitoring” component. It appears in the frontal-central areas of the scalp and emerges in a potentially conflict situation where a prepared action might need to be withheld or if an action was executed but mismatch between the outcome and the task requirements was detected after that. Further investigation of the P900 component is required.

REFERENCES

- Andreassi, J. L. (2000). *Psychophysiology: Human behavior and physiological response* (4th ed.). Mahwah, NJ: Erlbaum.
- Kropotov, J. (2009). *Quantitative EEG, event-related potentials and neurotherapy*. New York: Elsevier.
- Labkovsky, E., & Rosenfeld, J. P. (2008). Detecting simulated amnesia for self-referred information with enhanced ERP(P300)-based protocol. *Archives of Clinical Neuropsychology*, 23, 664.
- Labkovsky, E. B., Rosenfeld, J. P., & Sokolovsky, A. (2009). Putative novel ERP component (P900) as an indicator of countermeasure use in a modified complex trial protocol. *Psychophysiology*, 46, s1-s142.
- Luck, S. J. (2005). *An introduction to the event-related potential technique*. Cambridge, MA: MIT Press.
- Rosenfeld, J. P., Labkovsky, E., Winograd, M., Lui, M., Vandenboom, C., & Chedid, E. (2008). The Complex Trial Protocol (CTP): A new, countermeasure-resistant, accurate P300-based method for detection of concealed information. *Psychophysiology*, 45, 906-919.
- Rosenfeld, J. P., Soskins, M., Bosh, G., & Ryan, A. (2004). Simple effective countermeasures to P300-based tests of detection of concealed information. *Psychophysiology*, 41, 205-219.

A Novel “Double-Probe Complex Trial Protocol” for Assessment of Memory Deficit and Malingering

Elena Labkovsky, PhD, and J. Peter Rosenfeld, PhD

Northwestern University
<e-labkovsky@northwestern.edu>

Introduction

The Complex Trial Protocol (CTP) is a P300-based memory deficit and malingering test. It proved effective and accurate with self-referred (autobiographical) information experiments (hit rate: 92-100%; Rosenfeld et al., 2008).

In the CTP protocol, each trial contains two parts. There is a Probe (P; a relevant to the participant item [his/her birthday] or Irrelevant (I; any date, *not* relevant to the participant) in the first part of a trial, and a Target (T; an item with “assigned significance”) or Non-Target (NT; item without any specific meaning) in the second part of each trial. The two stimuli are separated by about 1 s.

In the present study we examine a novel “Double-Probe” CTP. It differs from the original CTP in that we replace NT stimuli in the second part of a trial with a new set of a P (P2) and three Is (NT1, NT2, and NT3). All the stimuli in the second part are from a different category compared to the first part. Thus, there were dates in the first part and city names—in the second part of a trial.

We hypothesize that introducing the second set of a P and Is will increase accuracy of the protocol.

Methods

Participants ($N = 13$) were undergraduate students recruited from a student pool at Northwestern University.

In the first part of a trial there were 4 different I stimuli (I1, I2, I3, and I4) and 1 P (P1). All stimuli in the first part were dates. The P was a participant's birth date.

In the second part the stimuli were city names. There were 3 Is (NT1, NT2, and NT3), 1 P (P2), and one T. The Is were irrelevant to the participant city names. The P was a participant's hometown name, and the T was a city with an assigned significance ("Chicago" was used as the T).

For ERP calculations we use a composite of all 4 Is combined in the first part of a trial (Iall1) and 3 Is combined (Iall2) in the second part of a trial.

There were 2 response boxes for recording behavioral data. A participant was instructed to randomly press 1 of 5 buttons on one response box to the first stimulus (P1 or I1, I2, I3, and I4). Then the participant pressed 1 of 2 buttons on another response box for the stimuli in the second part of a trial: the left button for P2 or any I from the second part and the right button for Ts.

Results

Individual analysis: Bootstrapped based hit rates at .9 confidence level was 12/13 (92%; for both (a) first part of a trial (dates) based on P1 versus Iall1 (all 4 Is from Part 1 combined) and (b) second part of a trial (city names), based on P2 versus Iall2 (all 3 Is from Part 2 combined). One of the participants did not show significantly bigger probe amplitude compared to Iall in the first part (dates), but the difference between P2 and Iall2 amplitudes was significant. Thus, a combined result from both parts of the trial is a "hit." A different participant showed nonsignificant difference between P2 and Iall2 amplitude (second part of a trial) but in the first part

"P1-Iall1" difference was significant, again making a combined result a "hit." Eventually, the "Double-Probe" approach leads to 100% detection.

Group analysis: The averaged P300 amplitudes (peak-to-peak, uV) were (a) first part of a trial: 9.108 (P1), 3.477 (Iall1); and 2) second part of a trial: 11.835 (P2), 7.173 (Iall2). The t test for the first part of a trial showed significant difference between P1 and Iall1 amplitudes, $t(12) = 5.472$, $p < .001$. In the second part of a trial, a difference between P2 and Iall2 amplitudes was also significant, $t(12) = 5.378$, $p < .001$.

Conclusions

The results demonstrate that our novel "Double-Probe Complex Trial Protocol" can be effectively used for assessment of memory deficit, malingering, and detection of deception. In situations when the difference between P300 probe and combined irrelevant amplitude is nonsignificant or marginal in one part of a trial, data from the second part can provide extra information for making a clinical or forensic decision. As a next step, further investigation of the new protocol is required, including running a control group.

REFERENCES

- Andreassi, J. L. (2000). *Psychophysiology: Human behavior and physiological response* (4th ed). Mahwah, NJ: Erlbaum.
- Kropotov, J. (2009). *Quantitative EEG, event-related potentials and neurotherapy*. New York: Elsevier.
- Labkovsky, E., & Rosenfeld, J. P. (2008). Detecting simulated amnesia for self-referred information with enhanced ERP(P300)-based protocol. *Archives of Clinical Neuropsychology*, 23, 664.
- Labkovsky, E. B., & Rosenfeld, J. P. (2009). P300-based protocol (with acoustic stimuli) for assessing memory deficit, malingering, and deception in clinical and forensic settings. *Journal of Neurotherapy*, 13, 274-275.
- Luck, S. J. (2005). *An introduction to the event-related potential technique*. Cambridge, MA: MIT Press.
- Polich, J. (1986). Normal variation of P300 from auditory stimuli. *Electroencephalography and Clinical*

Neurophysiology/ Evoked Potentials Section, 65, 236–240.

- Polich, J., & Kokb, A. (1995). Cognitive and biological determinants of P300: An integrative review. *Biological Psychology*, 41, 103–146.
- Rosenfeld, J. P., Labkovsky, E., Winograd, M., Lui, M., Vandenboom, C., & Chedid, E. (2008). The Complex Trial Protocol (CTP): A new, countermeasure-resistant, accurate P300-based method for detection of concealed information. *Psychophysiology*, 45, 906–919.
- Rosenfeld, J. P., Soskins, M., Bosh, G., & Ryan, A. (2004). Simple effective countermeasures to P300-based tests of detection of concealed information. *Psychophysiology*, 41, 205–219.
- Soskins, M., Rosenfeld, J. P., & Niendam, T. (2001). The case for peak-to-peak measurement of P300 recorded at .3 Hz high pass filter settings in detection of deception. *International Journal of Psychophysiology*, 40, 173–180.

Does Sexual Reassignment Surgery Rewire the Brain: A Comparison of Pre/Post qEEG Results

J J Miles, PhD, and Stuart Donaldson, PhD
University of Calgary
<jmilesenator@gmail.com>

As identified by the quantitative electroencephalogram (qEEG), statistically important brain wave patterns related to gender identity might be observed when a transgendered person has undergone sexual reassignment surgery (SRS). The observed differences in brain wave patterns may prove useful in treatment, and or assessment for readiness for postsurgery success for a transsexual person. Reassessment of brain wave patterns immediately after diagnosis, treatment, and surgery will perhaps reveal that the previous pattern was not altered and no longer statistically important. This pilot study might support the concept that brain wave patterns related to gender identity do have a correlated and measurable energetic effect. In addition, this study may objectively identify an immediate energetic change after HRT/ surgeries in the direction of normalcy and health. Results of the study, including serendipitous findings, will be available for discussion regarding these questions and many others.

Rationale for Pilot Study

The qEEG is very useful in revealing the underlying abnormal brainwave patterns associated with Attention Deficit Hyperactivity Disorder (ADHD) and many other disorders. The system can discriminate with more than 90% accuracy ADHD from learning difficulties and from normal. Many psychiatrists, pediatricians, and psychologists involved in the diagnosis of learning disorders and ADHD are unaware of a significant body of research that supports the use of topometric (visual) qEEG analysis as a diagnostic tool for differentiating between organic and functional brain disorders including learning difficulties, ADHD, schizophrenia, epilepsy, and cerebral atrophy associated with alcohol abuse, depression, and anxiety. Psychophysicologists have established normative qEEG databases. The differences in brain wave patterns revealed in these comparisons point to subtypes of ADHD that are not documented in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.). Studies of qEEG patterns of ADHD children and adults are consistent with findings revealed by Positron Emission Topography, functional Magnetic Resonance Imaging, Single Photon Emission Computed Tomography (SPECT), and other neuroimaging studies. More recently, research from SPECT brain studies by Daniel Amen and his colleagues have identified six subtypes of ADHD that correlate to qEEG patterns found in individuals with Attention Deficit Disorder (ADD). Although various psychometric measurements are available to clinicians, presently there are no data available that document the qEEG for use in assessments. Thus, topometric (or Visual) QEEG analysis is a powerful adjunct to psychometric assessment in this area (Duff, 2002).

qEEG

A qEEG is a topographic/visual enhancement of a traditional EEG. During the procedure, electrical activity of the brain, at rest and during stimulation, is recorded for

analysis. Each area of the brain normally spends a characteristic amount of time in alpha, beta, theta, and delta activity. By comparing a patient's brain mapping to a control population, it may be possible to localize areas of focal slowing and enhanced areas of electrical activity. qEEG is not an invasive procedure; it can be used on all age groups but requires the interpretation of a specialist trained in quantitative encephalographic analysis. Interpretation of the qEEG involves an assessment of the statistical degree of congruence or lack of congruence between a patient and the normal population, or the degree of similarity between a given patient and a qEEG profile that may be characteristic of some defined clinical group. The quantitative approach can display not only variations in the qEEG profiles but also progressive changes in neurophysiological function over time. qEEGs are presently utilized in the evaluation of (a) attention deficit disorder, (b) anxiety, (c) depression, (d) substance abuse disorders, (e) psychiatric disorders, and (f) closed head injuries (Donaldson, 2009).

The most common and well-documented use of neurofeedback is in the treatment of ADHD with multiple studies showing neurofeedback to be useful in the treatment of ADD. Other areas where neurofeedback has been researched include treatment of substance abuse, anxiety, depression, epilepsy, obsessive compulsive disorder, learning disabilities, bipolar disorder, conduct disorder, anger and rage, cognitive impairment, migraines, headaches, chronic pain, autism spectrum disorders, sleep, posttraumatic stress disorder, and mild traumatic brain injury (Donaldson, 2009). Hence, the effort to discover the usefulness of the qEEG as a tool for providing empirical data for assessment, treatment, and prediction of success for postsexual reassignment surgery in transsexuals would seem appropriate. Does sexual reassignment surgery create or cause new neural networks that literally change the brain and allow it to operate with greater efficiency, productivity, and functionality in the postsurgery transsexual compared to genetic men and women?

REFERENCES

- Cairns, S., Crozier, S., VanderWerf, J., Mottosky, R., & Miles, J. J. (2005, May 18). *Outcome measure: One year later*. Paper presented at the Alberta Services for Students Conference, Mount Royal College, Calgary, Alberta, Canada.
- Miles, J. J. (2004). "The New Sexual Performance Drugs", Alberta Services for Students Conference, Red Deer, Alberta, 12/05/2004, Refereed.
- Miles, J. J. (2005, June 1). *Time management for work and life balance*. Law Clerks' Forum: Advanced Litigation and Corporate Commercial Issues, Insight Information conference, Calgary, Alberta, Canada.
- Miles, J. J. (2006a, September). *Alcohol use amongst the transgendered community*. Paper presented at the BACCHUS Western Regional Conference, Olds College, Alberta, Canada.
- Miles, J. J. (2006b, May). *Working with clients with stuttering disorder*. Paper presented at the Alberta Services for Students Conference, Northern Lakes College, Alberta, Canada.
- Miles, J. J. (2008). *Therapy for sleep issues and the integration of a comprehensive holistic treatment model*. Paper presented at the International Conference on Integrative, Complimentary Alternative Medicine and Mental Health, Toronto, Ontario, Canada.
- Miles, J. J., & Donaldson, S. (2009). *The use of the qEEG in assessment of gender identity*. Paper presented at the World Professional Association for Transsexual Health, Oslo, Norway.
- Miles, J. J., & Shaw, M. (2009). *The use of a journal in a comprehensive treatment paradigm*. Paper presented at the World Professional Association for Transsexual Health, Oslo, Norway.

Tracking Emotion Dysregulation in Cocaine Addiction Using Event Related Theta and Alpha Oscillations

Muhammad Adeel Parvaz, MS, Thomas Maloney, PhD, Greg Hajcak, PhD, and Rita Z Goldstein, PhD

Stony Brook University
<mparvaz@gmail.com>

Introduction

Event related oscillations (EROs) are the brain rhythms evoked at the onset of an

external stimulus. They are isolated by decomposition of electroencephalogram (EEG) time series into weighted combinations of simultaneous sinusoidal functions that could be superimposed to reconstitute a given complex waveform. Of these oscillations, increase in poststimulus theta (3–7 Hz) band is associated with orientation, concentration of attention, and the processing of emotional information (e.g., International Affective Picture System [IAPS] pictures), whereas desynchronization in lower alpha band (7–10 Hz) is associated with external attention encompassing vigilance and expectancy. In the current study, our goal was to use EROs to study emotional processing in individuals with current cocaine use disorder (CUD), a psychopathology that has only recently been targeted for this purpose.

Methods

Evoked EEG synchronization and desynchronization in the lower and upper theta and alpha bands were quantified in 37 individuals with CUD, and 31 demographically matched healthy control participants while they passively viewed pleasant, unpleasant, neutral (IAPS), and cocaine-related pictures.

Results

Replicating previous results in controls, upper theta band showed increased synchronization when viewing pleasant as compared to unpleasant images (F3 electrode, 600–640 msec, $Z = 4.46$, $p = .014$), with a trend in the same direction in the lower theta band (F3 electrode, 780–880 msec, $Z = 3.81$, $p = .053$), whereas alpha rhythm showed higher desynchronization in response to all emotionally valenced versus neutral stimuli (CP3 electrode, 760–794 msec, $Z = 4.86$, $p < .001$). In CUD, however, theta band did not differentiate between the pleasant and unpleasant pictures ($p > .1$); instead it showed significantly higher synchronization

when viewing the drug-related as compared to neutral images (C3 electrode, 480–5203 msec, $Z = 4.54$, $p = .02$). Alpha rhythm showed the highest desynchronization in response to these drug cues, less so to the other emotional pictures (pleasant and unpleasant images) and the least to the neutral stimuli (cocaine > pleasant/unpleasant > neutral; PZ/P4, 875–1150 msec, $Z > 4.2$, $p < .001$).

Discussion

In healthy controls, we successfully replicated earlier studies by showing that the theta band indexes valence (separating pleasant from unpleasant pictures), whereas the alpha band tracks arousal (distinguishing neutral from all other emotionally valenced pictures). In CUD, however, both bands differentiated between the drug and the other pictures (theta: drug vs. neutral pictures; alpha: drug vs. the other emotionally valenced images). Therefore, both bands may be used to assess emotional dysregulation in drug addiction, quantifying attention-bias toward drug-related stimuli as postulated by the incentive-sensitization theory. The potential significance of results (especially the arousal/valence differences compared to healthy controls and practical clinical uses to enhance emotion regulation and decrease craving in CUD) remains to be explored.

REFERENCES

- Aftanas, L. I., Reva, N. V., Varlamov, A. A., Pavlov, S. V., & Makhnev, V. P. (2004). Analysis of evoked EEG synchronization and desynchronization in conditions of emotional activation in humans: Temporal and topographic characteristics. *Neuroscience and Behavioral Physiology*, *34*, 859–867.
- Moeller, S. J., Maloney, T., Parvaz, M. A., Dunning, J. P., Alia-Klein, N., Woicik, P. A., et al. (2009). Enhanced choice for viewing cocaine pictures in cocaine addiction. *Biological Psychiatry*, *66*, 169–176.

Efficacy of Neurofeedback Training in Children with High Functioning Autism Spectrum Disorders at the Level of Overt and Covert Behavior

Marjan Saeb, MA, and Reza Rostami, MD
University of Social Welfare and Rehabilitation
<marjan.saeb@gmail.com>

Aim

The aim of the present study was to investigate the efficacy of Neurofeedback (NF) training for alleviating the symptoms and improving the performance in children with high functioning Autism Spectrum Disorders (ASD) at two levels: overt behavior, and covert behavior.

Method

The study was conducted in a “single-subject” design with multiple baselines and follow-ups. Eight children with high-functioning ASD and ranging in age from 8 to 13 (M age = 10.12) participated in the study. Participants were divided into 3 groups differing with each other in time of onset of the training and thus the number of baseline records (3 records for the first group, 5 for the second, and 7 for the third one). All participants went through 30 sessions of Neurotherapy. Assessments were done at two levels of overt behavior (symptoms and performance) and covert behavior (brain functioning) during the baseline, training course, and follow-up. The acquired data at the level of overt behavior were analyzed using two main methods: visual analysis and effect size. At the level of covert behavior, the data were analyzed via visual analysis and paired t test. At the overt behavior level, repeated measurements were used during baseline, training course, and follow-up via two researcher-made scales called Symptom Assessment Scale and Performance Assessment Scale. Assessment at the level of covert behavior was done

through pre and post-Quantitative Electroencephalography for each participant.

Results

At the level of overt behavior, NF training resulted in symptom reduction in 75% of the recruited children suffering from ASD. Comparing the three core symptoms, NF was more efficacious in alleviating the symptoms related to communication. Besides, improvement was also recorded in the performance of 75% of the subjects following treatment. At the level of covert behavior, analysis of posttraining results revealed changes in brain functioning of 87% (7 of 8) of participants; the noticed changes were mostly (62%) in form of reduction within the range of Delta and High Beta.

Conclusions

Considering the following reasons, we can claim that the reduction of symptoms is resulted from the NF training: First, reduction of symptoms have taken place in the treatment phase not in the baseline; second, because 3 groups of participants entered the training phase at different times, groups that were still in the baseline could be regarded as a control for the participants in the treatment phase (the control group in baseline had no variance in severity of symptoms, whereas reduction trend of the group in the training phase was taking place); third, reduction in the severity of symptoms in ASD after NF was shown in the previous studies too. The same argument applies for the improvement in performance. At the covert behavior level, Reduction in the absolute powers of Delta and High Beta frequency bands is concordant with the treatment goals in other researches such as Jarusiewicz (2002). Considering all the acquired information, we claim that the use of NF training can lead to partial improvements at the level of overt and covert behavior in children with ASD. But this *does not* apply to every child with Autism, and in some cases it may cause some serious adverse effects.

REFERENCES

- Coben, R., & Padolsky, I. (2007). Assessment-guided neurofeedback for autistic spectrum disorder. *Journal of Neurotherapy*, *11*, 5–23.
- Jarusiewicz, B. (2002). Efficacy of neurofeedback for children in the autistic spectrum. *Journal of Neurotherapy*, *6*, 39–49.
- Koujzer, M., Moor, J., Gerrits, B., Congedo, M., & Schie, H. (2008). Neurofeedback improves executive functioning in children with Autism Spectrum Disorders. *Research in Autism Spectrum Disorders*, *3*, 145–162.
- Paoletti, J. L., & Kaiser, D. A. (2006, July). Neurotherapeutic assessment and training of an autistic individual. *What's New in Neurofeedback*, *9*. Retrieved from <http://start.eegspectrum.com/Newsletter/jul2006.htm>
- Sichel, A., Fehmi, L., & Goldestein, D. (1995). Positive outcome with neurofeedback treatment in a case of mild autism. *Journal of Neurotherapy*, *1*, 60–64.

Neurofeedback Training to Improve Attention and Control Alertness in ADHD

Estato Sokhadze, PhD, Guela Sokhadze, and Lonnie Sears, PhD
University of Louisville
<tato.sokhadze@louisville.edu>

Introduction

Neurofeedback-base treatment of ADHD has received substantial empirical support in recent years (Arns et al., 2009). Neurofeedback effects were manifested not only in clinical improvements but also in normalizations of qEEG patterns (Gevensleben et al., 2009) and ERP (Kropotov et al., 2005). Positive effects of neurofeedback in ADHD were achieved with several different protocols (e.g., SMR/theta/beta, slow beta/theta, slow cortical potential, etc.); however, in most studies it was outlined that number and lengths of neurofeedback sessions, location of EEG electrodes, and motivation of participants are important factors determining success of treatment in children with ADHD. In this exploratory study we investigated effects of 12 sessions of prefrontal neurofeedback on behavioral

performance in IVA + Plus test in 8 patients with ADD/ADHD. One of the aims was to investigate whether using EEG measures to control size and brightness of a DVD is effective for maintaining motivational engagement of children with ADHD during training.

Method

All 8 patients with ADHD diagnosis were evaluated and referred to the lab from the Weisskopf Child Evaluation Center. Neurofeedback training was conducted on weekly basis with 30-min-long sessions using 12 different fragments of documentary films depicting nature scenes (BBC *Planet Earth* series). EEG was recorded from the prefrontal site (FPz) referenced to the left earlobe. To enhance focus, participants were trained to suppress wide band spectrum, whereas alertness parameter was a wideband measure of the upward shift of the frequencies in the EEG. Visual feedback was arranged in a form of control of brightness, size, and continuation of the documentary by the “focus and alertness” measures. Auditory feedback was used to inform the participant when these two measures were under the threshold level. The IVA+Plus test was administered before and after neurofeedback course in each participant. Another clinical behavioral outcome included measures from the Aberrant Behavior Checklist (ABC). The ABC (Aman & Singh, 1994) is a clinician-administered rating scale assessing five problem areas: Irritability, Lethargy/Social Withdrawal, Stereotypy, Hyperactivity, and Inappropriate Speech, and is based on caregiver reports.

Results and Conclusion

Neurofeedback training aimed at enhancement of focus and alertness measures was accompanied by improved performance on IVA+Plus test and lowered Hyperactivity and Irritability scores of the ABC. Self-regulation of prefrontal EEG measures of focus and alertness using

protocol with DVD-control as a visual feedback was effective in maintaining interest and motivational engagement of children with ADHD. Twelve 30-min-long sessions of neurofeedback were sufficient to achieve ability to control EEG parameters of interest in most of ADHD participants.

REFERENCES

- Aman, M. G., & Singh, N. N. (1994). *Aberrant Behavior Checklist-Community. Supplementary manual*. East Aurora, NY: Slosson Educational.
- Arns, M., de Ridder, S., Strehl, U., Breteler, M., & Coenen, A. (2009). Efficacy of neurofeedback treatment in ADHD: The effects on inattention, impulsivity and hyperactivity: A meta-analysis. *Clinical EEG Neuroscience*, *40*, 180–189.
- Gevensleben, H., Holl, B., Albrecht, B., et al. (2009). Distinct EEG effects related to neurofeedback training in children with ADHD. *International Journal of Psychophysiology*, *74*, 149–157.
- Kropotov, J. D., Grin-Yatsenko, V. A., Ponomarev, V. A., et al. (2005). ERPs correlates of EEG relative beta training in ADHD children. *International Journal of Psychophysiology*, *55*(1), 23–34.

Understanding Infra-Low Frequency Neurofeedback

Lincoln Stoller, PhD
Tenger Research, LLC
<ls@tengerresearch.com>

Infra-low Frequency (ILF) neurofeedback training is loosely defined as training EEG signals below 0.5 Hz. This is generally well understood in the context of slow cortical potentials (SCP) but poorly understood in cases where trainees claim to discriminate between harmonics differing by hundredths of a Hertz in the 0.1 Hz EEG range (Othmer, 2010). I provide a theoretical analysis of signals of this frequency to explain what is happening when we train these EEG components. The short-time averaging used for training does not violate the calculation of the EEG components over a longer time epoch. The electrical responses provided by the trainee to low frequencies and the DC signal are qualitatively different. ILF

training can affect both the SCP and long wavelength cortical activity.

REFERENCES

- Collura, T. F. (1995). Basic electronics and physics. In K. Levin & H. Luders (Eds.), *Comprehensive clinical neurophysiology*, (pp. 1–10). New York: Elsevier.
- Collura, T. F. (2009, January). Practicing with multi-channel EEG, DC, and slow cortical potentials. *NeuroConnections*, *34*.
- Othmer, S., & Othmer, S. (2010, spring). Introduction to infra-low frequency training. *NeuroConnections*, *14*.
- Strehl, U. (2009). Slow cortical potentials neurofeedback. *Journal of Neurotherapy*, *13*, 117.

The Effect of Different Photic Entrainment Frequencies used During Live Z Score Neurofeedback: A Preliminary Examination

Jeff Tarrant, PhD
Spring Grove Counseling
<Dr.Tarrant@hotmail.com>

Introduction

Many neurofeedback practitioners utilize a range of technology-based therapies in their practice. One technique commonly used is audiovisual entrainment. This strategy utilizes light and/or sound stimulation at specific frequencies to impact brain wave functioning. Demos (2005) reviewed the use of Audio Visual Entrainment for relaxation, pain management, and “brain brightening” as well as specific applications including ADHD and depression. AVE is also used to help a client learn what it “feels like” to produce a desired brainwave state (Siever, 2009). It can be used as a stand-alone treatment or in conjunction with neurofeedback training. Clinicians have used the visual feedback with view holes in the eyeset during a neurofeedback session to encourage a specific response and facilitate the neurofeedback process. Although most people will entrain between 5 and 15 Hz, it has been noted that some people have differing responses to the stimulation (Siever, 2000).

The immediate impact of AVE and photic stimulation has become even more noticeable with the recent advent of live z score neurofeedback training, which allows the therapist to observe client brainwave activity in comparison to a normative database in “real time” while neurofeedback is occurring. The current study explores trends and response patterns when using specific frequencies or immersive photic stimulation during live z score neurofeedback training.

Method

Fifteen child and adolescent clients in the author’s neurofeedback practice participated in the study. Each client received 4 channel live z score training at placements specific to their symptoms and pretreatment QEEG. During 5 consecutive sessions, the first 5 min of the session was recorded with neurofeedback only. The second 5 min included the same neurofeedback protocol along with one of five visual stimulation conditions (no AVE, 10 hz, 14 hz, 18 hz, immersive) using eyesets with view holes. The order of exposure was counterbalanced between clients. The data from each of the 5-min segments was analyzed through the NeuroGuide Normative Database (Thatcher, 1987). The BrainMaster Atlantis 4×4 was used along with the Applied Neuroscience, Inc. Z DLL software for all neurofeedback sessions. The DAVID Paradise XL+ with white light True-Vu Omniscreen Eyesets was used to administer all AVE sessions. Immersive photic stimulation utilized the BrainMaster Atlantis immersive glasses.

Results

Absolute and Relative Power z scores were obtained for each of the 5-min segments for all 4 channels of each participant using the 8 standard bands in NeuroGuide Deluxe 2.5.7 (delta, theta, alpha, beta, high beta, beta1, beta2, beta3). These data were transformed into change scores by subtracting the z score obtained from the first 5-min segment (control) of each session from the second (experimental) segment. Each

electrode for each client was considered a separate “subject” resulting in 60 “subjects” (15 clients \times 4 electrodes each).

Within-subjects analyses of variance (ANOVAs) are conducted for each of the 8 NeuroGuide bands. The Dependent Variable is the treatment condition, and the Independent Variable is the change scores for each frequency band. These analyses determine if any of the experimental conditions (10 hz, 14 hz, 18 hz, immersive) demonstrate significantly more change in EEG bands than a standard live z score neurofeedback treatment. Positive ANOVA findings are followed by post hoc analysis to determine which independent variables are responsible for the significance.

Conclusions

The results are considered and discussed in relation to the possible mechanisms of audiovisual entrainment. For example, if different patterns of response are discovered, can this be attributed to different mechanisms of action based on individual differences of the clients. Is there a way to determine what types of photic stimulation may be most beneficial for specific brainwave patterns? What are the implications for using audio visual entrainment simultaneously with neurofeedback? What directions does this provide for future research in this area?

REFERENCES

- Demos, J. (2005). *Getting started with neurofeedback*. New York: Norton & Co.
- Siever, D. (2000). *The rediscovery of audio-visual entrainment technology*. Edmonton, Canada: Comprotronic Devices Limited.
- Siever, D. (2009). Audio-visual entrainment: History, physiology, and clinical studies. In J. R. Evans (Ed.), *Handbook of neurofeedback: Dynamics and clinical applications* (pp. 155–183). New York: Informa Healthcare.
- Thatcher, R. W. (1987). Federal copyright (TXu-347-139) of the “Life span EEG Normative Database” and all of its derivatives, transformations, and revisions.

Post Hoc Analysis of the QEEG Reveals the Most Desirable Treatment Protocols and Identifies Clients Vulnerable to Overtraining

Thomas Matthews, PhD

Optimum Performance Solutions

<thinkwell@optimumperformance.info>

Introduction

This neurofeedback case series demonstrates an approach to post hoc scoring of the qEEG to develop neurofeedback treatment plans. The findings illustrate that otherwise nonobvious treatment protocols are empirically identified and lead to substantial clinical improvement in targeted issues. The further evidence that clients can be vulnerable to overtraining offers support of previous findings.

Method

Participants: All 30+ neurofeedback cases in a private practice clinic since initiation of the assessment procedure were asked to participate, representing more than 1,000 treatment sessions. Those who provided informed consent were included in a retrospective review of assessment and treatment outcome. Most clients were adults with history of brain injury, who function across a range from disabled to high-functioning professionals. Age range was 9 to 68.

Clinical Records Data Pool: The Clinical Records comprised assessment and treatment records. Assessment records consisted of (a) screening review of function, medical history, and prior assessments to develop localization hypotheses in a formal report; (b) quantitative assessment including qEEG; and (c) post hoc scoring of qEEG output.

Treatment records consisted of the detailed protocol sheet and chart note for every session. Treatment itself was EEG operant conditioning using any protocol that appeared to be desirable for that client based on assessment and treatment response, in the context of a multimodal approach. The clinician provided (a) conventional “beta”/“SMR” neurofeedback training along the

sensory motor strip, or (b) single channel increase or decrease training at any indicated “10–20 system” location, or (c) 4-channel simultaneous z score neurofeedback. Sessions were 1 hr, one or two per week (occasionally less often), and the total number of sessions was determined by consideration of the resources available and attaining client goals.

The multimodal approach required occasional counseling sessions devoted to progress review and to functional issues raised by the client, family or the case-management team. Many of the clients had serious brain-based disabilities and adequate financial resources, so treatment was rather extensive in those cases. Several clients started neurofeedback before the case series began and were included in the study when their treatment planning included the post hoc qEEG scoring procedure. Not all clients completed treatment during the study time frame. Most had exhausted other treatment options before seeking neurofeedback, but some were referred relatively soon after serious head trauma and consequently had concurrent conventional treatments such as OT, PT, or speech therapy; many had psychotropic and other medications.

Data sample procedure: Data were compiled from the treatment charts by an undergraduate intern supervised by biology faculty of a local university, scored, and summary statistics conducted. Treatment success was measured by scoring client and third-party reports of progress that had been written by the clinician in the chart notes during the course of treatment, and in a few instances where available by repeat quantitative assessment. Particular attention was given to examples where the planning tool indicated otherwise unexpected protocols for 4-site simultaneous z score training. Vulnerability to overtraining was measured by scoring the chart record for (a) shifts in treatment protocol toward reduced training time in each session, or (b) shifts toward less-fatiguing protocols, as opposed to completing the expected number of sessions with a protocol and moving on, or (c) frank overtraining episodes.

The post hoc qEEG scoring procedure evolved over the period of the study. Consequently some refinements were available only in the final version of the treatment planning tool. To conduct the study, the qEEG data for all participants was reevaluated employing the final version of the planning tool. It was hypothesized that high SD(D) scores for a given protocol (i.e., standard deviation of the deviance scores computed by the planning tool) would predict vulnerability to overtraining for that client if using that protocol. Furthermore it was expected that vulnerability would be modulated by (a) severity of TBI, (b) hypoglycemia or hyperglycemia, and possibly by exceptionally high general intelligence (see Matthews, 2007). The clinician also evolved in his capacity to use the information. For clients later in the case series, the clinician reserved such very high-SD(D) protocols until the client demonstrated adequate stabilization in response to other neurofeedback.

Results

The findings illustrate that otherwise non-obvious treatment protocols are empirically identified by the post hoc scoring of the qEEG, and lead to substantial clinical improvement in targeted issues. Examples are reviewed to provide a clinical appreciation of how the procedure incrementally improved planning, treatment, and outcome.

The further evidence that clients can be vulnerable to overtraining offers support of previous findings.

Conclusion

Otherwise nonobvious treatment protocols can be empirically identified, which lead to substantial clinical improvement in targeted issues. The clients vulnerable to overtraining can be identified and treatment tailored to their needs, offering support of previous findings. Neurofeedback is a specific and potent intervention to which there can be adverse response (Hammond et al., 2001; Hammond & Kirk, 2008; Lubar et al., 1981; Ochs, 2007; Whitsett et al., 1982). Matthews (2007) offered the hypothesis that clients vulnerable to overtraining may be identified and the training tailored to their needs. Supporting examples are discussed to clarify this relationship.

The author/clinician is Clinical Director of Optimum Performance Solutions LLC in Topeka, Kansas, which in addition to the clinic does business as AcuityZ. AcuityZ makes available the post hoc qEEG scoring procedure used in this study. Alternative treatment-planning procedures include the symptom-to-location tool included in the training module of NeuroGuide[®] offered by Applied Neurosciences Inc., the BMANS Manual Series by Brownback Mason Associates, the CN 1020 from EEG Professionals, and the Mini-Q by NewMind Apps.