ISNR 20th Annual Conference

September 19-23, 2012

Pre Conference Workshops September 17-19

Schedule and Abstracts
### Pre-Conference Workshops Schedule (Monday-Wednesday)

#### Monday September 17, 2012

<table>
<thead>
<tr>
<th>Time</th>
<th>Pre-Conference Workshop 1.1 (Day 1) LENS Foundation Training (3 day workshop) – Michael Beasley &amp; Len Ochs</th>
</tr>
</thead>
<tbody>
<tr>
<td>10:00 AM-1:00 PM &amp; 2:30-6:00 PM</td>
<td>Pre-Conference Workshop 1.1 (Day 1) LENS Foundation Training (3 day workshop) – Michael Beasley &amp; Len Ochs</td>
</tr>
<tr>
<td>10:00 AM-1:00 PM &amp; 2:30-6:00 PM</td>
<td>Pre-Conference Workshop 2.1 (Day 1) Advanced LENS Training (3 day workshop) – Len Ochs &amp; Cathy Wills</td>
</tr>
</tbody>
</table>

#### Tuesday September 18, 2012

<table>
<thead>
<tr>
<th>Time</th>
<th>Pre-Conference Workshop 1.2 (Day 2) LENS Foundation Training (3 day workshop) – Michael Beasley &amp; Len Ochs</th>
</tr>
</thead>
<tbody>
<tr>
<td>10:00 AM-1:00 PM &amp; 2:30-6:00 PM</td>
<td>Pre-Conference Workshop 2.2 (Day 2) Advanced LENS Training (3 day workshop) – Len Ochs &amp; Cathy Wills</td>
</tr>
<tr>
<td>8:00 AM-12:15 PM &amp; 1:15-5:30 PM</td>
<td>Pre-Conference Workshop 3.1 (Day 1) Introduction to the Practice of Neurofeedback: Assessment leads to Appropriate Intervention (2 day workshop) – Lynda Thompson &amp; Michael Thompson</td>
</tr>
<tr>
<td>1:15-5:30 PM</td>
<td>Pre-Conference Workshop 8 – SPONSORED WORKSHOP* Integrating Norms-Based Neurofeedback within a Holistic Psychophysiological Treatment Approach – Linda Walker</td>
</tr>
</tbody>
</table>

#### Wednesday September 19, 2012

<table>
<thead>
<tr>
<th>Time</th>
<th>Pre-Conference Workshop 1.3 (Day 3) LENS Foundation Training (3 day workshop) – Michael Beasley &amp; Len Ochs</th>
</tr>
</thead>
<tbody>
<tr>
<td>10:00 AM-1:00 PM &amp; 2:30-6:00 PM</td>
<td>Pre-Conference Workshop 2.3 (Day 3) Advanced LENS Training (3 day workshop) – Len Ochs &amp; Cathy Wills</td>
</tr>
<tr>
<td>8:00 AM-12:15 PM &amp; 1:15-5:30 PM</td>
<td>Pre-Conference Workshop 3.2 (Day 2) Introduction to the Practice of Neurofeedback: Assessment leads to Appropriate Intervention (2 day workshop) – Lynda Thompson &amp; Michael Thompson</td>
</tr>
<tr>
<td>8:00 AM-12:15 PM &amp; 1:15-5:30 PM</td>
<td>Pre-Conference Workshop 4 - Electroencephalographic Abnormalities in EEG/QEEG: Subclinical Findings Redefined Treatment Implications and Considerations- Ron Swatzyna &amp; Jay Gunkelman</td>
</tr>
<tr>
<td>8:00 AM-12:15 PM &amp; 1:15-5:30 PM</td>
<td>Pre-Conference Workshop 5 - Enhancing Neurotherapy by Activating the Brainstem-cerebellum-midbrain-cortex Pathways Through Primary-Reflex Movements - Practical Workshop – Suzanne Day</td>
</tr>
<tr>
<td>8:00 AM-12:15 PM &amp; 1:15-5:30 PM</td>
<td>Pre-Conference Workshop 6 - Biological Markers in Neurology and Psychiatry: Guidelines for Applying Event Related Potentials for Diagnosis and Treatment - Juri Kropotov</td>
</tr>
<tr>
<td>8:00 AM-12:15 PM &amp; 1:15-5:30 PM</td>
<td>Pre-Conference Workshop 7 – SPONSORED WORKSHOP* QEEG and Neurofeedback using the BrainAvatar, Live Zscores, Live sLORETA Projection (LLP) and Combined Protocols- Tom Collura, Mark Smith &amp; Penijeane Rutter</td>
</tr>
</tbody>
</table>

*ISNR does not offer CEUs for this workshop. CEUs may be offered by the Sponsor directly.*
Program Schedule (Wednesday)

<table>
<thead>
<tr>
<th>Time</th>
<th>Wednesday September 19, 2012 Conference Schedule</th>
</tr>
</thead>
<tbody>
<tr>
<td>8:00 AM-12:15 PM &amp; 1:15-5:30 PM</td>
<td>Pre-Conference 8 Hour Workshops - See Workshop Schedule</td>
</tr>
<tr>
<td>8:00 AM-4:00 PM</td>
<td>ISNR Golf Tournament (Off Site Golf course, Scramble format). Pre-registration required, please contact the ISNR office to register. A portion of proceeds go to the ISNR Research Foundation. Prizes awarded at banquet dinner. *Tee times will be arranged by the golf event coordinator after registration.</td>
</tr>
<tr>
<td>2:00-7:30 PM</td>
<td>Vendor Setup in Vendor Area</td>
</tr>
<tr>
<td>5:30-7:30 PM</td>
<td>ISNR Board of Directors Meeting</td>
</tr>
<tr>
<td>7:30-9:30 PM</td>
<td>Outdoor Welcome Dessert Reception (Inclement Weather provision in Vendor Area)</td>
</tr>
</tbody>
</table>
### Program Schedule (Thursday)

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
</tr>
</thead>
<tbody>
<tr>
<td>8:00-8:15 AM</td>
<td><strong>President’s Welcome</strong> – Richard E. Davis, MS</td>
</tr>
<tr>
<td>8:15-8:45 AM</td>
<td><strong>Potential Neurofeedback Side Effects, Adverse Reactions &amp; Recommendations for Liability Protection</strong>; Cory Hammond</td>
</tr>
<tr>
<td>8:45-9:15 AM</td>
<td><strong>Enhancing Neurotherapy by Means of Brainstem Activation Through Primary-reflex Rhythmic Movements</strong>; Suzanne Day</td>
</tr>
<tr>
<td>9:15-9:25 AM</td>
<td>Break</td>
</tr>
<tr>
<td>9:25-9:40 AM</td>
<td><strong>Student Presentation</strong> - Single Trial Time-Frequency Domain Analysis of Error Processing in Post-Traumatic Stress Disorder; Zachary Clemans, Ayman El-Baz, Christopher Stewart &amp; Estate Sokhadze</td>
</tr>
<tr>
<td>9:40-10:10 AM</td>
<td><strong>Investigation of Theta-Beta Neurofeedback for Adult ADHD; Session Data</strong>; Sarah Wyckoff</td>
</tr>
<tr>
<td>10:10-10:40 AM</td>
<td><strong>Training Performance and Effects of Slow Cortical Potential Neurofeedback for Adult Attention Deficit/Hyperactivity Disorder</strong>; Kerstin Mayer, Sarah Wyckoff &amp; Ute Strehl</td>
</tr>
<tr>
<td>10:40-10:55 AM</td>
<td>Break</td>
</tr>
<tr>
<td>10:55-11:45 AM</td>
<td><strong>Invited Speaker</strong> - C. Shawn Green, PhD; Video Games, Learning to Learn and Brain Plasticity</td>
</tr>
<tr>
<td>11:45-11:55 AM</td>
<td>Break</td>
</tr>
<tr>
<td>11:55-12:55 PM</td>
<td><strong>KEYNOTE Speaker</strong> - Mark Jensen, PhD; Effects of Non-pharmacological Pain Treatment on Brain States</td>
</tr>
<tr>
<td>12:55-1:30 PM</td>
<td>Lunch Available for Purchase</td>
</tr>
<tr>
<td>12:55-8:30 PM</td>
<td>Break - Visit our Vendor Area</td>
</tr>
<tr>
<td>1:30-2:30 PM</td>
<td><strong>Small Group Discussion</strong> – Exercise and it's effects on the brain/complimentary to NFB training; Dan Williams</td>
</tr>
<tr>
<td>2:45-6:00 PM</td>
<td><strong>Workshops</strong> - See Workshop Schedule</td>
</tr>
<tr>
<td>6:30-8:30 PM</td>
<td><strong>Poster Session &amp; Cocktail Reception</strong> - See Program for Poster Listings</td>
</tr>
<tr>
<td>8:30-10:00 PM</td>
<td>By invitation only - <strong>International Attendee Reception</strong> (hosted by ISNR President)</td>
</tr>
</tbody>
</table>
# Program Schedule (Friday)

<table>
<thead>
<tr>
<th>Time</th>
<th>Friday September 21, 2012 Conference Schedule</th>
</tr>
</thead>
<tbody>
<tr>
<td>8:00-8:30 AM</td>
<td>The Enhancement of Neurofeedback with a Low Cost and Easy-To-Use NeuroSky EEG Biofeedback Training Device: The MindReflector Protocols; Thomas Fink</td>
</tr>
<tr>
<td></td>
<td>Crossing The Bar: Neurofeedback as an Adjunct Therapy to Addiction Recovery; Judith Miller</td>
</tr>
<tr>
<td>8:30-9:00 AM</td>
<td>Functional Disconnections in Trauma and Abuse: From Victimized Children to Murderers on Death Row; David Kaiser</td>
</tr>
<tr>
<td>9:00-9:10 AM</td>
<td>Break</td>
</tr>
<tr>
<td>9:10-10:00 AM</td>
<td>Invited Speaker- Christine Moravec, PhD; Self-Regulation in the Treatment of Chronic Heart Failure</td>
</tr>
<tr>
<td>10:00-10:15 AM</td>
<td>Student Presentation- Investigation of Unspecific Placebo Effects in Slow Cortical Potential Neurofeedback for Adult Attention Deficit/Hyperactivity Disorder (AD/HD): Kerstin Mayer, Sarah Wyckoff &amp; Ute Strehl</td>
</tr>
<tr>
<td></td>
<td>Student Presentation- An Event-Related Potential Study of Visual Spatial Attention Deficits in Autism; Guela Sokhadze, Lonnie Sears, Ayman El-Baz, Estate Sokhadze &amp; Manuel Casanova</td>
</tr>
<tr>
<td>10:15-10:25 AM</td>
<td>Break</td>
</tr>
<tr>
<td>10:25-11:25 AM</td>
<td>Keynote Speaker- Erik Peper, PhD; An Evolutionary Approach to Return to Health</td>
</tr>
<tr>
<td>11:25-11:35 AM</td>
<td>Break</td>
</tr>
<tr>
<td>11:35-12:35 PM</td>
<td>Keynote Speaker- Israel Liberzon, PhD; Functional Neuroanatomy of Emotions and Stress</td>
</tr>
<tr>
<td>12:35-1:15 PM</td>
<td>Lunch Available for Purchase</td>
</tr>
<tr>
<td>12:35-5:00 PM</td>
<td>Break - Visit our Vendor Area</td>
</tr>
<tr>
<td>1:00-2:00 PM</td>
<td>Small Group Discussion – Annual ISNR Research Foundation Update – David Trudeau &amp; Cynthia Kerson</td>
</tr>
<tr>
<td></td>
<td>Small Group Discussion- Call for Professionals - helping students and mentoring-Sarah Wyckoff and Judy Crawford</td>
</tr>
<tr>
<td></td>
<td>Small Group Discussion – Updates in Autism- Rob Coben</td>
</tr>
<tr>
<td></td>
<td>Small Group Discussion – The impact of medications on neurofeedback – Mike Cohen</td>
</tr>
<tr>
<td>2:15-5:30 PM</td>
<td>Workshops - See Workshop Schedule</td>
</tr>
<tr>
<td>2:15-5:15 PM</td>
<td>BCIA Exams - Must be pre-registered with BCIA to sit for exam</td>
</tr>
<tr>
<td>5:30-6:30 PM</td>
<td>ISNR Committees meet independently (Scheduled by Committee Chairs)</td>
</tr>
</tbody>
</table>
| 6:30-9:00 PM    | Featured Speaker Experience, Reception and Book Signing. Limited space with required pre-purchased registration. This function has an additional charge of $20 (purchase in the ISNR online store) and tickets are required for entry. Featuring Steven Kotler  
<p>| 6:00-8:00 PM    | Wine and Cheese Reception hosted by Nexalin Technology                                                          |
| 7:00-9:00 PM    | Sponsored Reception hosted by BrainMaster, Inc                                                                  |
| 9:00-11:30 PM   | By invitation only- Student Reception hosted by the ISNR Student Committee                                      |</p>
<table>
<thead>
<tr>
<th>Time</th>
<th>Saturday September 22, 2012 Conference Schedule</th>
</tr>
</thead>
<tbody>
<tr>
<td>8:00-9:00 AM</td>
<td>Comparison of the Effectiveness of Z-Score Surface/LORETA 19-Electrode Neurofeedback to Standard 1-Electrode Neurofeedback; Lucas Koberda, Andrew Moses, Paula Koberda &amp; Laura Koberda</td>
</tr>
<tr>
<td></td>
<td>Heart - Brain Connections: Neuroanatomy Underlies the Effectiveness of Interventions that Combine Neurofeedback with Biofeedback; Lynda Thompson &amp; Michael Thompson</td>
</tr>
<tr>
<td>9:00-9:10 AM</td>
<td>Break</td>
</tr>
<tr>
<td>9:10-9:40 AM</td>
<td>Combining Neuroeconomics with LORETA Biofeedback to Improve Self-Control and Promote Health Behavior; Jordon Silberman</td>
</tr>
<tr>
<td></td>
<td>Comparing the Effects of Neurofeedback and Hyperbaric Oxygen Therapy in Autism Spectrum Disorder: A Case Series; Robert Coben, &amp; Patrick Elliott</td>
</tr>
<tr>
<td>9:40-10:10 AM</td>
<td>Self Regulation of Slow Cortical Potentials in Patients with Intractable Epilepsy - Eight Years After; Ute Strehl, Sarah Birkle, Boris Kotchoubey</td>
</tr>
<tr>
<td>10:10-10:20 AM</td>
<td>Break</td>
</tr>
<tr>
<td>10:20-10:50 AM</td>
<td>An EEG Interface for Continuous Performance Testing and Event-Related Potentials; Andrew Greenberg, Chris Cholder &amp; Tom Collura</td>
</tr>
<tr>
<td></td>
<td>In Search of Depression; Kelly Callaway, Rex Cannon, Kenneth Phillips, Gregory Stuart, Deborah Baldwin &amp; Deborah Welsh</td>
</tr>
<tr>
<td>10:50-11:20 AM</td>
<td>Randomized, Controlled Cross-Over Research of Performance Brain Training™ Effects in Elite College Golfers; Noel Larson, Leslie Sherlin, Ashley Baker, &amp; Jeff Trosch</td>
</tr>
<tr>
<td>11:20-11:30 AM</td>
<td>Break</td>
</tr>
<tr>
<td>11:30-12:20 PM</td>
<td>Invited Speaker, David Cantor, PhD; Neurotoxins: Effects on Brain &amp; Behavior &amp; Therapy</td>
</tr>
<tr>
<td>12:20-12:30 PM</td>
<td>Break</td>
</tr>
<tr>
<td>12:30-1:30 PM</td>
<td>Keynote Speaker- Mario Beauregard, PhD; Neurofeedback Training Induces Changes in Grey and White Matter</td>
</tr>
<tr>
<td>1:30-2:00 PM</td>
<td>Lunch Available for Purchase</td>
</tr>
<tr>
<td>1:30-5:00 PM</td>
<td>Break- Visit the Vendor Area</td>
</tr>
<tr>
<td>2:00-3:00 PM</td>
<td>Small Group Discussion – BCIA Recertification &amp; Mentoring – Judy Crawford</td>
</tr>
<tr>
<td></td>
<td>Small Group Discussion- QEEG Application in Forensics and findings in Murderers- Jim Evans</td>
</tr>
<tr>
<td></td>
<td>Small Group Discussion – High Performance Training with Athletes- Leslie Sherlin</td>
</tr>
<tr>
<td></td>
<td>Small Group Discussion – Cognitive decline in the elderly – Helen Budzynski &amp; Jean Tang</td>
</tr>
<tr>
<td>3:15-6:30 PM</td>
<td>Workshops - See Workshop Schedule</td>
</tr>
<tr>
<td>7:00-7:30 PM</td>
<td>Members’ Meeting</td>
</tr>
<tr>
<td>7:30-8:45 PM</td>
<td>Banquet Dinner &amp; Recognitions</td>
</tr>
<tr>
<td>8:45-11:00 PM</td>
<td>After Dinner Entertainment</td>
</tr>
<tr>
<td>Time</td>
<td>Sunday September 23, 2012 Conference Schedule</td>
</tr>
<tr>
<td>------------------</td>
<td>-------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>8:00-8:30 AM</td>
<td>Real-time Functional Magnetic Resonance Imaging Neurofeedback to Attain Volitional Control Over Brain Activity and Associated Mental Functions: A Systematic Review; Gunther Meinlschmidt, Seung-Schik Yoo, &amp; Marion Tegethoff</td>
</tr>
<tr>
<td></td>
<td>On the Relation Between $\alpha$ and $\Theta$ in Specific Parieto-frontal Networks in Adult Attention Deficit/Hyperactivity Disorder (ADHD); Rex Cannon, Deborah Baldwin, Cynthia Kerson, Tiffany Shaw, Dominic Diloreto, Sherman Phillips &amp; Coleman Garner</td>
</tr>
<tr>
<td>8:30-9:00 AM</td>
<td>Multi-Modal Treatment of Stuttering: A Case Study Showing Neurofeedback Coupled with Traditional Speech Therapy; Becky Bingham</td>
</tr>
<tr>
<td>9:00-9:05 AM</td>
<td>Break</td>
</tr>
<tr>
<td>9:05-9:35 AM</td>
<td>The Impact of an Eight Week Heart Rate Variability Biofeedback (HRV) Training on Quantitative EEG and LORETA Following a Cognitive Stressor; Jeffrey Tarrant, Heather Eastman-Mueller, Ae Kyung Jung, Laura Sinquefield, Brett Woods &amp; Chad Cross</td>
</tr>
<tr>
<td></td>
<td>60 Minutes on the LENS Effects; Len Ochs</td>
</tr>
<tr>
<td>9:35-10:05 AM</td>
<td>In Pursuit of Happiness; Sarah Fischer &amp; Rex Cannon</td>
</tr>
<tr>
<td>10:05-10:15 AM</td>
<td>Break</td>
</tr>
<tr>
<td>10:15-10:30 AM</td>
<td>Student Presentation- Theta-Beta Neurofeedback for Adult ADHD: EEG and Behavioral Changes; Sarah Wyckoff, Kerstin Mayer, &amp; Ute Strehl</td>
</tr>
<tr>
<td>10:30-11:00 AM</td>
<td>Combined Neuromodulation Method Aimed to Improve Frontal Functions in Autism; Estate Sokhadze, Ayman El-Baz, Allan Tasman, Lonnie Sears &amp; Manuel Casanova</td>
</tr>
<tr>
<td></td>
<td>Role of QEEG Guided Neurofeedback in the Overall Treatment of Fetal Alcohol Spectrum Disorder (FASD); Ajeet Charate &amp; James Kowal</td>
</tr>
<tr>
<td>11:00-11:30 AM</td>
<td>Neurofeedback Protocol for the Treatment of Phonetic and Expressive Speech Impediments: Report of Two Cases; Jorge Palacios</td>
</tr>
<tr>
<td>11:30-11:45 AM</td>
<td>Closing Remarks - Incoming President – Randall Lyle, PhD</td>
</tr>
<tr>
<td>9:00 AM-2:00 PM</td>
<td>Last Chance to Visit the Vendor Area *Note Closing hour</td>
</tr>
<tr>
<td>12:00-6:00 PM</td>
<td>Vendor Seminar/Workshops - See Vendor Seminar Schedule for exact meeting times and location</td>
</tr>
<tr>
<td>12:00-3:00 PM</td>
<td>ISNR Board Meeting</td>
</tr>
</tbody>
</table>
## Workshop Schedule (Thursday - Saturday)

<table>
<thead>
<tr>
<th>Time</th>
<th>Thursday September 20, 2012 Workshop Schedule</th>
</tr>
</thead>
<tbody>
<tr>
<td>2:15-5:30 PM</td>
<td>Workshop 1 - Fundamentals in Research Methodology: An ISNR Research Foundation Workshop – David Trudeau, Estate Sokhadze, &amp; Rex Cannon</td>
</tr>
<tr>
<td></td>
<td>Workshop 2 - Autism Spectrum Disorders: Integrating Clinical Knowledge and Individual Symptoms and Neurophysiology in the Formation of Neurofeedback Treatment Plans – Robert Coben</td>
</tr>
<tr>
<td></td>
<td>Workshop 3 - Video Games As Exceptional Learning Environments- C. Shawn Green</td>
</tr>
<tr>
<td></td>
<td>Workshop 4 - Neurofeedback Intermediate - Advanced (BCIA Review Course)- Lynda Thompson &amp; Michael Thompson</td>
</tr>
<tr>
<td></td>
<td>Workshop 5 - A Clinician’s Guide to Understanding Recent Developments in Neurofeedback: Amplitude, Live Z-score and sLORETA Training Explained- Tom Collura &amp; Penijean Rutter</td>
</tr>
<tr>
<td></td>
<td>Workshop 6 - Psychopharmacology of Depression- Fred Shaffer</td>
</tr>
<tr>
<td></td>
<td><strong>Time</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Friday September 21, 2012 Workshop Schedule</strong></td>
</tr>
<tr>
<td>3:15-6:30 PM</td>
<td>Workshop 7 – (Day 1 of 2) Setting up for Success with Aspergers and Autism Spectrum Disorders (2 day WS)- Michael Thompson &amp; Lynda Thompson</td>
</tr>
<tr>
<td></td>
<td>Workshop 8 - Ethics and Neurofeedback: Thoughtful Discussions – Rex Cannon</td>
</tr>
<tr>
<td></td>
<td>Workshop 10 - Breaking Down Barriers to Peak Performance Brain Training™ in Elite Athletes – Leslie Sherlin &amp; Noel Larson</td>
</tr>
<tr>
<td></td>
<td>Workshop 11 - Infra-low Frequency Training in Clinical Practice – Mark Smith</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Time</th>
<th>Saturday September 22, 2012 Workshop Schedule</th>
</tr>
</thead>
<tbody>
<tr>
<td>2:45-6:00 PM</td>
<td>Workshop 7.2 (Day 2 of 2) - Setting up for Success with Aspergers and Autism Spectrum Disorders (2 day WS)- Michael Thompson &amp; Lynda Thompson</td>
</tr>
<tr>
<td></td>
<td>Workshop 13 - ADHD and Learning Disabilities: Integrating Clinical Knowledge and Individual Symptoms and Neurophysiology in the Formation of Neurofeedback Treatment Plans- Robert Coben &amp; Anne Stevens</td>
</tr>
<tr>
<td></td>
<td>Workshop 14 - Biofeedback and Neurofeedback with Professional and Olympic Athletes – Michael Linden, Penny Werthner, Wes Sime &amp; Sanford Silverman</td>
</tr>
<tr>
<td></td>
<td>Workshop 15 - sLORETA and Z Score Neurofeedback: A Clinical Symbiosis- Mark Smith</td>
</tr>
<tr>
<td></td>
<td>Workshop 16 - HRV Biofeedback Training Strategies- Fred Shaffer</td>
</tr>
<tr>
<td></td>
<td>Workshop 17 – Validating Emotionally Charged Ipsative Assessments Using EEG Gamma Asymmetry- Tom Collura &amp; Ronald Bonnstetter</td>
</tr>
</tbody>
</table>
ISNR 2012 Pre-Conference Workshops

Monday, September 17, 2012

Pre WS 1.1: LENS Foundations Training (Day 1 of 3)
(Lecture, Experiential, Demonstration)
Michael Beasley, MS, Private Practice, mikebeasley@earthlink.net
Len Ochs, PhD, Ochs Labs, lochs@earthlink.net

Credits: 6.5

Level of Difficulty: Basic

Abstract
This 3-day workshop is a learning arena for the practitioner, which includes essential concepts, core paradigms, principles, and areas of applicability of the Low Energy Neurofeedback System (LENS) and how to integrate the concepts into the practitioner's practice. The workshop will offer hands-on training in the LENS in addition to a foundational knowledge in assessing the client, development of a treatment plan, using the concepts presented and how to reevaluate the effectiveness of the treatment plan.

References


Marcus, L. (2001). EEG Amplitude and Variability Changes Following Low-Intensity Neurofeedback-Based Stimulation for Fibromyalgia. Palo Alto, CA, Western Graduate School of Psychology. Ph.D.


Ochs, L. (1997). EDS: Background and operation, EEG-driven pico-photonic stimulation. Walnut Creek, CA, Flexyx, LLC.


Ochs, L (2010) Underlying Treatment Issues in Neurofeedback as Exemplified by Treatment of
Seizure Disorders. Journal of Neurotherapy, Spring. 29-33.


**Learning Objective**

Define the core clinical principles of the LENS approach so that the central elements, issues, approaches, and practices make sense to the attendees.

To have participants start and stop LENSware, and manipulate the essential controls.

Learn how to conduct an initial evaluation.

Understand the concepts of sensitivity, hardiness and reactivity, as will be the CNS questionnaire, and cover the basics of topographic map reading as one of the cornerstones of treatment.

Internalize the theoretical and conceptual overview of the LENS approach, especially in relation to the theory, concepts, and practices of traditional neurofeedback. To define the core paradigms and principles of the LENS approach so that the central elements, issues, approaches, and practices make sense to the attendees.

Provide experience of skin preparation, electrode attachment, good-impedance recognition, and knowledge of 10-20 sites.

Summarize work with the evaluation to form a basis for considering topographic mapping.

**Outline**

10:00-11:00 (1 hour) Introduction to concepts, core paradigms, principles, and areas of applicability.

11:00-11:30 (1/2 hour) Basic features of the LENS software.

11:30-11:45 (1/4 hour) Break

11:45-1:00 (1 1/4 hours) Introduction of concepts of sensitivity, hardiness, reactivity, and suppression. Conduction of initial evaluation.

1:00-2:30 (1 1/2 hours) Lunch

2:30-4:00 (1 1/2 hours) Practicum: first session with participants. 4:00-4:15 (1/4 hour) Break

4:15-6:00 (1 3/4 hours) Practicum continues. Clinical decision making when using the LENS

**Financial Interest:** Mike Beasley has no financial interest or relationship other than as an independent occasional trainer for OchsLabs, Inc. Len Ochs continues to design systems on a volunteer basis, receiving no moneys or position from OchsLabs, Inc.

**Pre WS 2.1: Advanced LENS Training (Day 1 of 3)**
Abstract
This 3-day Advanced LENS Training will begin with a review of fundamentals; Treatment flow from evaluations to treatment and re-evaluations; Understanding Maps and their significance; Advanced offset management; In-depth analysis of LENS Application components and how they relate to the concepts of Sensitivity, Reactivity, Incompletely resolved childhood problems, Advanced management of suppression and over stimulation with time spent on Suppression Maps; Clarification of differences between aberrant reactions, background medical problems, and releases of suppression/necessary transitional states. The core for discussions will be the new settings screen, which will serve as a focus for settings relevant to the LENS.

References


Marcus, L. (2001). EEG Amplitude and Variability Changes Following Low-Intensity Neurofeedback-Based Stimulation for Fibromyalgia. Palo Alto, CA, Western Graduate School of Psychology. Ph.D.


Ochs, L. (1997). EDS: Background and operation, EEG-driven pico-photic stimulation. Walnut Creek, CA, Flexyx, LLC.


Learning Objective
Demonstrate knowledge of the LENS basics principles and practices.
Demonstrate deepening knowledge of mapping analysis in order to better track patient changes.
Demonstrate deepening knowledge brain access systems via the variety of LENS maps.
Increase knowledge of brain and vascular physiology in relation to components of the LENS applications and signal types.
Discuss Sensitivity and the Reactivity/Suppression/Hardiness and Behavioral Suppression questionnaire.
Have an in-depth discussion of components of LENS Applications as related to the above concepts in the Sensitivity questionnaire.
Demonstrate capability of discussing several aspects of EEG suppression and suppression mapping.

Outline
· Review, What’s New, Treatment Strategies: – 8 hours

Objective: To have participants demonstrate knowledge of the LENS basics principles and practices

Content:
· Review of basic certification exam (40) - experiential
· Discussion of answers to the exam (40) - experiential

Objective: Participants demonstrate deepening knowledge of mapping analysis in order to better track patient changes.

Content:
· Descriptive review of sequences of maps (60) – didactic and experiential

Objective: Participants demonstrate deepening knowledge of EEG dynamics in order to better track patient changes.

Content: · Review of Band and Frequency transformations in sequences of maps (90) - experiential

Objective: To increase flexibility of therapist functioning during evaluation and treatment sessions

Content:
· Working with patients in sessions: interview, evaluations and/or treatment sessions, anticipating and predicting problems (120) – demonstration, didactic
· Classification of session content by participants, as options, keyed to outcome enhancement (60) - experiential

Objective: Discussion of Sensitivity and the Reactivity/Suppression/Hardiness questionnaire

Content:
Material on Reactivity/Suppression/Hardiness, as well as transition states as different from over-stimulation reactions (70) - experiential

Financial Interest: Len Ochs is the inventor and developer of the LENS. I have no ownership, paid or unpaid position with OchsLabs, Inc. as my entire income is from Social Security retirement; I am not an employee of OchsLabs, Inc. Cathy Wills is an employee of OchsLabs.

Tuesday, September 18, 2012

Pre WS 1.2: LENS Foundations Training (Day 2 of 3) (Lecture, Experiential, Demonstration)

Michael Beasley, MS, Private Practice, mikebeasley@earthlink.net
Len Ochs, PhD, Ochs Labs, lochs@earthlink.net

Credits: 6.5

Level of Difficulty: Basic

Abstract
This 3-day workshop is a learning arena for the practitioner, which includes essential concepts, core paradigms, principles, and areas of applicability of the Low Energy Neurofeedback System (LENS) and how to integrate the concepts into the practitioner's practice. The workshop will offer hands-on training in the LENS in addition to a foundational knowledge in assessing the client, development of a treatment plan, using the concepts presented and how to reevaluate the effectiveness of the treatment plan.

References


Hawthorne Medical Press.


Marcus, L. (2001). EEG Amplitude and Variability Changes Following Low-Intensity Neurofeedback-Based Stimulation for Fibromyalgia. Palo Alto, CA, Western Graduate School of Psychology. Ph.D.


Ochs, L (1997). EDS: Background and operation, EEG-driven pico-photic stimulation. Walnut Creek, CA, Flexyx, LLC.

Ochs, L (2006). Thoughts about EEG-Driven stimulation after three years of its uses: Ramifications for concepts of pathology, recovery, and brain function.


**Learning Objective**

Practice doing topographic maps.

Concretize the paradigms and principles by the use of the Report Generator to show how it becomes a tool to generate treatment plans and treatment re-evaluations.

Illustrate issues important in the conduct of the LENS approach so that the clinician can more intelligently inform the prospective client about the risks and benefits of his/her particular involvement.

Introduce EEG suppression through the use of the suppression maps, showing site sorts according to the coefficient of variation.

Demonstrate the use of the Report Generator in relation to mapping, data management, and treatment planning.

Integration of data related to dose, patient characteristics, and patient education.

Demonstrate the management of data from its export from LENSware², to its import and generation of information, treatment plans, and treatment evaluation in the Report Generator.

**Outline**

10:00-11:30 (1 1/2 hours) Principles of Dominant Frequency, frequency offset, feedback frequency, sensitivity, hyper- and hypo-reactivity to stimulation, cortical permeability and integration, and structural vs. functional impairments and improvements.

11:30-11:45 (1/4 hour) Break

11:45-1:00 (1 1/4 hours) Practicum with participants. Use of evaluation with clients for development of treatment plan.

1:00-2:30 (1 1/2 hours) Lunch

2:30-4:00 (1 1/2 hours) Practicum continues. Treatment plan reevaluation.

4:00-4:15 (1/4 hour) Break

4:15-6:00 (1 3/4 hours) Practice in performing topographic brain maps and producing reports. Introduction of suppression concepts.
Financial Interest: Mike Beasley has no financial interest or relationship other than as an independent occasional trainer for OchsLabs, Inc. Len Ochs continues to design systems on a volunteer basis, receiving no moneys or position from OchsLabs, Inc.

Pre WS 2.2: Advanced LENS Training (Day 2 of 3)
(Lecture, Experiential, Demonstration)
Len Ochs, PhD, Ochs Labs, lochs@earthlink.net
Cathy Wills, MSN, Ochs Labs, cathywills@ochslabs.com

Credits: 6.5

Level of Difficulty: Advanced

Abstract
This 3-day Advanced LENS Training will begin with a review of fundamentals; Treatment flow from evaluations to treatment and re-evaluations; Understanding Maps and their significance; Advanced offset management; In-depth analysis of LENS Application components and how they relate to the concepts of Sensitivity, Reactivity, Incompletely resolved childhood problems, Advanced management of suppression and over stimulation with time spent on Suppression Maps; Clarification of differences between aberrant reactions, background medical problems, and releases of suppression/necessary transitional states. The core for discussions will be the new settings screen, which will serve as a focus for settings relevant to the LENS.

References


Marcus, L. (2001). EEG Amplitude and Variability Changes Following Low-Intensity Neurofeedback-Based Stimulation for Fibromyalgia. Palo Alto, CA, Western Graduate School of Psychology. Ph.D.


Ochs, L. (1997). EDS: Background and operation, EEG-driven pico-photic stimulation. Walnut Creek, CA, Flexyx, LLC.


Ochs, L. (2010) Underlying Treatment Issues in Neurofeedback as Exemplified by Treatment of
Learning Objective
Name and define the elements of the LENS applications

List elements of the considerations of customizing the LENS applications for those of different degrees of sensitivity

List elements of the considerations of customizing the LENS applications for treatment-resistant problems

Describe different types of sensitivity as a biological and perceptual trait, rather than as the culturally familiar one of reactivity, and detail the consequences of different types of sensitivity as they interact with different components of the LENS approach.

Describe different types of reactivity as a biological trait, rather than as the culturally familiar one of sensitivity, and detail the significance that reactivity holds for those administering and receiving LENS sessions.

Describe the incomplete resolution, energy as it pertains to anxiety and seizure spectrum problems, and the use of the trait of hardiness.

Outline
Objective: Participants demonstrate deepening knowledge of mapping analysis in order to better track patient changes.

Content: • Descriptive review of sequences of maps (60) - experiential

Objective: Participants demonstrate deepening knowledge of EEG dynamics in order to better track patient changes.

Content: • Review of Band and Frequency transformations in sequences of maps (120) - experiential

Objective: To increase flexibility of therapist functioning during evaluation and treatment sessions

Content:

• Working with patients in sessions: interview, evaluations and/or treatment sessions (120) - demonstration

• Classification of session content by participants, as options, keyed to outcome enhancement (90) - experiential

Objective: Discussion of Sensitivity and the Reactivity/Suppression/Hardiness questionnaire

Content:

• Material on Reactivity/Suppression/Hardiness, as well as transition states as different from over stimulation reactions (90 – didactic
**Financial Interest:** Len Ochs is the inventor and developer of the LENS. I have no ownership, paid or unpaid position with OchsLabs, Inc. as my entire income is from Social Security retirement; I am not an employee of OchsLabs, Inc. Cathy Wills is an employee of OchsLabs.

**Pre WS 3.1: Introduction to the Practice of Neurofeedback:** Assessment Leads to Appropriate Intervention (Day 1 of 2)
(Lecture, Experiential, Demonstration)
Lynda Thompson, Ph.D., The ADD Centre, landmthompson@gmail.com
Michael Thompson, M.D., The ADD Centre, landmthompson@gmail.com

**Credits:** 8

**Level of Difficulty:** Basic

**Special Note:** Participants who wish to obtain their BCIA certification will earn 16 hours of credits for the didactic material (rubrics I, II, and III) of the Biofeedback Certification International Alliance Blueprint of Knowledge, by completing this two day workshop.

**BCIA Rubrics I, II, III.**

I. Orientation to Neurofeedback (During the morning of day 1. It includes learning theory as it is applied to NFB training sessions)

II. Basic Neurophysiology and Neuroanatomy (During the morning of day 2. It includes an emphasis on how understanding functions of cortical Brodmann Areas can lead to designing your NFB intervention.)

III. Instrumentation & Electronics (Afternoons, with demonstrations, both days. It includes details of reading the raw EEG, rejecting artifacts, graphing your data, and how assessment procedures lead to decisions concerning appropriate intervention.)

For detail as to the content for these rubrics, go to [www.BCIA.org](http://www.BCIA.org).

**Abstract**

Note: Participants are welcome to sign up for one day or two days. The purpose of the first day is to introduce new practitioners to the basics of EEG biofeedback (Neurofeedback). The second day will review and expand on the basics of EEG assessment and Neurofeedback and introduce how to combine Neurofeedback with peripheral Biofeedback and metacognitive strategies for common disorders.

**Day 1:**

Introduction: scientific basis for Neurofeedback (NFB), basic terms & definitions, understanding the EEG, artifacts, single channel assessment, learning theory (operant & classical conditioning) as it applies to NFB, designing training sessions, tracking results.

This introductory workshop begins with a brief history of the scientific basis of NFB followed by defining the basic terms and concepts including: the electroencephalogram (EEG) and understanding brainwaves (frequency, morphology, amplitude, magnitude, power, location, reactivity and origin); artifacts; impedance; high and low pass filters, the differential amplifier; international 10-20 sites and relation to Brodmann Areas (BAs); basic functional neuroanatomy, such as how networks involve specific functional areas of the cortex and their specific connections through the basal ganglia to thalamus and back to functionally related areas of the cortex. This provides a basic understanding of why you train at particular sites. Montages discussed include: referential, sequential and Laplacian. This discussion will note how these are used for EEG assessment and training decisions. Discussion of these terms is enhanced by hands-on demonstration to show in detail how electrodes are applied, impedance is checked, artifacts are identified and removed, and how the single channel EEG results in one Hz bins (1 to 60 Hz) with key ratios (e.g., theta/beta) are evaluated and graphed. For the graphing we use Excel because this is available to most practitioners regardless of the equipment
used as long as their equipment can show the raw EEG and do statistics. (Graphing learning curves is also shown for training sessions.) We explain the logic of combining EEG assessment results, with knowledge of functions of relevant areas (BAs) and the client's key symptoms, to plan for successful NFB intervention. ADHD and learning difficulties will be used as the first examples and case examples of clients' EEGs will be shown.

There will be discussion of symptom pictures that require a 19 channel EEG assessment that a beginning practitioner could ask an experienced colleague to do in order guide treatment. Beginners in NFB need to be able to understand presentations at the ISNR meeting, therefore we will show data from 19 channel (full cap) EEG assessments, including LORETA analysis, to introduce this more advanced level of assessment and intervention. With both single channel and 19 channel assessments, the EEG findings, knowledge of functional neuroanatomy, and the client's symptom picture are all used to determine the site and frequency ranges for training.

The afternoon will emphasize how to do NFB using operant and classical conditioning, shaping, measurement of sustaining desired EEG activity, tracking the percentage of time “in the zone”, and doing amplitude training of each targeted frequency band. Graphing of progress during the session (and across sessions) using Excel will be shown. Designing appropriate interventions is stressed and discussion will center on how the triad of symptom picture, neuroanatomy, and EEG findings leads to a logical placement of electrodes for enhancement or inhibition of specific frequency bands. There will be mention of z-score training in addition to the usual amplitude and coherence training paradigms.

We want the workshop participants to learn to avoid the pitfall of expecting the machine to do the work. Their coaching is an important component of their client’s success, so we explain how to combine NFB with work on metacognitive strategies and show how to combine simple biofeedback methods, especially respiration and heart rate variability training, to encourage the client to relax while remaining alert and focused.

We do not wish to frighten the new comers but we want them to be realistic about how much time and effort it really takes to get excellent results. We ourselves are still learning from every client, and that is one reason why applied neuroscience is such an interesting field.

References


Goals/Objectives

Define the International 10 – 20 system Electrode Placement System.

List standard EEG bandwidths and describe typical mental states associated with each of these bandwidths at the Cz site.

Explain how the practitioner uses the basic principles of learning theory during each training session with a client.

Explain how the differential amplifier allows the practitioner to accurately assess brain waves while excluding major artifacts that are in-common to each site.

Identify the common EEG findings in children who have a diagnosis of ADHD.

Put electrodes on to the scalp (CZ) and ears with good impedance readings.

Do a single Hertz bin EEG assessment, identify common artifacts in the EEG such as eye-blink and muscle and make an appropriate decision as to which bandwidths to enhance or to inhibit for training that client.

Do a neurofeedback training session with a volunteer “client” to appropriately enhance and inhibit particular bandwidth amplitudes.

Outline

History: includes major contributions to what a practitioner does in day-to-day NFB plus BFB practice. Learning theory as the basis of a NFB practitioner’s day to day practice.

Essential Terminology Including: Frequency (Hz), Amplitude (μV), Magnitude, Power (pW), Impedance, Differential Amplifier, Z-scores.

EEG Waves including: Delta, Theta, Alpha (low & high), SMR, Beta, Beta Spindling, Gamma, in terms of their Frequency, Morphology, Amplitude, Location, and Reactivity as well as their correlations with mental states

10-20 System; Commonly used montages: referential, sequential and, for 19-channel assessments, Laplacian. Describe single channel QEEG assessment and discuss its utility.

Provide examples of EEG patterns using examples from clients with ADHD and explain how to read the raw EEG (time vs. amplitude), an EEG spectrum (frequency vs. magnitude), and line graphs. Show examples of: Delta, Theta, Alpha (low and high), Beta (low frequency and high frequency), Gamma.

Begin hands-on demonstration of assessment (one channel single Hz ‘bins’ from 1 to 60 Hz) and simple training using a volunteer from among the workshop participants. This will include instruction regarding attachment of electrodes, choice of sites, choice of reference and rationale for it, checking impedance and importance of this.

Demonstrate Quantitative EEG Assessment using data collected during the hands-on demonstration,
including how to recognize and remove artifacts, statistical analysis, and graphing of results. Discuss and demonstrate training techniques, pretending that the volunteer is a child with ADHD. Calculation of important ratios from the data collected, and discuss the research that helps understand their meaning (e.g., theta / beta power ratios).

Explanation of more advanced terms that the beginner should be able to recognize and understand when they are found in the literature or heard in lectures. This includes definition and explanation of Brodmann Areas, coherence and phase, LORETA.

Financial Interest: Lynda Thompson is co-author of THE A.D.D. BOOK. Michael and Lynda are co-authors of SETTING UP FOR CLINICAL SUCCESS. Michael and Lynda Thompson are co-authors of THE NEUROFEEDBACK BOOK. It is likely that these books may be on sale at the meeting. The authors will state their interest in these books at the workshop.

Pre WS 8: Integrating Norms-Based Neurofeedback within a Holistic Psychophysiological Treatment Approach
Preconference Sponsor Workshop – Thought Technology
(Lecture, Demonstration)
Linda Walker, LPC, Private Practice, c2sail@yahoo.com

Credits: ISNR does not provide CEUs for this workshop. CEUs may be offered by the Sponsor directly.

Level of Difficulty: Basic to Intermediate

Abstract
As norms-based neurofeedback shows increasing promise as a treatment tool, integrating it into a full psychophysiological program can increase its impact on whole-person optimization.

Also known as Z-Score neurofeedback, norms-based EEG treatment has shown promise in holistically treating and balancing the brain in a variety of conditions. Adding biofeedback methods -- especially heart rate variability and EMG training -- can help the client balance underlying physiological systems that may contribute to dysregulation.

Combining neurofeedback and biofeedback interventions is certainly nothing new. What makes the discussion relevant to Z-score neurofeedback, with its potential to train 248 metrics simultaneously over four 10-20 sites, is helping the client integrate feedback in a way that is helpful, and not overwhelming.

This workshop will review the foundations and application of Z-score neurofeedback as it is implemented on the Thought Technology platform and consider strategies to effectively implement this intervention alongside biofeedback interventions. Case studies, as well as treatment examples, methods and rationales will be discussed.

References


Outline
A review of Z-score training and the present state of research.
An overview of psychophysiological assessment and examples of useful assessments to help clinicians integrate Z-score nfb and psychophysiological approaches for treating the client's symptoms and complaints.
A discussion of case studies and examples to illustrate how Z-score training and biofeedback are woven together in the treatment session to create the greatest impact.

Financial Interest: Certain portions of this presenter's travel and conference expenses were sponsored by Thought Technology.

ISNR does not provide CEUs for this workshop. CEUs may be offered by the Sponsor directly.

Wednesday, September 19, 2012

Pre WS 1.3: LENS Foundations Training (Day 3 of 3)
(Lecture, Experiential, Demonstration)
Michael Beasley, MS, Private Practice, mikebeasley@earthlink.net
Len Ochs, PhD, Ochs Labs, lochs@earthlink.net

Credits: 6.5
Level of Difficulty: Basic

Abstract
This 3-day workshop is a learning arena for the practitioner, which includes essential concepts, core paradigms, principles, and areas of applicability of the Low Energy Neurofeedback System (LENS) and how to integrate the concepts into the practitioner's practice. The workshop will offer hands-on training in the LENS in addition to a foundational knowledge in assessing the client, development of a treatment plan, using the concepts presented and how to reevaluate the effectiveness of the treatment plan.

References


Learning Objective
Learn how to make topographic maps and offset evaluations.

Establish a logical thread from intake, sensitivity evaluations, possible offset evaluation, and mapping that will lead to the choices of applications and settings in further treatment.


Marcus, L. (2001). EEG Amplitude and Variability Changes Following Low-Intensity Neurofeedback-Based Stimulation for Fibromyalgia. Palo Alto, CA, Western Graduate School of Psychology. Ph.D.


Ochs, L. (1997). EDS: Background and operation, EEG-driven pico-photic stimulation. Walnut Creek, CA, Flexyx, LLC.


Understand a picture of the energetic diagnoses (ADD, ADHD, Autism, Asperger’s, Anxiety, Seizure Spectrum, clinical, and EEG data into something that makes sense to the clinician and prospective client.

Conceptualize detail of the range of seizure spectrum problems that don’t look like actual seizure problems, but which require specific applications to which actual seizures respond well.

Integrate data related to dose, patient characteristics, and patient education

Establish the beginnings of a rationale by which the Provider can begin to refer his or her clients out to another healthcare provider, based on some of the data already obtained.

Learn how to talk about the LENS to clients and other professionals, what not to say, and to catch in their errors of speaking the provider from making fools of themselves.

Outline
10:00-11:30 (1 1/2 hours) Clinical decisions: Putting together Offset and Map Evaluations for treatment plan.

11:30-11:45 (1/4 hour) Break

11:45-1:00 (1 1/4 hours) Practicum, using integration of above information

1:00-2:30 (1 1/2 hours) Lunch

2:30-4:00 (1 1/2 hours) Relationship of dose to sensitivity, reactivity, suppression, and hardiness related to treatment plan

4:00-4:15 (1/4 hour) Break

4:15-6:00 (1 3/4 hours) Review and Integration of all concepts presented. Questions, Discussion, Exam and Evaluation.

Financial Interest: Mike Beasley has no financial interest or relationship other than as an independent occasional trainer for OchsLabs, Inc. Len Ochs continues to design systems on a volunteer basis, receiving no moneys or position from OchsLabs, Inc.

Pre WS 2.3: Advanced LENS Training (Day 3 of 3)
(Lecture, Experiential, Demonstration)
Len Ochs, PhD, Ochs Labs, lochs@earthlink.net
Cathy Wills, MSN, Ochs Labs, cathywills@ochslabs.com

Credits: 6.5

Level of Difficulty: Advanced

Abstract
This 3-day Advanced LENS Training will begin with a review of fundamentals; Treatment flow from evaluations to treatment and re-evaluations; Understanding Maps and their significance; Advanced offset management; In-depth analysis of LENS Application components and how they relate to the concepts of Sensitivity, Reactivity, Incompletely resolved childhood problems, Advanced management of suppression and over stimulation with time spent on Suppression Maps; Clarification of differences between aberrant reactions, background medical problems, and releases of suppression/necessary transitional states. The core for discussions will be the new settings screen, which will serve as a focus for settings relevant to the LENS.

References


Society for Neuronal Regulation, National Conference. Ft. Lauderdale.

Marcus, L. (2001). EEG Amplitude and Variability Changes Following Low-Intensity Neurofeedback-Based Stimulation for Fibromyalgia. Palo Alto, CA, Western Graduate School of Psychology. Ph.D.


Ochs, L (1997). EDS: Background and operation, EEG-driven pico-photic stimulation. Walnut Creek, CA, Flexyx, LLC.

Ochs, L (2006). Thoughts about EEG-Driven stimulation after three years of its uses: Ramifications for concepts of pathology, recovery, and brain function.


Learning Objective

Demonstrate adjustment of the Offset on LENSware² software and list the considerations as a fine-tuning adjustment

List uses of the Photonic Stimulator as a blocker of sympathetic activity and as a tool for assisting in the return mitochondrial learning and adaptation in relation to client sensitivity, hardiness and reactivity, especially in relation to treatment-resistant problems across the age spectrum

List uses of the LENS with pain management issues, focusing on migraine and other vascular pain, diabetic neuropathy, fibromyalgia, phantom limb pain, and painful swelling

Demonstrate an understanding of multiple ways to address the problems with the LENS approach, adding the advantages and disadvantages of the different approaches.

Discuss the range of Seizure Spectrum Disorders and attach elements of these problems to elements of the LENS settings in LENSware².
List the effects of inflammation, infections, and trauma on the degree of spiking and fast waves as measured on the scalp, and how these may be shown and hidden in relation to the kinds and degrees of EEG suppression.

Outline
Objective: Participants demonstrate deepening knowledge of mapping analysis and mapping dynamics in order to better track patient changes.

Content:
- Explaining maps to patients (60) – didactic and experiential

Objective: To increase flexibility of therapist functioning during evaluation and treatment sessions

Content:
- Working with patients in sessions: interview, evaluations and/or treatment sessions (120) – demonstration and experiential
  
- More classification of session content by participants, as options, keyed to outcome enhancement (60) - experiential
  
- Tying mapping changes to both suppression release and behavioral changes (60) - didactic
  
- Managing difficult patients and difficult problems, while appreciating the easy ones (60) - didactic

Objective: Participants demonstrate scope of advanced knowledge

- Examination and Discussion of both questions and responses (150) - experiential
  
- Course evaluation (30) – Experiential

Financial Interest: Len Ochs is the inventor and developer of the LENS. I have no ownership, paid or unpaid position with OchsLabs, Inc. as my entire income is from Social Security retirement; I am not an employee of OchsLabs, Inc. Cathy Wills is an employee of OchsLabs.

Pre WS 3.2: Introduction to the Practice of Neurofeedback: Assessment Leads to Appropriate Intervention (Day 2 of 2)

(Lecture, Experiential, Demonstration)
Lynda Thompson, Ph.D., The ADD Centre, landmthompson@gmail.com
Michael Thompson, M.D., The ADD Centre, landmthompson@gmail.com

Credits: 8

Level of Difficulty: Intermediate

Special Note: Participants who wish to obtain their BCIA certification will earn 16 hours of credits for the didactic material (rubrics I, II, and III) of the Biofeedback Certification International Alliance Blueprint of Knowledge, by completing this two day workshop.

BCIA Rubrics I, II, III.
I. Orientation to Neurofeedback (During the morning of day 1. It includes learning theory as it is applied to NFB training sessions)
II. Basic Neurophysiology and Neuroanatomy (During the morning of day 2. It includes an emphasis on how understanding functions of cortical Brodmann Areas can lead to designing your NFB intervention.)

III. Instrumentation & Electronics (Afternoons, with demonstrations, both days. It includes details of reading the raw EEG, rejecting artifacts, graphing your data, and how assessment procedures lead to decisions concerning appropriate intervention.)

For detail as to the content for these rubrics, go to www.BCIA.org.

Abstract
First we will answer questions arising from Day 1. We will then introduce the participants to basic neurophysiology and neuroanatomy and how this knowledge contributes to designing your NFB intervention. This covers basic functional neuroanatomy, such as how networks involve specific functional areas of the cortex and their specific connections through the basal ganglia to thalamus and back to functionally related areas of the cortex. This provides a basic understanding of why you train at particular sites. We will then cover more advanced definitions of terms used in the field of biofeedback, including autonomic nervous system, heart rate, respiration rate, electrodermal response (EDR), electromyography (EMG); peripheral skin temperature. These are all ‘TONIC’ measures related to sympathetic nervous system tone. More time will be spent on heart rate variability, which measures an ‘OSCILLATING’ system that also reflects parasympathetic activity. There will be some emphasis on the synergy inherent in combining BFB training with the NFB training. The demonstrations on the second day will combine the EEG assessment with a psycho-physiological stress assessment that measures all of these biofeedback modalities. We emphasize how single and two channel assessments can be done to do a reliability check on 19 channel findings. A hands-on demonstration will show how single channel NFB is combined with BFB and with learning strategies to address basic disorders such as ADHD where anxiety can be an important comorbidity. Children with Asperger’s are often initially diagnosed as having ADHD; thus this disorder, that has ADHD symptoms plus anxiety, executive functioning problems, and major social difficulties will be discussed. The emphasis is on the importance of accurate diagnosis and being able to address both the ADHD symptoms plus the other accompanying symptoms using a combination of NFB + BFB + learning strategies.

As time allows and according to participants’ interest, we can touch upon other disorders such as seizure disorders, different types of depression, Tourette’s syndrome, head injury (TBI) and pain management. However, this will remain an ‘introductory’ workshop and we cover some other disorders in more advanced detail in another workshop.

References


Goals/Objectives
Carry out a basic psychophysiological stress assessment and identify basic psychophysiological responses to stress and patterns found during recovery from stress.

Do a neurofeedback training combined with basic biofeedback training session with a volunteer.

Integrate metacognitive strategies (on-task training) into the neurofeedback training session.

Outline some characteristics of clients who would require a 19-channel EEG assessment and understand how these are interpreted, including coherence measures and LORETA.

Define the term „Brodmann Areas“ and describe their general location on the cortex.

List potential side effects of NFB and of BFB with an emphasis on over-breathing.

Discuss the efficacy guidelines for research on NFB and BFB as developed by the joint ISNR/AAPB committee and state for which two disorders NFB has the highest level of efficacy.

Define “z-score” and explain how z-scores are used in EEG assessment and training.

Define “coherence” and describe how to train coherence between two sites.

Outline
Review with the participants the highlights from Day One including: basic terms, waves, examples of child then adult ADHD EEGs. Review the importance of collection of baseline data during each session and how to graph session statistics to show learning curves within and across sessions. Emphasize importance of tracking client progress during each session and between sessions to show their ability for self-regulation.

Review EEG artifacts. Review the EEG Instrument including: filters, impedance, differential amplifier, optical isolation, etc.

EEGs of seizure disorders, emphasis on cases incorrectly referred by physicians as ADHD (Ethics of appropriate practice are emphasized here: these cases must be sent back to appropriate physicians for reassessment and decisions concerning medical treatment of the seizures).

Basic terms and measurements in biofeedback: Autonomic Nervous System - Heart Rate & Respiration Rate; Electrodermal Response (EDR) – used to be called the Galvanic Skin Response (GSR)

Electromyography (EMG): Peripheral Skin Temperature. These are all ‘tonic’ measures related to ‘sympathetic’ tone versus Heart Rate Variability (HRV), which measures an ‘oscillating’ system and parasympathetic activity. Discuss, briefly, anabolic and catabolic states.

Hands-on demonstration with audience participation of a psychophysiological stress assessment
followed by demonstration of combined operant conditioning of both NFB (EEG) and BFB modalities. The Demonstration will show Heart Rate Variability training combined with peripheral skin temperature, electrodermal, and electromyogram (EMG) feedback with the emphasis being on reduction and control of anxiety and tension plus how to generalize the work done in a session to everyday life.

Continue with demonstration and audience participation, showing graphing results of assessment and of the training sessions. Discuss how a 2-channel assessment is carried out and when it is appropriate, with patterns found in depression and/or ADHD plus dyslexia as the example(s).

Discuss how NFB ‘top-down’ training works synergistically with HRV ‘bottom-up’ training, providing explanations of the neuroanatomical connections that support the combination. Discuss how the addition during training sessions of metacognitive strategies assists in two ways: (1) directing the neural networks being affected by the training and (2) promoting generalization to everyday functioning. Recognize which clients require a 19-channel EEG assessment and understand how these are interpreted, including overview of what a brain map is and what it looks like and what a LORETA image looks like and means.

Results of NFB + BFB with statistics from our clinical research with ADHD and with Asperger’s Syndrome. Delineation of efficacy levels and discussion of levels of efficacy attributed to Neurofeedback for various disorders. Time for further review of topics covered, as desired by the participants, and answering of participants’ questions.

Financial Interest: Lynda Thompson is co-author of THE A.D.D. BOOK. Michael and Lynda are co-authors of SETTING UP FOR CLINICAL SUCCESS. Michael and Lynda Thompson are co-authors of THE NEUROFEEDBACK BOOK. It is likely that these books may be on sale at the meeting. The authors will state their interest in these books at the workshop.

Pre WS 4: Electrocerebral Abnormalities in EEG/QEEG: Subclinical Findings Redefined Treatment Implications and Considerations (Lecture, Demonstration)

Ron Swatzyna, PhD, Tarnow Center, ron@tarnowcenter.com
Jay Gunkelman, QEEG-T, Brain Science International, qegjay@sbcglobal.net

Credits: 8

Level of Difficulty: Intermediate

Abstract

Introduction

The utility of conventional EEG has been discounted for years and supported by the old adage that “you do not treat the EEG.” Although this misnomer is widely accepted in the field of neurology, failure to properly scrutinize the raw EEG can greatly inhibit responsible analysis and accurate diagnosis. We have found that EEG combined with QEEG analysis provides a treasure trove of supporting evidence of underlying pathology.

The overarching reason for doing an EEG is to find evidence of organic brain abnormalities (Hughes, 1996) and link it to cerebral dysfunction (Daly & Pedley, 1997) and psychological pathology (Asokan, Pareja & Niedermeyer 1987). However, certain abnormal EEGs can be very elusive. Those in which the occurrence is intermittent and/or the severity very mild, miss “clinical threshold.” Many encephalographers dismiss these "borderline" rhythms as being insignificant when in fact they are
Decades ago, Ernst Niedermeyer found that there was insufficient data to identify the value of "borderline" EEG rhythms which remain controversial to this day. There was not sufficient proof. William Cowper, a great and widely read 18th century English poet wrote "The absence of evidence is not evidence of absence." To that end, Niedermeyer started the collection of evidence that supports the importance and utility of EEG and QEEG technology. In his 1987 paper Niedermeyer concluded: "Modern views in clinical electroencephalography tend to minimize or even ignore such minor deviations. Such trends can be detrimental to EEG by depriving the electroencephalographer of important clinical-electrical correlations and withholding valuable information form the referring clinician". The linking of abnormal electrocerebral activity to observable pathology has come of age. Methods:

The presenters of this workshop just completed a case series study of over 200 clinical patients. Approximately 40 percent of their EEG/qEEGs were identified as abnormal. Conclusion:

A number of cases will be presented to demonstrate the utility and value of EEG/QEEG in “evidence-based” diagnosis, medication selection, developing personalized neuromodulation treatment protocols and at times suggesting further testing which may be critical to a patient's health and wellbeing.

References


Goals/Objectives

Explain the rationale for identifying electrocerebral abnormalities.

Identify the different types of commonly seen abnormalities.

Explain the link between ischemic migraine and paroxysmal discharges.

Know how to locate the source of paroxysms.

Understand how medication recommendations can be based on the EEG/QEEG.

Know when to refer for further testing.

Use methods taught to unravel difficult cases.

Know where to go to learn more about cerebral dysrhythmias.
Outline
Introduction and literature review (JG)
Case 1 (RS)
Case 2 (RS)
Case 3 (JG)
Discussion (RS & JG)
Cautions and Clinical implications (RS)
Q & A (RS & JG)
Summary (RS)

Financial Interest: Ron Swatzyna has no financial interest with any commercial supporter, product or service. Jay Gunkelman is a stockholder and the Chief Science Officer for Brain Science International, Inc, a California Corporation which provides EEG/ERP analysis and educational/mentoring services. I am a stockholder along with my siblings of an LLC called "FJJCSB" with which we manage and pay our elderly parent's living expenses. I provide independent consultations on EEG technical issues internationally. He is on various non-profit boards, including the Behavioral Medicine Research and Training Foundation and the Biofeedback Society of California, though none with financial interests in the field. We will not directly promote, market or sell any service or product at the workshop.

Pre WS 5: Enhancing Neurotherapy by Activating the Brainstem-Cerebellum-Midbrain-Cortex Pathways Through Primary-Reflex Movements - Practical Workshop (Half Day PM Workshop) (Experiential)
Suzanne Day, Master Psych, Wise Choice Educational Services, Suzanne.day@rogers.com

Credits: 4

Level of Difficulty: Basic

Abstract
Neurologists agree that the lack of inhibition (or integration) of primary reflexes, and consequently lack of brain development results in developmental delay and other mental problems. Such a disruption not only interferes with brainstem function, but also with basal ganglia-cerebellum networking and cortical processing, thus affecting learning, movement and attention. A lesser known fact is that these primary reflexes can be re-activated and integrated through specific rhythmic movements, “rebooting the brain’s software” and giving brain maturity a “second chance”. Michael Merzenich argued that, “The key in developing exercises is to give the brain the right stimuli in the right order with the right timing to drive plastic change”. Norman Doidge stated: “...many 'circuits' and even basic reflexes that we think are hardwired are not”.

The main goal of this practical workshop is to enable neurofeedback practitioners to learn and practice some of these vital reflex movements so that they, in turn, may be more confident in include this intervention to their usual neurofeedback training practice. The first section of this workshop introduces the neurology of the primary reflexes based in the brainstem, the functions of the cerebellum, as well as some postural reflexes based in the midbrain.
For the past 20 years, the presenter has witnessed significant improvements in her clients as she has implemented these movements as an initial intervention before adding neurofeedback training. Case studies using qEEG, academic performance, and continuous performance data will be provided in order to support the validity of using Primary-Reflex Rhythmic Movements as a viable, adjunct intervention to neurofeedback.

References


Masgutova, Svetlana and Denis Masgutov (2002-2009), Archetype Movements, A Blueprint for Movement and Cognitive Development, Dr. Svetlana Masgutova Institute (Polland).


Goals/Objectives
Understand the importance of the integration of the primary reflexes and be able to teach some the movements to their clients in order to better integrate these reflexes

Familiarize themselves to the sequential development of the central nervous system in term of functions.

Understand the benefits of the integration of the primary reflexes for learning and attention span.

Be confident with the sequence of the movements in order to be better prepared to teach them to their clients.

Outline
90 minutes on the neurology of the primary reflexes and the need for their integration. More detailed explanation of 4 main primary reflexes

150 minutes of experiential learning some rhythmic movements (mainly on the floor )

Financial Interest: I do not have any financial interest not relationship with commercial or manufacturer that is discussed in this presentation.

Pre WS 6: Biological Markers in Neurology and Psychiatry: Guidelines for Applying Event Related Potentials for Diagnosis and Treatment (Lecture, Demonstration)
Juri Kropotov, PhD, Institute of the Human Brain, yurykropotov@yahoo.com

Credits: 8

Level of Difficulty: Intermediate

Abstract
Multi-channel Event Related Potentials (ERPs) provide the only neuroscience method that allows clinicians to assess brain functions at high temporal resolution. ERPs are quite independent of spontaneous EEG oscillations – indexes of cortical self regulation. The effect size in ERP discriminating power (patients vs. healthy) is much higher than the effect size in quantitative EEG. The workshop is intended to introduce the ERP methodology to those who want to extend their clinical practice. The methodology of recording and analysis of ERPs will be presented. The focus will be made on recently emerged tools such as Independent Component Analysis, sLORETA imaging and HBI reference database for ERPs. Application of ERP for diagnosing different psychiatric conditions such as ADHD, dyslexia, autism, schizophrenia, OCD, depression, stroke and TBI will be discussed. ERP based endophenotypes of these conditions as well as application of biomarkers for constructing protocols of medication and neurotherapy of different neurological and psychiatric conditions will be presented.

References


Goals/Objectives
Describe biological markers (MRI, PET, EEG/ERP) markers of psychiatric and neurological conditions.

Learn the basics of ERP recoding and analysis.

Understand the differences between quantitative EEG and ERPs.

Explain the physiological meaning of different ERP waveforms such as MMN, P3a, P3b, P3 NOGO etc.

Describe independent component analysis as the method of artifact correction.

Understand independent component analysis as the method of extracting functionally meaningful components from a collection of ERPs.

Learn the discriminative power of independent components of ERPs in diagnosis of ADHD, schizophrenia, TBI, OCD, dyslexia.

Be able to practice assessment of brain dysfunction by means of comparing individual ERP with the normative data.

Outline
General description of biological markers in psychiatry and neurology (0.5 hour)

Methods of recording, computation and analysis of ERPs (0.5 hour)
Extraction independent components associated with different psychological operation (0.5 hour).

Association of ERPs components with functioning of brain systems (0.5 hour).

Reflection of dysfunctioning of brain systems in ERPs components (1 hour).

Recommendations for neurotherapy and medication on the basis of ERP assessment. (1 hour)

Demonstration of application of ERP methodology for assessment of various cases (TBI, ADHD, OCD, schizophrenia, depression, stroke, anxiety, autism).

Financial Interest: I am a co-founder of HBImed, a Swiss company.

Pre WS 7: QEEG and Neurofeedback using the BrainAvatar, Live Z-Scores, Live sLORETA Projection (LLP), and Combined Protocols

(Lecture, Demonstration)
Pre-Conference Sponsor Workshop

Thomas Collura, PhD, BrainMaster Technologies, Inc., tomc1@brainm.com

Mark Smith, MSW, Private Practice, marksmith50@verizon.net

Penijean Rutter, LMHC, Stress Therapy Solutions, penijean@gmail.com

Ronald Bonnstetter, University of Nebraska, rjb@unl.edu

Credits: ISNR does not provide CEUs for this workshop. CEUs may be offered by the Sponsor directly.

Level of Difficulty: Intermediate

Abstract
This full-day workshop will present a combination of clinical data and practical demonstrations showing the use of basic and advanced protocols that combine existing and emerging methods. The emphasis will be on neurofeedback that is simple, easy to understand and use, yet addresses brain function in terms of connectivity and global behavior. Research describing brain functional hubs and functional connectivity will be presented, with data relating to fMRI, QEEG, sLORETA, and behavioral data. Application to protocol design and application will be emphasized. Clinical case study results will be shown, using QEEG, behavioral, and clinical outcomes to support results. Practical design and use of protocols will be described, along with clinical criteria used to determine optimal designs and selections of protocol sets. Disorders including autism, depression, anxiety, and PTSD will be discussed as specific applications.

No CEs or CMEs are available for this preconference sponsor workshop.

References


**Goals/Objectives**
Design protocols using live z-scores, sLORETA, conventional, and new methods.

Conduct neurofeedback sessions using the above methods.

Determine the best protocols based upon QEEG clinical evaluations.

Assess the results of neurofeedback using the above protocols using QEEG and behavioral data.

**Outline**
Brain physiology, functional hubs, and connectivity (1 Hr)

QEEG methods to assess brain function and connectivity (1 Hr)

Case studies showing QEEG pre and post assessment and QEEG data (2 Hr)

Design of Combined Protocols (1 Hr)

Examples of advanced protocols and their clinical sue (2 Hr)

Experiential and Demonstration of advanced combined protocols (1 Hr)

**Financial Interest:** Dr. Collura has a financial interest in BrainMaster Technologies Inc. Part of the workshop will describe products provided by BrainMaster Technologies, along with other providers. Mark Smith receives no financial benefit from nor do I have any financial interest in BrainMaster Technologies, Inc. As part of StressTherapy Solutions, Inc. faculty, Penijeann Rutter has no financial gain or interest in BrainMaster Technologies. Dr. Bonnstetter is a Senior Vice President of Target Training International.

ISNR does not provide CEUs for this workshop. CEUs may be offered by the Sponsor directly.
Potential Neurofeedback Side Effects, Adverse Reactions, & Recommendations for Liability Protection (R,C)

D. Corydon Hammond, PhD, University of Utah School of Medicine, d.c.hammond@utah.edu

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: .5

Abstract
This presentation will review previously published research and clinical reports of side effects and more serious adverse reactions to neurofeedback training. Some of these problems have included seizures, increased anxiety and agitation, panic attacks, manic-like behavior, headaches, nausea, fatigue, sleep disturbance, anger and irritability, crying and emotional lability, incontinence, enuresis, an increase in depression, decline in cognitive functioning (decreased concentration, mental fogginess), increase in obsessional rumination and OCD symptoms, increase in somatic symptoms, Tourette’s tics (physical and vocal), slurred speech, loss of previous symptomatic improvements, and temporary disorientation or dissociation. Afterward this review further case reports will be presented, followed by practical recommendations for minimizing adverse effects, informed consent, and for practitioner liability protection.

References
Learning Objective
Understand the potential for side effects and adverse reactions from neurofeedback.

Be aware of several steps that practitioners can take for liability protection.

Outline
Review research and clinical reports of both side effects and more serious adverse reactions that have occurred.

Make recommendations for liability protection.

Financial Interest: No financial interests.

Enhancing Neurotherapy by Means of Brainstem Activation Through Primary-Reflex Rhythmic Movements (C)

Suzanne Day, Master Psych, Wise Choice Educational Services, Suzanne.day@rogers.com

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: .5
Abstract
Piaget argued that the “sensorimotor stage” is the first stage of development, facilitating all cognitive functions. Hebb proposed that neural structures, which he termed “cell assemblies”, constituted the material basis of mental concepts. Rita Levi-Montalcini discovered nerve growth factors which causes axonal growth. Michael Merzenich argued that, “The key in developing exercises is to give the brain the right stimuli in the right order with the right timing to drive plastic change.” Norman Doidge concludes: “...many 'circuits' and even basic reflexes that we think are hardwired are not.”

Neurologists agree that the lack of inhibition (or integration) of primary reflexes and lack of brain development are the causes of developmental delays and other mental problems. Such a disruption not only interferes with the brainstem functions but also with the basal ganglia-cerebellum networking and cortical processing, thus affecting learning, movement and attention. What is less known is how to re-activate these reflexes in order to ease their integration with rhythmic movements that gives brain maturity a “second chance”, “re-booting the brain software”. This session introduces the concept of a “bottom-up approach” (from brainstem to midbrain to cortex).

For the past 20 years, the author has witnessed significant improvements in her clients as she teaches her clients these movements as an initial intervention before adding neurofeedback training. Case studies using qEEG, academic performance and continuous performance data will be provided in order to support the validity of using primary-reflex rhythmic movements as a viable adjunct intervention to neurofeedback.

References


Masgutova, Svetlana and Denis Masgutov (2002-2009), Archetype Movements, A Blueprint for Movement and Cognitive Development, Dr. Svetlana Masgutova Institute (Polland).


Learning Objective
Recognize the important role of the primary-reflexes rhythmic movements in the neurodevelopment of the CNS and the benefit in using these movements as part of a neurotherapy intervention in order to mature the brain.

Outline
(3 min) Introduction

(5 min) Profile of development
STUDENT PRESENTATION

Single Trial Time-Frequency Domain Analysis of Error Processing in Post-Traumatic Stress Disorder (R)

Zachary Clemans, BS, University of Louisville, zaclem01@louisville.edu
Ayman El_baz, PhD, University of Louisville
Christopher Stewart, MD, University of Louisville
Estate Sokhadze, PhD, University of Louisville

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: .25

Abstract
Introduction: Post-traumatic stress disorder (PTSD) causes deficiencies in the error processing system. Electroencephalography (EEG) recordings of individuals with PTSD are often used to study error monitoring and correction deficits. Traditionally, many error trials are collected during a task with EEG systems and averaged together to obtain an estimate of a measure known as an event-related potential (ERP). Two ERPs are often used to study error processing, those being the error-related negativity and positivity (ERN and Pe) (Sokhadze et al., 2008; Yeung & Cohen, 2006). We have developed an alternate time-frequency domain single-trial analysis technique to assist in analyzing data sets with low numbers of error trials and examining the contribution that single error trials make in PTSD.

Methods: A wavelet transform of the single trial information collected from PTSD subjects (n = 10) and controls (n = 10) during an Eriksen’s flanker test was carried out using a custom MATLAB script. A measure of the ERN and Pe in the time-frequency domain (time-frequency ERN and Pe) was found in each single trial using a wavelet transform, and statistical analysis was carried out to determine if any significant differences between groups in latency or amplitude of the time-frequency measures were present. Behavioral analysis was also conducted.

Results: It was found that the PTSD group exhibited attenuated time-frequency ERN and Pe amplitudes as compared to the controls. Specifically, they exhibited less negative amplitudes in the time-frequency ERN and less positive amplitudes in the time-frequency Pe. Behavioral deficiencies such as slower reaction time and lower accuracy of responses were also revealed in the PTSD group.

Discussion: The averaging process to obtain the ERN and Pe can be difficult if not enough error trials are present in the data collection and can destroy information in the single trial recordings. To rectify this, we developed a new method of measuring single trial error trials in the time-frequency domain using a wavelet analysis technique. It was shown that this analysis technique was able to differentiate the between the single trial errors of the control group and PTSD group. The differences
in the time-frequency ERN and Pe amplitudes found were posited to occur due to the hypofunctionality of the anterior cingulate cortex in the PTSD group (Woodward et al., 2006).

Conclusion: The time-frequency domain analysis technique can be used to find single trial error differences in the time-frequency ERN and Pe measure of a control and PTSD population.

References


Learning Objective
Learn about a time-frequency domain analysis technique that is used in quantifying error processing in PTSD.

Outline
Introduction – 4 min Methods - 4 min
Results and discussion -4 min Questions and answers – 3 min

Financial Interest: No financial interests for any authors.

Investigation of Theta-Beta Neurofeedback for Adult ADHD:
Session Data (R,C)
Sarah Wyckoff, MA, University of Tübingen, wyckoffsarah@yahoo.com
Kerstin Mayer, MSc, University of Tübingen
Ute Strehl, PhD, University of Tübingen

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: .5

Abstract
Objectives
Attention–Deficit/Hyperactivity Disorder (ADHD) is one of the most common disorders of childhood and persists into adulthood for approximately 5% of the population worldwide (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007). The primary symptoms of ADHD include inattentiveness, impulsivity, and hyperactivity. Analysis of resting state EEG from adults with ADHD has produced a variety of activity patterns in power, coherence, and asymmetry measures, as well as the typical increases in theta/beta ratios seen in pediatric populations (Bresnahan, Anderson, & Barry, 1999; Bresnahan & Barry, 2002; Clarke et al., 2008a). Neurofeedback training is a treatment method that utilizes operant conditioning to reinforce specific EEG activity. In a recent meta-analysis, a large effect size (ES) was found for neurofeedback on impulsivity and inattention in controlled studies and pre- and post-designs (Arns, de Ridder, Strehl, Breteler, & Coenen, 2009). Studies indicated that ADHD children are able to self-regulate cortical activity (Drechsler et al., 2007; Leins et al., 2006; Strehl et al., 2006), which lead to changes in spontaneous EEG activity (Gevensleben et al., 2009; Monastra, Monastra, & George, 2002). However, limited research has investigated the efficacy of neurofeedback as a treatment for adult ADHD. This study will investigate whether adults with ADHD
are able to learn self-regulation of theta-beta activity and if mastery of this skill produces changes in core ADHD symptomatology.

Methods

Adult participants that met DSM-IV criteria for ADHD (combined, inattentive, or hyperactive type), without additional serious physical, neurological, or psychiatric disorders, and a full scale IQ > 80 agreed to receive 30 sessions of neurofeedback training in which theta (4-8Hz) activity was inhibited and beta (13-21Hz) activity was augmented at CZ (referenced to A1, ground A2). Each session consisted of a 2-minute baseline, (3) 7-minute blocks of continuous feedback of theta and beta frequency band amplitudes, and (1) 7-minute transfer block in which amplitude feedback was not presented. EEG amplitudes were calculated for each training task (theta, beta), condition (baseline, feedback, transfer), and assessment point (sessions 2+3; sessions 15+16, session 29+30). Self-assessed symptom questionnaires were administered pre/mid/post training and were correlated to training performance.

Results

This investigation is in progress. Session performance and self-assessed questionnaire data will be examined by a repeated measure ANOVA. The most current data will be presented at the time of the conference.

Conclusion

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

References


Kinderpsychiatr. 55, 384–407.


Learning Objective
Understand and report protocol and disorder specific EEG and behavioral outcomes extracted from a 30-session course of theta-beta neurofeedback for adult ADHD

Assess if adult ADHD patients are able to learn to self-regulate neurofeedback parameters and determine if mastery of this skill correlates with core symptom changes.

Outline
Background on EEG and session data findings in adult ADHD; description of neurofeedback protocol/collection methods: (15 min) Study population demographics, EEG/session data processing methods, and results: (10 min) Discussion of treatment implications, study limitations, and future directions: (5 min)

Financial Interest: I have no significant financial interest or relationship with the commercial supporter(s) or manufacturer(s) of any commercial product or service that is discussed as part of my presentation.

Training Performance and Effects of Slow Cortical Potential Neurofeedback for Adult Attention-Deficit/Hyperactivity Disorder (R,C)

Kerstin Mayer, MSc, University of Tübingen, kerstin.mayer@uni-tuebingen.de

Sarah Wyckoff, MA, University of Tübingen

Ute Strehl, PhD, University of Tübingen

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: .5

Abstract
Objectives

Attention deficit/hyperactivity disorder (ADHD) is characterized by symptoms of inattention, impulsivity, and hyperactivity (Faraone, Biederman, & Mick, 2006). Compared to ADHD in children, only a few studies have investigated ADHD in an adult population, and even less have investigated new forms of treatment such as neurofeedback. Neurofeedback has been applied effectively in various areas, especially in the treatment of children with ADHD (Arns, De Ridder, Strehl, Breteler, & Coenen, 2009; Strehl et al., 2006). This study is designed to assess whether adults with ADHD are
able to learn self-regulation with Slow Cortical Potentials (SCP) neurofeedback training and whether this is correlated with changes in symptomatology.

Methods

Participants received 30 sessions of SCP neurofeedback training at Cz (referenced to A1, ground A2) in which participants had to move the feedback object in the cued direction up for an activation (negativation) and down for deactivation (positivation) 20 times each. Each session consisted of four 8min blocks SCP-training, in which the third block was always a transfer block without visual feedback. For analysis the training performance was assessed by time over and under the midline and differentiation between activation and deactivation trials (see: Mayer, Wyckoff, Schulz, & Strehl (2012) for a full description). Self-assessed symptom questionnaires were administered before, after 15 sessions, and post training and were correlated to training performance.

Results

This investigation is in progress. Training data and correlations between training performance and symptom reduction will be presented at the time of the conference. Conclusion. SCP neurofeedback training has not been previously conducted for adult ADHD and may yield valuable findings about an alternative treatment. Treatment implications, study limitations, and future directions in research will be addressed.

References


Learning Objective

Understand the prospects of slow cortical potential neurofeedback in the treatment of adult ADHD.

Outline

Neurofeedback for Adult Attention-deficit / Hyperactivity Disorder (ADHD): Investigation of Slow Cortical Potential Feedback (20min of background and result presentation, 10min of discussion of treatment implications, study limitations, and future directions)

Financial Interest: No financial interests for any authors.

INVITED PRESENTATION

Video Games, Learning to Learn and Brain Plasticity (R)

C. Shawn Green, PhD, University of Wisconsin, csgreen2@wisc.edu
**Abstract**

As video games have grown in popularity, so too has scientific interest in the behavioral and neural consequences of video game play (Bavelier, Green, & Dye, 2010; Greenfield, 2009). However, although popular culture tends to lump all video games into a single unitary construct (i.e. Are “video games” bad for children?), the scientific evidence strongly indicates that because different games/game genres have different characteristics and processing demands, they, not surprisingly, have different effects on the brain and behavior. The effects of online role-playing games (Martin & Steinkehuler, 2010) differ from the effects of real-time strategy games (Basak, Boot, Voss, & Kramer, 2008), which differ from the effects of pro-social games (Greitemeyer & Osswald, 2010).

My own research has focused primarily on so-called “action” video games (Bavelier, Green, Pouget, & Schrater, 2012; Green & Bavelier, 2012; Spence & Feng, 2010). Over the past decade, action video games have been shown to enhance a wide variety of basic visual, visual attentional, and visuo-motor abilities. Action video game experience leads to better visual resolution and sensitivity, more effective allocation of attentional resources, an enhancement in the ability to process visual information over time, better perceptual decision making, increased visual capacity, and speeds responses to visual stimuli, more effect task-switching, and less susceptibility to distraction (Chisholm, Hickey, Theeuwes, & Kingstone, 2010; Dye, Green, & Bavelier, 2009; Green & Bavelier, 2003, 2006, 2007; Green, Pouget, & Bavelier, 2010; Green, Sugarman, Medford, Klobusicky, & Bavelier, 2012; R. Li, Polat, Makous, & Bavelier, 2009).

These findings stand in contrast with much of the literature on cognitive training, wherein subjects show improvements on the trained task, but demonstrate no transfer of learning to even seemingly highly similar tasks. For example, subjects trained to differentiate between a field of dots moving +3° and -3° from vertical will quickly move from chance to ceiling levels of performance. However, when they are then asked to differentiate between the same dots moving +3° and -3° from horizontal, their performance returns to chance levels (i.e. they have to learn the new task essentially from scratch). Such specificity has been a major obstacle for those who have sought to use behavioral training interventions to treat real-world visual deficits (such as in amblyopia – or “lazy eye”) and thus, the highly general learning brought on by action video game training has lead to a host of new interventions to address such real-world practical problems (R. W. Li, Ngo, Nguyen, & Levi, 2011; McKinley, McIntire, & Funke, 2011; Schlickum, Hedman, Enochsson, Kjellin, & Fellander-Tsai, 2009).

The current focus of my lab is on the question of “why” action video games result in such broad enhancements in cognitive and perceptual function with the roles of arousal, motivation, and reward being of particular interest. However, the overarching hypothesis being tested is that rather than teaching myriad individual skills (i.e. one for each laboratory task that has been examined), what action video games “do” is teach individuals to quickly and efficiently perform new tasks – or in other words, to “learn to learn” (Bavelier et al, 2012; Green et al, 2010).

**References**


Chisholm, J. D., Hickey, C., Theeuwes, J., & Kingstone, A. (2010). Reduced attentional capture in action


Learning Objective
Understand the neural and behavioral effects of action video game experience.

Outline
Not all games are created equal
Effects of action video games – behavioral

Effects of action video games - neural

Contrast with typical learning environments

Action video games and learning to learn

Financial Interest: One patent pending for action-video game based on mathematics training.

KEYNOTE PRESENTATION

Effects of Non-Pharmacological Pain Treatment on Brain States (R,C)
Mark Jensen, PhD, University of Washington, mjensen@uw.edu

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: 1

Abstract
Chronic pain is a significant problem for many individuals, and available treatments are often inadequate. Non-invasive neuromodulatory treatments, such as neurofeedback (NF), have the potential to benefit individuals with chronic pain. However, little research has examined the neurophysiological mechanisms of these treatments. Knowledge concerning these mechanisms is critical for knowing how to develop effective interventions. To address this knowledge gap, 31 individuals with spinal cord injury and chronic pain were given single 20-minute sessions of four neuromodulation procedures (meditation, hypnosis, a NF protocol reinforcing alpha and inhibiting beta activity at T3 and T4, and transcranial direct current stimulation (tDCS)), as well as a single session of sham tDCS, in random order. EEG activity and pain intensity were assessed just before and just after each session. We predicted that (1) the procedures would result in significant decreases in pain, (2) the procedures would result in changes in EEG activity, and (3) pre- to post-session changes in EEG activity would be associated with decreases in pain intensity. Exploratory analyses allowed us to determine whether: (1) any pain-related changes in EEG activity found were global (i.e., similar across many electrode sites) or site-specific; (2) the different procedures had similar or different effects on EEG (indicating similar or different mechanisms, respectively), and (3) pre- session EEG activity predicts treatment response. All of the procedures had immediate effects of EEG activity, two of the procedures (hypnosis and meditation) had significant immediate effects on pain intensity, and a third (tDCS) showed a non-significant trend to decrease pain for participants in neuropathic pain. However, (1) each procedure had different effects on EEG, (2) other than some indication that any change in T3 activity was associated with improvements in pain, the bandwidths and electrode sites associated with treatment response were not consistent across the procedures, (3) the patterns associated with outcome and changes in pain differed as a function of pain type, and (4) different pre-session EEG patterns were associated with treatment response different for each procedure. The findings indicate that (1) different neuromodulatory treatments have different mechanisms for producing pain relief and (2) there is no clear EEG activity pattern associated with greater pain relief – these treatments may work because they produce a change in activity rather than a change in specific bandwidths at specific sites. Given that NF training is known to alter EEG activity, the findings support NF as a potential treatment of refractory pain. If the current findings were to replicate in additional samples, they suggest that NF clinicians treating pain should: (1) consider including the T3 site in training; (2) tailor treatment to each patient’s pain condition; and (3) think in terms of interrupting/changing activity patterns (for pain treatment), perhaps using training
protocols that improve co-morbid symptoms (e.g., cognitive performance, mood, sleep quality) to maximize treatment benefits.

References


Learning Objective
Understand the associations between pain and EEG bandwidth activity and the implications of this for providing neurofeedback pain treatment.

Outline
Present the results of a study examining the impact of neuromodulatory pain treatment procedures on spinal cord-related pain and EEG activity, and the associations between EEG activity and pain intensity.

Discuss the clinical implications of the findings.

Financial Interest: Mark P. Jensen has received consulting fees from Endo Pharmaceuticals, RTI Health Solutions, Covidien, Bristol-Myers Squibb, Schwartz Biosciences, Depomed, Eli Lilly, Pfizer, Merck, and Smith & Nephew within the past 36 months.

Thursday, September 20, 2012

Plenary Room 2

QEEG Subtype Based Neurofeedback Effects on IQ, Attention, Socialization, Communication and Diffuse Tensor Imaging in Students with Autistic Spectrum Disorder (R,C)
Abstract
This paper will describe the use of QEEG to discover which subtype of Autistic (6) and Aspergers (2) to assist in diagnosis and to guide neurofeedback protocol selection. Pre- and post-neurofeedback QEEG and CPT case study data and research studies will be explained for Autistic and Aspergers patients. Previous QEEG and neurofeedback research with ASD will be reviewed.

The authors will present new data from their multi-site study of the effects of Mu based and QEEG Guided Neurofeedback on IQ, socialization, attention, communication and brain imaging (QEEG, Diffuse Tensor Imaging/DTI). The study consisted of both ASD and typical students aged 8-15 who underwent 45 sessions of either Mu Suppression or QEEG Guided Neurofeedback. Dependent variables of TOVA, WASI IQ, behavior rating scales, QEEG and DTI were administered and analyzed pre-post neurofeedback.

To date the post DTI results show greater brain functioning improvement in ASD vs typical students who completed the course of Neurofeedback.

References


Learning Objective
Understand different QEEG subtypes patterns in those with Autism and Asperger and review new research on QEEG Guided and Mirror Neuron Neurofeedback effects with ASD on behavior, communication, socialization and brain imaging.

Outline
EEG and QEEG Subtype patterns with ADD and ASD. 15 min

QEEG Guided Amplitude and Coherence Neurofeedback. 15 min

New research by the presenters in the areas of ASD. 30 min

Financial Interest: Dr. Linden has worked part-time for BSI who provided the QEEG reporting service at a very reduced fee. No conflicts of interest for Dr. Pineda.
STUDENT PRESENTATION

The Dynamics of Brain Networks Involved in Deep Relaxation Regulation Guided by EEG Neurofeedback (R,C)

Sivan Kinreich, MA, Tel Aviv University, sivankin@yahoo.com
Iana Podlipsky, MSc, Tel Aviv University
Nathan Intrator, PhD, Tel Aviv University
Talma Hendler, MD, PhD, Tel Aviv University

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: .25

Abstract

Introduction: A common protocol of EEG-NF training aims to guide people via a closed-loop operation shifting from high-amplitude of alpha (8-14Hz) to high-amplitude of theta (4-7 Hz) oscillations resulting in greater theta/alpha ratio (T/A)1. The induction of such a shift in EEG oscillations has been shown to be useful in reaching a state of relaxation2. However, the clinical implication of this practice in psychiatry remained elusive and considered of relatively low therapeutic yield3,4, possibly due to its widespread cortical representations. The current project aims to use simultaneous acquisition of Functional Magnetic Resonance Imaging (fMRI) and EEG in order to unfold in high spatial and temporal resolutions, respectively the neural modulations of the mental state of relaxation induced via T/A EEG-NF. We used signal characteristics and temporal modulation of theta and alpha for revealing the dynamics of brain network related to the relaxation process. Three main networks were revealed including cortical and deep limbic brain structures. The first neural network involved motor inhibition, the second in managing relaxation and the third in relaxation stabilization. We presume that better understanding of the neural mechanism underlying the T/A NF process might help to optimize the neurofeedback procedure at the individual level and thus will increase its specificity per mental condition.

Methods: 50 healthy subjects participated in a pre-scanning 15 minutes training with eyes closed to apply EEG-neurofeedback for increasing the ratio of theta to alpha. In the 3T MRI scanner subjects followed a similar EEG neurofeedback protocol twice. BrainVoyager, EEG-Lab and at-home software packages were used for preprocessing and analyzing the raw brain signals in correspondence to induced mental states.

Analysis & Results: A data driven algorithm implemented in Matlab (Mathworks, Framingham, MA) employed the criteria of T/A power increase above 1 (“crossover”) for more than a third of the scan to classify each subject’s scan as a responder to the NF procedure, or otherwise as a non-responder. General linear model for the whole brain using the modulating power of theta, alpha and the theta/alpha ratio as predictors was calculated. Defined contrast between responders and non-responders for each of the bands revealed three main networks involved in the mental dynamics of deep relaxation. The first revealed the motor inhibition network (i.e. bi lateral cerebellum, right BA47 and left caudate). The second is related to relaxation management (i.e. dorsal medial prefrontal, Thalamus and putamen) and the third related to the relaxation stabilization (Insula and ventral anterior cingulate).

Conclusions: Simultaneous fMRI during EEG feedback via alpha/theta ratio modulation probed activation variation in brain networks related to the mental process of deep relaxation. The use of the modulation and characteristics of the bands used in the NF procedure enabled identification of the brain networks involved in deep relaxation. Altogether our results clearly demonstrate the advantage in combining EEG NF and fMRI for unfolding the brain mechanism underlying mental states. Methodological and practical aspects of such approach will be further discussed.
References


Learning Objective
Understand the research setup of simultaneous fMRI/EEG neurofeedback.

Learn about the brain mechanisms underlying the mental state of relaxation guided by EEG neurofeedback.

Improve EEG protocol based on new discoveries related to brain networks.

Outline
Understand the research setup of simultaneous fMRI/EEG neurofeedback.

Learn about the brain mechanisms underlying the mental state of relaxation guided by EEG neurofeedback.

Improve EEG protocol based on new discoveries related to brain networks.

Financial Interest: No financial conflicts of interest.

Biomarkers of Neurological and Psychiatric Dysfunctions: Clinical Applications for Diagnosis and Treatment (R,C)

Juri Kropotov, PhD, Institute of the Human Brain, yurykropotov@yahoo.com

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: 1

Abstract
In the first part the paper reviews studies of event related potentials (ERPs) in the normal and diseased brain. It is shown that the ERP negative and positive fluctuations such as N1, mismatch negativity (MMN) and N2 waves as well as various P300 waves could be considered as biomarkers of neurological and psychiatric conditions. Indeed these indexes information flow in the cortex 1) have high test-retest reliability; 2) consistently reflect experimental manipulations in stimulus sensory and emotional modality, probability, behavioral meaning etc. 3) are associated with executive functions such as action selection, action preparation, action suppression and monitoring conflict between competing actions. The ERP waves discriminate a selected psychiatric condition from healthy population with quite large effect sizes. However, majority of ERP waves appear to be not single entities but can be further decomposed into separate components with distinct functional meanings. In the same time, each psychiatric disease appears to be characterized by multiple dysfunctions in complex brain systems, and consequently must be indexed by multiple ERP components obtained in different behavioral paradigms. The second part of the paper deals with
new methodological approaches emerged recently to overcome these hurdles in ERP clinical application. They are: 1) ICA-based ERP decomposition into separate functionally meaningful components, 2) non-parametric methods for mapping generators of ERP components into 3D tomograms; 3) appearance of ERP normative database. The third part of the paper presents our own studies on application of the Human Brain Index (HBI) database for discriminating different psychiatric groups from healthy controls as well for designing protocols of treatment the corresponding brain dysfunctions.

References


Learning Objective
learn about biological markers (MRI, fMRI, PET, EEG, QEEG, ERP) markers of psychiatric and neurological conditions. The focus will be made on recently emerged methodology of decomposing Event Related Potentials (ERPs) into functionally meaningful components.

The participant will learn about new studies of applying this new methodology for diagnosis and treatment of different psychiatric conditions (such as ADHD, schizophrenia, OCD and depression).

Outline
Overview of biomarkers in psychiatry and neurology: MRI, fMRI, QEEG, ERPs (30 min)

Description of a new methodology for decomposing ERP waves into functionally meaningful components. Application of this methodology for diagnosis and treatment of different psychiatric conditions (such as ADHD, schizophrenia, OCD, depression). (30 min)

Financial Interest: I am a co-founder of HBImed, a Swiss company.

Friday, September 21, 2012

Plenary Room 1

The Enhancement of Neurofeedback with a Low Cost and Easy-To-Use NeuroSky EEG Biofeedback Training Device: The MindReflector Protocols (R,C)

Thomas Fink, PhD, Acorn Health Associates, PC, tomefink@comcast.net

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: .5

Abstract
A wireless, dry, inexpensive and easy to use EEG home training device, adapted from the NeuroSky MindWave headset, will be presented, along with proof of concept data, Beta testing reactions and preliminary efficacy findings. Attention will be given to the development of the device and its use of
four power-training protocols developed from the available NeuroSky bandwidth platform. Training and control screens will be illustrated. Finally, the benefits and limitations of the device will be discussed, with focus on its ease of use vs. the temporal, bandwidth and site restrictions of the training device.

References


Learning Objective
Describe the NeuroSky MindWave headset, including its current availability for EEG games (e.g., The Force Trainer) and its underlying EEG bandwidth platform.

Understand the MindReflector adaptation of the MindWave headset, including the development of four potentially relevant training protocols.

Explain the potential benefits, as well as limitations, of the current MindReflector system.

Describe proof of concept data demonstrating the use of the MindReflector device to train changes in relevant bandwidth amplitudes.

Discuss responses and reactions during Beta testing with the four MindReflector protocols.
Outline the usefulness and limitations of home training for an active Neurofeedback practice.

Outline
Description of the development of a home training Neurofeedback system from the NeuroSky MindWave headset – 15 minutes

Discussion of proof of concept data, beta testing responses and a preliminary efficacy study using the MindReflector protocols – 15 minutes

Financial Interest: The author has a business relationship with NeuroSky, the manufacturer of the EEG headset, and he is a 50% partner in MindReflector Technologies, LLC, the developer of the EEG training software that is used with the NeuroSky MindWave headset.

Functional Disconnections in Trauma and Abuse: From Victimized Children to Murderers on Death Row (R,C)

David Kaiser, PhD, Sterman-Kaiser Imaging Laboratory, Inc., davidkaiser@yahoo.com

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: .5

Abstract
Identification of the default mode network (resting, inward focus) and the role of the posterior cingulate in this network has been one of the major accomplishments of functional neuroimaging. Posterior cingulate cortex (PCC) disturbances are observed in fMRI recordings of individuals suffering PTSD, and similar PCC disconnections are seen in individuals who have a history of sexual, emotional, and/or physical abuse. Twelve death row inmates with a history of abuse and witnessing violence were shown to have PCC disconnections in their EEG, along with primary auditory and right dorsolateral cortex (Brodmann area 44R), an area involved in monitoring intentions and emotions, among other functions. We find the same disconnections in children with chronic history of abuse. The role of the posterior cingulate in emotional functioning will be discussed in this context, and two cases where children underwent successful neurotherapy to treat their issues will be discussed along with its implementation in real-time in Brainmaster Avatar.

References


Biofeedback training can be used to control autonomic input to the cardiovascular system. It has been well-established in our laboratory and others that heart failure is accompanied by hyper-activation of the sympathetic nervous system, and decreasing sympathetic input with a beta blocker or left ventricular assist device improves clinical status and also reverses cellular and molecular alterations associated with heart failure. We hypothesized that heart failure patients could be trained with biofeedback and that this method of regulating the sympathetic nervous system would also produce myocardial remodeling in the direction of recovery. In order to test this hypothesis, end-stage heart failure patients at the Cleveland Clinic were enrolled in a research study which included an initial assessment of psychophysiological reactivity to mental stress, six sessions of biofeedback-mediated stress management training with a certified biofeedback therapist, and a final assessment of psychophysiological reactivity to mental stress. Quality of life was also evaluated before and after biofeedback training using the SF-36 and Kansas City Cardiomyopathy questionnaires. Plasma norepinephrine and six minute walk distance were measured before and after biofeedback training, as a marker of clinical status. After biofeedback training, at the time of heart transplantation, explanted hearts were transported to the laboratory to study the heart failure phenotype. Left ventricular trabecular muscles were dissected and studied in a tissue bath, measuring the inotropic response to sympathetic stimulation. A single dose of isoproterenol, a synthetic norepinephrine analogue, was used as an index of sympathetic nervous system recovery. Beta adrenergic receptors on myocardial cell membranes were also measured, using radioligand binding and Scatchard analysis. Preliminary data suggest that biofeedback produces remodeling of the heart failure phenotype, in the direction of normal, similar to what we have previously shown in hearts supported with a left ventricular assist device.

References


Learning Objective
Explain the role of the autonomic nervous system in regulating cardiovascular function.

Discuss the regulation of the sympathetic nervous system in patients with heart failure.

Clarify the potential for parasympathetic regulation in heart failure.

Describe the role of biofeedback in treating patients with heart failure.

Outline
Ten minutes – introduction to cardiovascular disease

Ten minutes – role of the autonomic nervous system in cardiovascular disease

Ten minutes – rationale and design for using biofeedback in patients with cardiovascular disease

Ten minutes – results of biofeedback studies in patients with cardiovascular disease

Ten minutes – summary, conclusions, implications of our studies

Financial Interest: No financial interests.

STUDENT PRESENTATION

Investigation of Unspecific Placebo Effects in Slow Cortical Potential Neurofeedback for Adult Attention-Deficit/Hyperactivity Disorder (ADHD) (R,C)

Kerstin Mayer, MSc, University of Tubingen, kerstin.mayer@uni-tuebingen.de
Sarah Wyckoff, MA, University of Tubingen
Ute Strehl, PhD, University of Tubingen

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: .25

Abstract
Objectives:
Neurofeedback has been applied effectively in various areas, especially in the treatment of children with ADHD (Arns, De Ridder, Strehl, Breteler, & Coenen, 2009). However, unspecific treatment effects like expectations and patient-therapist relationship may have an influence on therapy outcome. These unspecific effects are usually hard to control for without placebo or waiting groups (Gevensleben, Rothenberger, Moll, & Heinrich, 2012). This study investigates Slow Cortical Potentials (SCP) neurofeedback training for adult attention deficit/hyperactivity disorder (ADHD) and its possible unspecific effects assessed via a self-rated placebo questionnaire (Vollmann, Hautzinger, & Strehl, 2009).

**Methods:**

Twenty adult participants with ADHD received 30 sessions of SCP neurofeedback training at Cz (referenced to A1, ground A2) (see Mayer, Wyckoff, Schulz, & Strehl (2012) for the methods). Every fifth session participants filled in the German questionnaire “Fragebogen zur Erfassung relevanter Therapiebedingungen” (FERT) which is a self-rated questionnaire to assess relevant treatment conditions, patient expectations, and patient-therapist interactions (Vollmann et al., 2009). The FERT was analyzed for expectation changes over the time course of the neurofeedback training, as well as, used as a covariant in the analysis of training performance and symptom changes.

**Results:**

This investigation is in progress. Expectation changes over the time course of the feedback and correlations between FERT and training performance, as well as, symptom changes will be presented at the time of the conference.

**Conclusion:**

Possible placebo effects have always been a concern in neurofeedback. Correlations may yield valuable findings about the impact of unspecific effects on neurofeedback. Study limitations, and future directions in research will be addressed.

**References**


**Learning Objective**

Understand the relation of slow cortical potential neurofeedback in the treatment of adult ADHD and unspecific/placebo effects.

**Outline**
Investigation of Unspecific Placebo Effects in Slow Cortical Potential Neurofeedback for Adult Attention-Deficit / Hyperactivity Disorder (ADHD) (10min of background and result presentation, 5min of discussion of treatment implications, study limitations, and future directions)

Financial Interest: None for any authors.

KEYNOTE PRESENTATION

An Evolutionary Approach to Return to Health (R,C)
Erik Peper, PhD, San Francisco State University, epeper@sfsu.edu

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: 1

Abstract
We are biologically, emotionally and socially much more the prehistoric mammal than the modern 24/7 human being. Many of the factors that determine health and illness are the result genetic mutations which fostered reproductive fitness. Whatever fostered reproductive fitness prevailed. Our biology and psychology patterns still reflect this evolutionary past. For most of our past we were “prey,” thus ongoing vigilance enhanced survival. Similarly, our digestive system reflects our million year old history of eating predominantly leaves, nuts, tubers, etc and not the recent processed foods. Our biological rhythms were synchronized by natural light patterns and not by electric lights or computer screens. By recognizing and integrating our evolutionary biological and social roots and combining this with the teaching self-regulation skills, numerous illnesses may be reversed and health improved.

References
Peper, E. & Lin, I-M. (in press). Increase or decrease depression-How body postures influence your energy level. Biofeedback

Learning Objective
Appreciate an evolutionary perspective of health and healing.
Recognize the damage of stress immobilization syndrome.
Recognize the importance of movement to increase energy and decrease pathology.
Understand the important role of somatic/body factors affecting and being effected by neurofeedback.

Outline
Evolutionary perspective of health
Options of how to reverse some chronic disorders
Self-care approaches to mobilize the immune system in the treatment of cancer

Financial Interest: No financial interests to report.

KEYNOTE PRESENTATION
Functional Neuroanatomy of Emotions and Stress (R)
Israel Liberzon, MD, University of Michigan, lizerzon@med.umich.edu

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: 1

Abstract
The emergence of affective neuroscience advanced our understanding of neuro-circuits involved in emotional responses. Functional neuroimaging methods now allow to study complex brain function in humans in vivo. Animal studies helped to elucidate the role of subcortical regions like amygdala, hippocampus and nucleus accumbence in fear and reward behavior, and functional neuroimaging expanded our knowledge of the role of cortical regions in complex human emotions. Indeed, abnormalities in the functioning of many of these regions have now been reported in disorders characterized by abnormal emotional responses like depression and PTSD. In parallel, decades of stress studies had outlined the function of the main stress response system – hypothalamo, pituitary adrenal (HPA) axis. Interestingly, abnormalities in HPA function have been also demonstrated in the same psychiatric conditions. Until recently however the precise link between the emotional and the stress response systems had not been established. In the last decade, studies integrating fMRI and neuroendocrine methods emerged. They allowed identification of key cortical regions like insula, medial prefrontal cortex (mPFC) and anterior cingulated, that link emotional and stress responses, and are likely critical for the understanding of the pathophysiology of stress.

Learning Objective
Understand the principles of functional neuroimaging methodology.
Identify key brain regions and circuits involved in emotional responses and stress response.
Understand the application of functional brain imaging methods in study of psychiatric disorders.

Outline
Functional neuroimaging methods - 10-15 minutes
Brain circuits of emotional and stress responses - 20-25 minutes
Functional imaging in study of psychiatric disorders (PTSD) - 15-20 minutes

Financial Interest: No financial conflicts of interest.

Friday, September 21, 2012

Plenary Room 2

Crossing The Bar: Neurofeedback as an Adjunct Therapy to Addiction Recovery (R,C)
Judith Miller, Courage to Change Addiction Recovery Ranch, redfeather7@earthlink.net

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: 1

Abstract
The purpose of this paper is threefold:

To present a historical perspective on the heralding of addiction as a social/moral disease to state-of-science-based evidence that addiction is a disease of the brain that can be managed and treated with
the use of holistic therapies including neurofeedback therapy

To present an analytical case study that reveals neurotherapy as a promising adjunct therapy for addiction solutions

To present an analytical synopsis regarding future drug-free applications of a neurological approach for sustainable addiction recovery. The research reported in this paper is based on an eight month study (2011 to 2012) of 100 addicted clients at an addiction recovery program. Following neurotherapy 100% of the subject's experienced profound relief from the symptoms of addiction suffered prior to treatment.

Power Point Presentation – The History of Addiction Recovery – a 100 year overview of how addiction therapy began to the present state-of-the-science treatment regime that includes neurofeedback as an adjunct therapy for sustainable addiction recovery.

References


Learning Objective
Understand the dynamics of addiction as a brain disease.

Learn about the history or evolution of addiction treatment practices from the early treatment attempts to current State of the Science.

Learn the role of neurofeedback as an adjunct therapy for successful addiction recovery.

Outline
The concept of addiction as a brain disease

History of Addiction Treatment

The Role of Neurofeedback in the treatment of addiction

Financial Interest: No financial interest whatsoever with anyone or any company.

STUDENT PRESENTATION

An Event-Related Potential Study of Visual Spatial Attention Deficits in Autism (R)

Guela Sokhadze, BS, University of Louisville, g0sokh01@louisville.edu
Lonnie Sears, PhD, University of Louisville
**Results:**

Reaction time (RT) analysis showed a Congruence X Group effect \(F=7.14, p=0.011\), in particular the ASD group had similarly slower RT both in valid and invalid pre-cued conditions, while controls responded faster to correctly prompted targets. Accuracy of responses was lower in the ASD group \(F=7.88, p=0.008\), mostly due to more omission error rate \(F=6.17, p=0.017\). Midline frontal N100 component yielded a marginal Position X Congruence X Group interaction \(F=4.14, p=0.049\), where ASD group had more negative N100 amplitude during diagonal target condition regardless of congruence of cues. Furthermore we found a significant Position X Congruence X Hemisphere X Group effect \(F=4.52, p=0.040\) where above effect was more pronounced at the right hemisphere. Amplitude of the midline frontal N200 component showed a Position X Congruency X Group interaction \(F=4.13, p=0.045\). The group differences of peak latency for both N100 and N200 components were not significant. The centro-parietal P300 (P3b) component showed between group differences at the midline \(F=5.38, p=0.026\) and at the left hemisphere \(F=4.80, p=0.035\) in invalidly
cued diagonal target condition and was significantly prolonged in the ASD group. Amplitude of the LRP in the ASD group was lower and delayed as compared to the control group (ps<0.05).

Discussion and Conclusions:

Most of ERP differences were observed at the frontal sites thus pointing at the possible frontal executive deficits in autism. Children with autism had more impaired responses to diagonal targets requiring more spatial orienting capacity. Of particular interest for the future studies are frontal hemispheric differences present at the pre-attentive early processing stages (N100), and less discrimination between correctly and incorrectly cued targets at the later stages of processing. This was manifested in the enhanced frontal N200 component resulting in a delayed cognitive P3b potential (Polich & Herbst, 2003) in the autism group. We have found that using a cued Posner’s spatial attention test and comparing the autistic patients behavioral performance and ERPs can be a very informative approach to understand the mechanisms of spatial orienting impairments and motor act preparation deficits typical for autism.

References


Learning Objective
Learn about spatial attention deficits in autism, and about assessment of spatial attention in the Posner cued attention test in a autism and typically developing individuals using event-related potential technique.

Outline
Introduction – 4 min

Methods - 4 min

Results and discussion -5 min

Questions and answers – 2 min

Financial Interest: No financial interest or conflict to report.
Comparison of the Effectiveness of Z-Score Surface/LORETA 19-Electrodes Neurofeedback to Standard 1-Electrode Neurofeedback (R,C)

J. Lucas Koberda, MD, PhD, Tallahassee NeuroBalance Center, JLKoberda@yahoo.com

Andrew Moses, Tallahassee NeuroBalance Center

Paula Koberda, Tallahassee NeuroBalance Center

Laura Koberda, Tallahassee NeuroBalance Center

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: 1

Abstract
The effectiveness of Z-score surface and low resolution electromagnetic tomography analysis (LORETA) Neurofeedback (NFB) has been retrospectively compared to standard (1-electrode) NFB treatment. This is multi-case report based on the analysis of 40 patients from a solo neurology practice who reported either improvement of symptoms with Z-score NFB or completed at least 10 Z-score (surface/LORETA) sessions. The analysis included subjective (self-reported) and objective (QEEG, computerized neuropsychological testing) response to NFB therapy. Quantitative electroencephalography (QEEG) and computerized neurocognitive testing (in selected patients) were completed before and after NFB treatment and analyzed for any major changes in frequency bands expression or an improvement in a cognitive function. Z-score surface/LORETA NFB patients were divided into four groups including patients suffering from headaches (frequently with anxiety and/or chronic pain), cognitive-, behavioral- problems as well as focal neurological disorders (stroke, epilepsy). The average Z-score NFB number of sessions per patient was 9 (range between 3 to 24). Patient’s analysis revealed 95% subjective improvement rate and 62.5% objective QEEG improvement rate after Z-score NFB therapy. These results retrospectively were compared to 25 patients who were treated in the same practice using a standard 1-electrode NFB technique and completed in at least 20 sessions with 84% of subjective improvement rate and 75% objective QEEG improvement rate. Above results indicate similar effectiveness of Z-score NFB and 1-electrode standard NFB in achieving positive response to EEG-biofeedback. However Z-score NFB seems to have higher potency since many patients required fewer sessions to achieve a desirable subjective response. Therefore, Z-score NFB application may contribute to increase patient’s compliance and may offer a more cost effective treatment. Several cases of marked improvement with Z-score NFB treatment will be discussed including a patient with intractable epilepsy and subsequent complete normalization of epileptoform EEG with NFB therapy. Also, two cases of major cognitive enhancement including improved verbal function and information processing speed will be presented.

References


Learning Objective
Become familiar with application and effectiveness of Z-score surface/LORETA 19-electrodes NFB in the
treatment of neuro-psychiatric symptoms. The retrospective comparison to 1-electrode standard NFB will be made.

**Outline**

Z-score surface/LORETA 19-electrodes NFB-10 min.

Results of treatment with surface/LORETA 19-electrode NFB- will include the analysis of 40 patients with different neuro-psychiatric problems.-20 min.

More detailed presentation of Z-score surface/LORETA NFB cases (2-3 cases)-including case of successful cognitive enhancement and epilepsy treatment.-10-15 min.

The retrospective comparison of the effectiveness of Z-score surface/LORETA NFB to previously analyzed group of 25 patients treated with 1-electrode standard NFB (from the same neurological practice).-15 min.

**Financial Interest:** No financial relationships.

**Combining Neuroeconomics with LORETA Biofeedback to Improve Self-Control and Promote Healthy Behavior (R)**

*Jordan Silberman, MA, University of Rochester School of Medicine and Dentistry,*
*Jordan_Silberman@urmc.rochester.edu*

*Miron Zudkerman, PhD, University of Rochester School of Medicine and Dentistry*

*Peter Manza, BA, University of Rochester School of Medicine and Dentistry*

**Credits:** CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: .5

**Abstract**

**Background:**

What occurs in the brain when a person foregoes a decadent dessert, and instead chooses a food that is healthy but bland? The field of neuroeconomics has begun to answer this question. Neuroeconomists have elucidated some of the neurophysiology that allows people to save rather than spend, to choose the gym over the couch, and to eat carrots rather than cookies.1-9 Researchers have identified neural mechanisms that may underlie self-control, but they have not applied this knowledge to develop tools for improving self-control ability. LORETA biofeedback (LB) may allow us to harness neuroeconomics findings in order to develop interventions for improving self-control. We define self-controlled behaviors simply as those in which large delayed rewards are chosen over smaller immediate rewards. Building on the neuroeconomics literature, we have developed an LB protocol designed to strengthen the neurophysiology underlying self-control. This protocol may improve self-control ability, and thereby increase an individual's capacity to exhibit heath behaviors for which self-control is required (eg, dieting and exercising). LORETA biofeedback may therefore offer a novel approach to health behavior promotion.

**Methods:**

A randomized, controlled, single-blind study was conducted to assess the effects of the LB protocol on self-control. Subjects attended 4 study sessions on 4 consecutive days. A self-control task involving food choice was administered during the first and last sessions. Subjects rated a series of foods on health and taste; subjects then chose between foods they had rated as bland-but-healthy and foods rated as tasty-but-unhealthy. Self-control was defined as choosing bland-but-healthy foods over tasty-but-unhealthy foods. Between the two administrations of the self-control food choice task,
treatment group subjects completed the LB protocol that was designed to improve self-control. This protocol, which was tailored for each subject, involved targeting the following regions of interest: right dorsolateral prefrontal cortex (dLPC), left dLPC, dorsal anterior cingulate cortex (dACC), and left supplementary motor area (SMA). Control subjects completed an LB protocol that was expected to have no effect on self-control performance. Logistic multilevel modeling was used to compare changes over time in self-control performance of the treatment group to those of the control group.

Results:

Eighty-five percent of sessions have been completed; results from the full study sample will be reported at the conference presentation. In our current dataset, a significant time X condition interaction is observed (p < .01). Consistent with predictions, self-control performance of the control group significantly decreased over time (p < .01), while that of the treatment group did not change over time (p > .5).

Discussion:

Preliminary results suggest that LB may be useful for preventing reductions over time in self-control; LORETA biofeedback may therefore offer a novel approach to health behavior promotion. Additional research is needed to determine whether or not these results—which were observed solely in the laboratory—can be replicated in real world contexts.

References


Learning Objective

Describe the current evidence regarding application of LORETA biofeedback to health behavior promotion.
Abstract
Introduction:

Besides being a part of what makes life worth living, positive emotions such as joy and happiness have been shown to have numerous benefits (Lyubomirsky, King & Diener, 2005). Resisting the common cold and flu are linked to the tendency to experience positive emotion (Cohen et al., 2003; 2006). Positive emotional style is associated with lower rates of stroke (Ostir et al., 2001) and better coronary recovery (Middleton et al., 1996). The lack of joy was found to be one of the most important symptoms linked with risk of depression after age 60 (Hein et al., 2003). Higher remission in major depressive disorder was found when engaging in positive psychotherapy than with regular therapy or regular therapy plus medications (Seligman, Rashid, & Parks, 2006). In addition to studies on benefits of emotion, individual differences regarding personality and emotional processing should be considered in behavioral studies, rather than attributing them to "statistical noise" (Vuoskoski & Eerola, 2011; Schiffer et al., 2007). Left frontal brain areas are activated during emotions that are characterized by approach, such as joy, happiness, interest, and that the right frontal areas are activated by avoidance or withdrawal emotions such as disgust or distress (Davidson et al., 1990). However, individual differences are seen in left-sided frontal EEG asymmetry, indicative of greater trait approach motivation (Master et al., 2009). The following studies will discuss personality and hemispheric differences in the context of the basic emotion joy.

Methods:

Study 1. After providing informed consent, a non-clinical sample of twenty-seven university students underwent continuous EEG recording while they performed two tasks designed to evoke joy: a) read self-referential statements of benefit, and b) recall a personal experience that brought them maximal joy. After baseline and task EEG recordings, participants also completed open-ended reports, health symptoms inventory (CHIPS) and optimism/pessimism scale (LOT-R), and ratings of self-referential statements. EEG source localization using sLORETA was performed and comparison of the self in experience of joy condition to baseline was made using all voxel-by-voxel t-tests. Voxels of significant difference were mapped onto a Montreal Neurological Institute (MNI) atlas containing 6,329 5mm voxels.

Results:

Correlations of significant differences of recall task to eyes-open baseline discussed with regard to regions of interest and frequency bands.

Study 2. After providing informed consent, a non-clinical sample of one hundred five university
students completed a packet of instruments including measures of personality (NEO Five Factor Inventory), gratitude (GQ-6), joy, life and satisfaction. Results: Significant correlations with joy and personality facets will be discussed.

References


**Learning Objective**
Describe some neural regions, personality differences, and benefits associated with positive affect vs negative affect.

**Outline**
So why study positive affect? (3-5 min.) - Depression was estimated to cost 83.1 billion dollars in 2000 (Greenberg, et al., 2003), and lack of joy was found to be one of the most important symptoms linked with risk of depression after age 60 (Hein, et al, 2003). - Through cross-sectional studies, happy people appear to be more successful in work, health, and relationships, and through longitudinal studies happiness precedes important outcomes and indicators of thriving (Lyubomirsky, King & Diener, 2005).

Treatment/intervention benefits: - listening to self-selected joyful music associated with absolute increase in brachial artery diameter of same magnitude seen with aerobic exercise and also with statin drug therapy commonly given to decrease cholesterol levels (Miller, Mangano, Beach, Kop, & Vogel, 2010) - higher remission in major depressive disorder when engaging in positive psychotherapy than with regular therapy or regular therapy plus medications (Seligman, Rashid, & Parks, 2006). - to effect a sustained change in happiness, change your actions, not circumstances (Sheldon & Lyubomirsky, 2006) - tendency to experience positive emotion linked to resisting the common cold and flu (Cohen et al., 2003; 2006), lower rates of stroke (Ostir et al., 2001), better coronary recovery (Middleton et al., 1996)

Selected Neurological and Electrophysiological Investigations of Affect and Hemispheric Asymmetry (5-8 min.) - Individual differences regarding personality and emotional processing should be considered in behavioral studies, rather than attributing them to "statistical noise" (Vuoskoski & Eerola, 2011; Schiffer et al., 2007).

- Hemispheric differences, left-sided frontal EEG asymmetry, indicative of greater trait approach motivation (Master et al, 2009) - Davidson and colleagues' work (1990) - left frontal brain areas are activated during emotions that are characterized by approach, such as joy, happiness, interest, and that the right frontal areas are activated by avoidance or withdrawal emotions such as disgust or distress.

- Dawson and colleagues (1992) looked at frontal lobe activity via EEG in infants with mothers presenting with depressive symptoms and found opposite pattern of frontal activations during approach and avoidance emotions, linked an idea presented by Tronick and Gianino (1986) about infants exposed to maternal depression

Results of current research (S. K. Fischer, 2011-2) Personality and Neural correlates of self experience of joy (10 min)

Study 1: - Non-clinical convenience sample of 15 university students - Continuous EEG recording during 2 tasks: envisioning a recalled personal experience of maximal joy and rating statements intended to evoke joy - Health, optimism, pessimism and affect ratings recorded - Correlations of significant differences of recall task to eyes-open baseline discussed Study 2: - Non-clinical convenience sample of 105 university students - Personality, joy, gratitude, life satisfaction, depression, anxiety measured - Significant correlations discussed Summary and Implications 5 min
Financial Interest: No financial interests or relationships.

An EEG Interface for Continuous Performance Testing and Event-Related Potentials (R,C)
Andrew Greenberg, MSEE, TOVA Company
Chris Holder, MA, TOVA Company
Thomas Collura, PhD, BrainMaster Technologies, tomc1@brainm.com

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: .5

Abstract
A new interface has been developed between the Test of Variables of Attention (T.O.V.A.) continuous performance test and EEG systems. This interface supports the real time capture of events by EEG systems of the T.O.V.A. test's stimuli and subject responses. With this combination, it is possible to capture event-related brain activity during the continuous performance test as part of a standardized assessment. The value of the T.O.V.A. has been established in assessing attention, and its usefulness in appraising the effects of neurofeedback has been explored. However, no studies including simultaneous EEG and T.O.V.A. performance have yet been published. Initial results with this new capability demonstrate the feasibility of identifying specific brain processes associated with task performance. When combined with live sLORETA-based localization, specific brain locations can be further monitored, permitting the identification of detailed processes related to specific task activities. Results demonstrating this connection and these relationships will be described in this talk.

References


Learning Objective
Describe how continuous performance tasks (CPT) are conducted. Describe how brain function is related to CPT performance.

Outline
The TOVA continuous performance task system, how it works.

How EEG can be added to the TOVA to improve continuous performance assessment. Case examples of live EEG taken during performance of the TOVA illustrating brain function.

Financial Interest: Andrew Greenberg and Chris Holder are employees of The TOVA Company. Thomas Collura has a financial interest in BrainMaster Technologies Inc.

Randomized, Controlled, Cross-Over Research of Performance Brain Training™ Effects in Elite College Golfers (R,C)

Noel Larsen, MA, Neurotopia, Inc., noel@neurotopia.com
Leslie Sherlin, PhD, Neurotopia, Inc., lesliesherlin@mac.com
Ashley Baker, MA, Neurotopia, Inc.
Jeff Troesch, MA, Neurotopia, Inc.

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: .5

Abstract
Introduction:

Over the years there has been a continued interest in the use of neurofeedback to enhance sports performance (e.g., Hammond, 2007; Vernon, 2005) and some have suggested this may be the next frontier of peak performance training (Harung et al., 2011). Despite the appeal, few studies have directly tested how neurofeedback influences sport performance outcomes (Arns, Kleinnijenhuis, Fallahpour, & Breteler, 2007; Landers et al., 1991). There is also limited understanding regarding what training protocols would best serve an athlete population; to date, no standardized training protocol exist for peak performance neurofeedback training (Vernon, 2005). Previous research has relied strictly on personalized electroencephalograph (EEG) profiles (Arns et al., 2007) or on theory drawn from EEG profiles of experts in the respective field (Landers et al., 1991). Each reported a certain degree of success, signifying that a combination of both approaches may be maximally effective. The aim of the current study was multifaceted. First to describe the EEG profiles of Division I National Collegiate Athletic Association (NCAA) golfers and second to understand to what degree Performance Brain Training™ (a specific neurofeedback training paradigm with protocols based on the NeuroPerformance Profile™) could alter the EEG and its reflection in a subsequent NeuroPerformance Profile™. Third, and perhaps most relevant to sport audiences, to demonstrate the effects of Performance Brain Training™ on sport performance related outcomes.

Method:

Participants included 16 Division I Pacific Athletic Conference (PAC-12) golfers (n = 6 females) ranging in age from 18 to 22 years of age (M = 19.81 years) randomly divided into two groups. Quantitative electroencephalographic (QEEG) data was collected and a NeuroPerformance Profile™ was calculated prior to randomization (time point 1). Both groups continued as normal with team practice, tournament play, and sport related coaching. Group 1 additionally received 20 sessions of Performance Brain Training™ using protocols based on their weakest NeuroPerformance Profile™
scale conducted over the course of 5 weeks with 2 to 3 sessions per week. Group 2 did not receive Performance Brain Training™ during this time. At time point 2, a second QEEG recording conducted and another NeuroPerformance Profile™ was calculated for all participants. Over the subsequent five weeks both groups maintained normal team activities while Group 2 also completed 20 sessions of Performance Brain Training™. Group 1 did not complete any Performance Brain Training™ sessions during this time. Following this training period (time point 3), all golfers underwent the third and final QEEG recording and a NeuroPerformance Profile™ was calculated. Sports performance data (greens in regulation [GIR], fairways in regulation [FIR], putting and score averages) was collected at each time point in the study. Group central tendencies were calculated from time point 1 measures. Additionally, MANOVA was implemented to compare QEEG, NeuroPerformance Profile™, and all performance data from time point 1 to time point 2 and from time point 2 to time point 3 for each group.

Results:

Analyses will be conducted once the trial comes to an end the final week of May. Findings of the previously described analyses will be presented illustrating 1) a composite description of the EEG activity in Division I golfers, 2) changes in EEG measures and 3) changes in performance outcomes following Performance Brain Training™.

Discussion:

This research is an initial investigation of the cortical activity of elite amateur golfers during baseline and a challenge task. Moreover, this study has illustrated how Performance Brain Training™ can influence EEG measures and specific golf performance outcomes. These results may inform future research in the field of peak performance neurofeedback training.

References


Learning Objective
Identify the relevance of neurofeedback training to sport performance application.

Outline
Basic background in neurofeedback for sport
INVITED PRESENTATION

Neurotoxins: Effects on Brain and Behavior and Therapy (R,C)
David Cantor, PhD, Psychological Sciences Institute, PC, cantor@psycscienceinst.com

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: 1

Abstract
Since the middle of the 20th century, the rapid proliferation of chemical compounds used in the environment, medical treatment, and alternative methods used for food production have led to grave concerns about the potential effects of these compounds and their resultants on the efficiency of the electrochemical workings of the human brain and its development. Some of these concerns were published in the popular media in mid-20th century by such authors as Rachel Carson in her now famous nonfunctional work “Silent Spring” and from publications resulting from the United Nations Environment Programme initiated by U Thant, 3rd Secretary-General of the United Nations. However it wasn’t until the rise of large-scale lawsuits against the offending industries coupled with the near epidemic rise of health problems in the late 1980’s and into the 21st century involving neurocognitive and neuromuscular disorders that there has been a global interest in this domain. This presentation will provide an overview of the types of neurotoxic intrusions currently present that are likely involving health risks in general with their associated economic factors but on the functionality of humans to be adaptive and optimally functional in an increasing complex and demanding world for cognitive performance efficiency. Examples of studies will be provided to illustrate the effects of exposure to metallotoxins and lipophilic toxins and the cascading changes of biochemical processes ultimately impacting on physiological efficiencies and alterations in the ways cell assemblies in the brain are modified leading to alterations in behavior and adaptive capabilities. Discussion will also be provided on how these effects impact significantly in the ways we assess and treat conditions of aberrant human behavior but also have implications on the potential limits with neurotherapies attempting to compensate for central nervous system functional inefficiencies.

References


Learning Objective
Identify basic elements or compounds which have been identified to affect CNS functioning and behavior.

Have an understanding about how neurotoxic agents can manifest their effects adversely in the course of a lifetime.

Recognize the manner that body burden levels of neurotoxic agents can influence qEEG findings and thwart Neurofeedback protocols.

Financial Interest: I am an owning partner of BrainDx, LLC which produces analytic software and report generation of electrophysiological data and am involved in ongoing forensic cases involving cases in litigation due to toxin exposure.

KEYNOTE PRESENTATION

Neurofeedback Training Induces Changes in Grey and White Matter (R)

Mario Beauregard, PhD, University of Montreal, Mario.beauregard@umontreal.ca

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: 1

Abstract
We demonstrated some time ago that increasing Beta1 band through neurofeedback training (NFT) can enhance activity in brain regions involved in various attentional processes (Beauregard et al., 2006; Lévesque et al., 2006). One objective of this structural magnetic resonance imaging (MRI) study was to investigate whether a NFT protocol designed to improve attention might induce changes in grey matter volume (GMV) in areas known to be implicated in attention. Another objective was to explore whether such a NFT protocol might lead to alterations in white matter tracts involved in attention processing.

Thirty university students (M: 22.2; SD: 2.4) with no history of neurological or psychiatric disorders were recruited. Participants were randomly assigned to an experimental group (EXP, NFT; n=12, M: 22.4; SD: 1.6), a sham group (SHAM, to control for a possible placebo effect; n=12, 9 M: 22.0; SD: 3.1), or a control group (CON, to control for the passage of time; n=6, 3 M: 20.7; SD: 1.0). NFT was conducted over a period of 13.5 weeks for a total of 40 sessions. Participants in the EXP group were trained to enhance the amplitude of their beta 1 waves in the right hemisphere. Electrodes were placed at F4 and P4. MRI data were acquired one week before (Time 1) and one week after (Time 2) NFT. Regional changes in GMV were analyzed using voxel-based morphometry (VBM). As for white matter, a diffusion tensor model was fitted to diffusion tensor imaging (DTI) data to produce whole
brain maps of fractional anisotropy (FA) that were compared between the two time points using tract-based spatial statistics (TBSS) (Smith et al., 2006). The attentional skills of all participants were assessed at Time 1 and Time 2 using the Integrated Visual Auditory continuous performance test (IVA).

In the EXP group, the scores on the IVA Full Scale Attention Quotient (which is based on measures of both visual and auditory attention) significantly increased at Time 2, compared to Time 1 (P < 0.005). Scores on auditory attention were also significantly higher (P < 0.005) following NFT. For participants in the SHAM group, scores on visual attention were greater (P < 0.005) at Time 2 relative to Time 1. No difference in attentional performance was noted at Time 2, compared to Time 1, for members of the CON group. In other respects, a significant (P < 0.001 uncorrected) grey matter volume increase was found in the EXP group, at Time 2 relative to Time 1, in a number of cortical areas located in the right hemisphere [RH] (inferior, middle and superior frontal gyri; inferior parietal lobule; inferior temporal gyrus) and left hemisphere [LH] (inferior and superior frontal gyri; inferior and superior temporal gyri; superior parietal lobule). With regard to white matter, significant increases in FA were measured in the superior longitudinal fasciculus (left hemisphere [LH], P < 0.0001), inferior longitudinal fasciculus ([LH], P < 0.005), anterior limb of the internal capsule (LH, P < 0.0005), anterior corona radiata (right hemisphere [RH], P < 0.005), cingulum ([RH] and [LH], P < 0.0001), and corpus callosum ( genu: P < 0.005; body: P < 0.001; splenium: P < 0.0005). No change in grey and white matter was noted for members of the SHAM and CON groups.

These findings suggest that NFT can induce changes in brain regions implicated in attention. Our findings also indicate that NFT can produce modifications in white matter tracts involved in attentional processes.

References


Learning Objective
Learn about the structural brain changes (grey matter and white matter) induced by neurofeedback training.

Outline
I will discuss the results of a magnetic resonance imaging study (MRI) recently performed by my research team. The main objective of this study was to investigate whether neurofeedback training can lead to structural changes in the brain related to grey matter and white matter. First, I will start the lecture by presenting the concept of neuroplasticity (10 min). Then, I will present the objectives of the study (5 min) and describe the methods used in this investigation (10 min). Next, I will present the results (15 min). Finally, I will discuss these results and offer a few concluding remarks (15 min).

Financial Interest: The study was supported by a grant from the Foundation Denis Guichard (Paris, France).
Plenary Room 2

Heart - Brain Connections Neuroanatomy Underlies the Effectiveness of Interventions that Combine Neurofeedback with Biofeedback (C)

Lynda Thompson, PhD, ADD Centre, addcentre@gmail.com
Michael Thompson, ADD Centre, addcentre@gmail.com

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: 1

Abstract
Heart rate variability training appears to have direct effects on many of the same basic neural structures that are also directly influenced by EEG biofeedback training. These include the thalamus, anterior cingulate cortex, amygdala, and hypothalamus, to name a few. Combining EEG biofeedback / neurofeedback (NFB) with peripheral biofeedback (BFB) appears to have effects not only on the neural network(s) concerned with emotions (the affect network) but also on the executive, salience and default networks and their associated functions. In our experience, attention and concentration, as well as other executive network functions, are influenced in a positive manner. In addition there are changes in social behaviour.

This presentation will use, for the most part, a single case study to exemplify how decisions are made concerning interventions. This will be integrated with an overview of the neural connections that underlie our hypothesis that BFB & NFB act on neural systems in a synergistic fashion. The patient example is a 24 year old law student who suffered a mild traumatic brain injury (TBI) / concussion in a car accident. This has resulted in what her family describes as a severe personality change. She is now depressed, anxious and impulsive. She suffers from frontal migraine headaches almost every day. She has labile affect that can range rapidly from clinging behaviour to sudden rages. After these outbursts she feels very badly about her behaviour. Perhaps due to changes in the „executive network“ functions she is, for the first time in her life, having difficulty with attention, concentration, and memory. In addition, her ability to understand social nuance and innuendo appears compromised. The raw EEG, brain maps, LORETA source correlations, and evoked potentials will be shown and differences from both the Neuroguide and WinEEG data bases will be outlined.

We will show how the Neuroguide findings with LORETA analysis correlate with her symptoms. These correlations are used for planning intervention using LORETA NFB. The brain map findings also led to a prescription of transcranial direct current stimulation (tDCS). For her headaches we added passive infra-red feedback (pIR). The psychophysiological stress assessment findings, which showed a failure to recover quickly after a minor stress, paralleled her difficulties self-regulating and controlling anxiety in stressful situations. These measurements led to decisions regarding the use of peripheral biofeedback and heart rate variability (HRV) training.

The combined approach reflects the ideas published several years ago in an article entitled, A Systems Theory of Neural Synergy. The concept of interactions and synergy is not new. Back in 1949 Walter Hess, in his address when receiving the Nobel Prize for Medicine and Physiology, said: "Every living organism is not the sum of a multitude of unitary processes, but is, by virtue of interrelationships and of higher and lower levels of control, an unbroken unity". Our work reflects this basic principle and helps explain the effects on the central nervous system (CNS) when NFB and
BFB are combined.

One example is that HRV training gives direct afferent input to the solitary nucleus in the medulla, and that nucleus connects with the locus coeruleus (where norepinephrine is produced), the amygdala, and the hypothalamus, structures that have a direct effect on the hypothalamic-pituitary-adrenal (HPA) axis and on the ability to calm one's self to control anxiety and stress. Perhaps even more importantly, the solitary nucleus connects to the posterior region of the thalamus. This may be why we have observed and reported on a rise in the amplitude of sensorimotor rhythm (SMR) when we do HRV training. Clearly, when we begin our NFB training, which we usually do over the central midline structures (CMS) and, in particular, over Cz, which lies above the anterior cingulate gyrus, Brodmann area (BA) 24 we may be having effects on the affect, executive, salience and the default networks. Whatever else we may enhance or inhibit, we almost always also raise SMR. We usually do the peripheral biofeedback and the neurofeedback training procedures simultaneously during sessions. We may alternate these sessions with sessions that use LORETA NFB, which has the advantage of being able to more directly target CMS that are deep in the cortex. LORETA NFB also means that we can have an effect on multiple nuclei within the same, or within more than one, network when their activity deviates outside the standard deviation (SD) limits that we set for that client.

This is a clinical presentation that we hope meets the goal of giving a rationale for combining NFB with peripheral BFB interventions. The ideas are as old as the Latin ideal of mens sana in corpore sano (a sound mind in a sound body) and as new as the latest neuroscience discoveries. The goal is to deepen practitioners' understanding of connections so they can plan multi-modal interventions that achieve success with every client.

References


**Learning Objective**

List reasons why initial NFB training over central midline structures, based on QEEG and LORETA assessment findings, is likely to produce improvement in some of the core symptoms of people with symptoms related affect such as anxiety, and to executive functions such as attention span and concentration.

Outline with reference to affect, executive, and distress networks, and the hypothalamic-pituitary-adrenal axis (HPA), why HRV training may have a positive influence on the outcomes of patients who have a combination of: affect difficulties such as anxiety, and executive problems such as low attention span.

Describe a neural pathway from the heart to the thalamus that may influence production of SMR.
Describe the most likely effect on cortical function of doing transcortical direct current stimulation with the anode placed over the left prefrontal cortex and the cathode over the right orbit.

Outline

First 15 Minutes: Dr. Lynda Thompson will briefly describe the presenting symptoms of a patient who suffered a mild traumatic brain injury (TBI) / concussion in a car accident. Drs. Lynda and Michael Thompson will describe the assessment results including the raw EEG from the 19 Channel EEG, brain map, LORETA and ERP findings. The findings of a psychophysiological stress assessment and the basic HRV data will be given. ii. 15-35 Minutes: The symptoms will be related (brief overview) to possible neural networks and the central midline structures of the brain. A brief outline will be shown of the neural connections of the heart and baroreceptors to the medulla and the links to the CMS of the brain. This overview leads to answering the question of “why” we combine NFB with BFB. iii. 35-50 Minutes: The presenters will describe how NFB + BFB sessions are implemented at our centre including descriptions of when and how LORETA NFB, tDCS, piR, strategies and counseling are integrated into the sessions. iv. 50-60 Minutes: Summary and results v. The last 5 minutes will be devoted to answering questions.

Financial Interest: Lynda Thompson is co-author of THE A.D.D. BOOK. Michael and Lynda are co-authors of SETTING UP FOR CLINICAL SUCCESS. Michael and Lynda Thompson are co-authors of THE NEUROFEEDBACK BOOK. It is likely that these books may be on sale at the meeting. The authors will state their interest in these books at the workshop.

Comparing the Effects of Neurofeedback and Hyperbaric Oxygen Therapy in Autism Spectrum Disorder: A Case Series (R,C)

Robert Coben, PhD, Private Practice, drcoben@gmail.com
Patrick Elliott, MD

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: 1

Abstract

The prevalence of Autism Spectrum Disorders (ASD) continues to rise at an alarming rate (CDC, 2012). As a result, the need for empirically validated treatments and knowing which treatment works best for whom becomes all the more pressing. Due to a dearth of this knowledge, Green et al. (2006) have shown that most children with ASD utilized multiple treatments and there is no guide as to which treatment might work best. We now present data on a comparison of two popular treatments for ASD with preliminary empirical support, Neurofeedback (NF) (Cohen & Wagner, 2011) and Hyperbaric Oxygen Therapy (HBOT) (Rossignol, 2007). After presenting information on the empirical support of these approaches, data will be presented in a case series format. These data will present symptom and neurophysiological (QEEG) changes derived from NF and HBOT delivered to separate patients with ASD. Lastly, A-B and A-B-A design data will be presented for NF and HBOT that were administered to the same patients at different times. This gives us, for the first time, the ability to compare the effects of these treatments in the same patients. The findings suggest that, while HBOT can be helpful in certain cases, NF seems to help more often, has a greater effect and is more specific in the changes that may be achieved. Clearly, more empirically based research is needed to confirm these findings.

References

Surveillance Summaries, 61 (SS03), 1- 19.


**Learning Objective**
State the varying neurophysiological affects that Neurofeedback and HBOT may have on autistic symptoms and brain functioning.

**Outline**
Autism symptoms and neurophysiology: 15 minutes

2. Effects of Neurofeedback on Autism neurophysiology: 15 minutes

Effects of HBOT on Autism neurophysiology: 15 minutes

Comparing the effects of Neurofeedback and HBOT – theoretical considerations: 15 minutes

**Financial Interest:** Dr. Patrick Elliott is the medical director of the Autism Treatment Center where this research was conducted. Dr. Coben has no conflict of interests.

---

**In Search of Depression (R,C)**

 Kelly Callaway, BS, University of Tennessee  
 Rex Cannon, PhD, University of Tennessee, rcannon2@utk.edu  
 Kenneth Phillips RN, PhD, University of Tennessee  
 Gregory Stuart, PhD, University of Tennessee  
 Deborah Baldwin, PhD, University of Tennessee  
 Deborah Welsh, PhD, University of Tennessee

**Credits:** CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: 1

**Abstract**
The DSM-IV-TR proposes that patients suffering from MDD will display state-dependent irregularities during examination by electroencephalogram and other experimental methods. This study sought to capture this state dependency by utilizing topographical EEG and connectivity and LORETA current source density in the alpha frequency domain would differ between groups as would pre-post task salivary cortisol levels. Methods: This study was conducted with 23 (13 with depression) participants, 16 female with a mean age of 20 ± 2.45. Depressed individuals had received a diagnosis of depression within the past year. We administered the SCID-R to depressed group. We collected salivary cortisol prior to any experimental conditions. Participants then provided 4 minute eyes-closed and eyes-opened baseline EEG recordings. The participants then completed the Beck Depression Inventory while EEG was continuously recorded. Items were presented for 8s in power point and responses were marked within the EEG record. These segments were extrapolated and compared for significance within and between groups. Post session cortisol was collected and
analyzed. Results: Minimal differences are seen between depressed and non-clinical groups for topographical absolute and relative power. Significant differences were found in asymmetry, coherence and phase measures between groups. Current source density in alpha differs between groups with depressed showing specific regional increases in right prefrontal regions. Notably, cortisol decreased relative to the BDI task in all subjects, with differences still evident between groups. Discussion: As with many other studies topographical power differences are sparse. Connectivity and LORETA current source density measures do reveal significant differences between groups and may provide a more accurate method for differential diagnosis of depressive disorder. Several studies have reported blunted cortisol responsivity in depression relative to stressors and our data appear to follow these results. Diagnostic, research and clinical implications are discussed.

References


D. Begic, M. Mahnik-Milos, J. Grubisin, EEG characteristics in depression, "negative" and "positive" schizophrenia, Psychiatr Danub 21 (2009) 579-584.


C. Bockting, A. Lok, I. Visser, J. Assies, M. Koeter, A. Schene, Lower cortisol levels predict recurrence in remitted patients with recurrent depression: A 5.5 year prospective study, Psychiatry Res (2012).


**Learning Objective**
Obtain information about glucocorticoids, EEG and LORETA current source density differences in depression.

**Outline**
Introduction to Depression and its neural correlates (10 min)
Beck Depression Inventory and neuroanatomy of depression (15 min)
Application for neurofeedback and diagnoses (10 min)
Potential implications in the etiology of depression (10 min)
Conclusions (10 min)
Questions (5 min)

**Financial Interest:** No financial interests.

---

**Sunday, September 23, 2012**

**Plenary Room 1**
**Real-time Functional Magnetic Resonance Imaging Neurofeedback to Attain Volitional Control over Brain Activity and Associated Mental Functions: A Systematic Review (R)**

*Gunther Meinlschmidt, PhD, Ruhr-University Bochum, Gunther.meinlschmidt@rub.de*  
*Dr. Seung-Schik Yoo, Brigham & Women’s Hospital; Harvard Medical School*  
*Marion Tegethoff, Department of Psychology, University of Basel*

**Credits:** CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: .5

**Abstract**

Background:

Technical advances have allowed processing functional magnetic resonance imaging (fMRI) data in real-time (RT) (Cox, Jesmanowicz, & Hyde, 1995), enabling its use for neurofeedback (NF) applications (Posse et al., 2003; Weiskopf et al., 2003; Yoo & Jolesz, 2002). This opened the way to conduct studies aiming at modulating brain activity and associated mental processes by RT-fMRI-NF, based on accumulated knowledge on brain activity related to mental functions (Caria, Sitaram, & Birbaumer, 2011; Weiskopf, 2011). We here present the first systematic review on RT-fMRI-NF for
volitional control attainment.

Method:

We identified articles on the use of RT-fMRI-NF to attain volitional control over brain activity in humans, by a systematic search in several scientific databases (Medline, Embase, Psycinfo, Web of Science). Two independent reviewers extracted relevant information. We assessed the study quality of the identified articles and quantitatively integrated the results. The systematic review was conducted in accordance with the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guidelines (Moher, Liberati, Tetzlaff, & Altman, 2009).

Results:

Nearly all identified studies reported that RT-fMRI-NF was associated with attainment of volitional control over hemodynamic brain activity, which was more pronounced than in control conditions (such as sham feedback, if included in the study design). Moreover, attainment of volitional control was mostly associated with modulation of mental functions or symptom improvement (e.g. DeCharms et al., 2005). However, the identified studies substantially varied in study and reporting quality.

Conclusion:

The accumulated body of evidence suggests that RT-fMRI-NF can be used to attain volitional control over hemodynamic brain activity (and most likely associated neuronal activity), and thereby over associated mental functions. However, high quality studies, including randomized controlled trials, are highly warranted. Based on the reviewed articles, we suggest a gold standard for conducting and reporting high quality RT-fMRI-NF studies.

References


Learning Objective
Evaluate the current state of research on the use of real-time fMRI neurofeedback to attain volitional control over brain activity and associated mental functions.

Outline
Background and basic idea of real-time fMRI neurofeedback to attain volitional control (5-10 minutes)

Current state, limitations, and future directions of the research field regarding real-time fMRI neurofeedback to attain volitional control (20-25 minutes)

Financial Interest: No conflicts of interest.

Multi-modal Treatment of Stuttering: A Case Study Showing Neurofeedback Coupled with Traditional Speech Therapy (C)

Becky Bingham, RN, NeuroSolution Center, Inc., Becky@NeuroSolutionCenter.com

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: .5

Abstract
A growing body of research has shown Neurofeedback to be effective to varying degrees for numerous disorders, yet little research has been conducted for stuttering. Here I present a case study highlighting the effect of treatment for stuttering using Neurofeedback combined with traditional speech therapy. The case is a 15-year-old boy rated as a moderate to severe stutterer. He had received extensive speech therapy in past years and had an ebb and flow in symptoms that was consistent with a stereotypical stuttering profile. Neurofeedback began with baselines established using IntegNeuro assessment coupled with a QEEG and Hudspeth analysis. The QEEG showed excessive slow wave activity from 5-9Hz and excess high beta from 18-25Hz. The Hudspeth coherence map showed hypo-connectivity on the left hemisphere especially between F7 and T5. A coherence reward was therefore targeted from 3-12Hz with the sensors placed at F7 and T5. Twenty sessions of coherence training Neurofeedback were followed by a repeat QEEG. In spite of coincidental decrease in frequency of speech therapy, therapist rated his improvement as significant, and the client reported significant reduction in speech blocks and those that are more mild and easier to overcome using speech therapy techniques. A NeuroRep comparison report showed textbook changes in delta not only in the area we are working on but also throughout the head, as well as changes in theta and alpha in the occipital and parietal areas in eyes closed. Eyes open showed improvement theta and alpha on the left hemisphere where we were working. Another set of 20 sessions of Neurofeedback coherence training was undertaken with sensors remaining at F7 and T5 reward of 1-19Hz and inhibit of 4-9Hz and 19-30Hz. The boy reports being almost completely free from stuttering; his speech therapist, school therapists and teachers observed only minimal stuttering.

Clearly there is significant room for further research on the effect of Neurofeedback and especially coherence training in helping stutterers lead normal lives.

References


**Learning Objective**
Describe the current state of research on the application of Neurofeedback to stuttering.

Explain how Neurofeedback can potentially be used to improve the outcomes of traditional speech therapy.

**Outline**
Summarize research on the potential application of Neurofeedback to stuttering 5 minutes

Presentation of case study 15 minutes

Discussion of protocols used 5 minutes

Questions 5 minutes

**Financial Interest:** No outside financial interests.

**The Impact of an 8-Week Heart Rate Variability Biofeedback (HRV) Training on Quantitative EEG and LORETA Following a Cognitive Stressor (R)**

Jeffrey Tarrant, PhD, University of Missouri, tarrantj@health.missouri.edu
Heather Eastman-Mueller, PhD
Ae Kyung Jung, MA
Laura Sinquefield, MA
Brett Woods, M Ed
Chad Cross, BA

**Credits:** CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: .5

**Abstract**
A study by Sherlin, Muench, and Wyckoff (2010) demonstrated that 15 minutes of respiratory sinus arrhythmia (RSA) biofeedback immediately following a cognitive stressor resulted in significant changes in both alpha and beta bands in certain limbic structures. This study suggested that RSA training may decrease arousal and enhance relaxation through impacting areas of the brain critical in the stress response. The current study expands on previous work by examining the impact of an 8-week breathing based biofeedback program.
The sample consisted of twenty-seven students at a large Midwestern university. Of those, seventeen participants were enrolled in a one-hour credit class entitled, “Transforming Stress: Heart Rate Variability Biofeedback” and considered the intervention group. A control group (n=10) consisted of students enrolled in a Learning Strategies course. All participants in the study had to complete a series of self-report questionnaires to examine levels of generalized anxiety (GAD-7; Spitzer, et., al., 2006), test anxiety (The Westside Test Anxiety Scale (Driscoll, 2007) and coping self-efficacy (Coping Self-Efficacy Scale-reduced form (Chesney, et., al., 2006). In addition, students were asked to engage in a series of physiological recordings that included EEG, respiration rate, skin temperature and conductance, and heart rate variability. These scales and measurements were completed at week one and week eight, following the class.

Our proposed hypotheses: 1) Significant decreases will be noticed in generalized anxiety, test anxiety and improved coping skills. This will be assessed through conducting a paired sample t-test to determine if any statistically differences existed between the control and intervention groups.

2) Statistically significant reductions in physiological activation readings will be reported from pre to post intervention for those members of the intervention group. Multi-comparison tests will be conducted to determine if any significance differences existed by comparing pre-intervention EEG recordings to post-intervention recordings. Separate analyses will be done to compare baseline recordings, cognitive challenge (modified Stroop task; Congedo, 2005) recordings and recovery recordings in the following ways.

a) Power means within the standard Neuroguide frequency bands will be summed across all electrode sites in both absolute and relative power. Within and between group differences (compared to control group) will be assessed using an ANOVA analysis.

b) Current source density comparisons between pre-intervention baseline and post-intervention baseline will be completed using data from LORETA, specifically with alpha and beta bands and in Brodmann areas consistent with the anterior cingulate (25, 24, 32, 33, 10). Within and between group differences (compared to control group) will be assessed using a ANOVA analysis.

3) Difference scores in absolute power and relative power (combining all sites for each Neuroguide band) will be calculated for baseline vs. recovery and cognitive challenge vs. recovery. This will be conducted for both pre and post intervention scores. The pre-post difference scores will then be compared using paired sample t-tests to determine if students improved in their ability to recover after the cognitive challenge.

4) We hypothesize that the greatest benefits noticed in the intervention group will be exhibited in those students with significantly elevated stress and anxiety at pre-testing. To test this hypothesis, subjects will be subdivided into low anxiety and moderate/high anxiety groups based on criterion scores established with the GAD-7 (Spitzer, et. al., 2006). It is likely that the number of subjects in each group will be insufficient to perform a meaningful ANOVA. Consequently, this comparison will be descriptive.

5) We also predict a direct inverse relationship between the amount of time students practiced biofeedback and their self-reported stress, anxiety, and coping as well as physiological functioning, meaning the longer the participant practices the less anxiety they will exhibit. This will be examined by conducting a multiple stepwise regression to determine if amount of practice was significantly associated with decreases in beta power (or increases in alpha) for the metrics identified above as well as self-report measures.

Limitations of the study include a small sample size of both intervention and control groups, thus precluding generalizability. Also, we only tested one cognitive stressor and therefore generalization
to other cognitive stressors is limited. Future directions will be discussed based on final results.

**References**


**Learning Objective**

Clearly articulate the ways that heart rate variability biofeedback impacts physiological recovery from a cognitive stressor.

**Outline**

Introduction to heart rate variability biofeedback (5 min)

Review of previous research using hrv and/or rsa biofeedback for stress (5 min)

Outline of current research study (5 min)

Description of results (10 min)

Conclusions/future directions (5 min)

**Financial Interest:** No financial interests to disclose.

**Self Regulation of Slow Cortical Potentials in Patients with Intractable Epilepsy - Eight Years After (R,C)**

Ute Strehl, PhD, University of Tubingen, ute.strehl@uni-tuebingen.de
Abstract
Objective:

The aim of this study was to answer the question whether the effects of a behavior therapy program for patients with intractable epilepsies that includes self-regulation of slow cortical potentials (SCP) were still present more than eight years after the end of treatment (Kotchoubey et al., 2001). In the main study the experimental group (SCP group) received a training of SCP regulation while the two control groups were treated either with respiratory feedback therapy (RES) or adjustment of antiepileptic medications (MED) in combination with psychosocial treatment.

Methods:

Seizure frequency, medication, psychological variables and neuropsychological functions of the patients of one experimental (SCP-Feedback) and two control (Respiratory Feedback -RESP- / Adjustment of antiepileptic medication -MED-) groups were assessed and compared. From 41 patients in the experimental group 19 patients were recruited and two patients from each control group (out of 12 RESP and 11 MED). The same psychological tests (WAIS-R; WMS; BDI; MMPI-2; d2; Locus of Control) were applied as in the original study. In addition, three SCP-training sessions were conducted. A comparison with the control groups was not feasible due to a lack of participants out of these groups.

Results:

A statistically significant decrease of seizures since the end of treatment was observed. With the exemption of those patients that underwent neurosurgery, participants were still able to self-regulate their slow cortical potentials during the feedback condition. IQ and memory values were worse compared to the one year follow-up but not below the level of pre-treatment assessments. Psychological variables were still in a non-clinical, normal range.

Conclusion:

About eight years after the end of an EEG-biofeedback treatment for patients with refractory epilepsy, a statistically significant trend to a lasting reduction in seizure frequency was observed. All patients except those who had received surgical treatment in the meantime still had the ability to self-regulate their slow cortical potentials. Given such a long follow-up period, the possible impact of confounding variables has to be taken into account. In addition, considering the small number of patients participating in this follow-up evaluation and the fact that members of the control groups refused to take part, causal conclusions cannot be drawn. Yet, given the positive and sustained development of the patients who participated in the EEG biofeedback training, future treatment planning and research should not only aim at optimizing conventional therapies, but should include EEG-biofeedback as an option in the treatment of patients with (intractable) epilepsies.

References

Learning Objective
Understand why slow cortical potentials feedback can reduce seizures.
Understand a slow cortical potentials training protocol in a behavioral medicine setting.

Learn about the results of a long term follow-up study.

Discuss design problems of long term follow-up study.

Outline
SCP-Feedback in intractable epilepsies (10 min)
Behavioral medicine setting (5 min)
Short and long term results (10 min)
Questions and answers (5 min)

Financial Interest: No financial interests or relationships.

STUDENT PRESENTATION

Theta-Beta Neurofeedback for Adult ADHD: EEG and Behavioral Changes (R,C)
Sarah Wyckoff, MA, University of Tubingen, wyckoffsarah@yahoo.com
Kerstin Mayer, M Sc, University of Tubingen
Ute Strehl, PhD, University of Tubingen

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: .25

Abstract
Objectives

Attention–Deficit/Hyperactivity Disorder (ADHD) is one of the most common disorders of childhood and persists into adulthood for approximately 5% of the population worldwide (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007). The primary symptoms of ADHD include inattentiveness, impulsivity, and hyperactivity. Analysis of resting state EEG from adults with ADHD has produced a variety of activity patterns in power, coherence, and asymmetry measures, as well as the typical increases in theta/beta ratios seen in pediatric populations (Bresnahan, Anderson, & Barry, 1999; Bresnahan & Barry, 2002; Clarke et al., 2008a). Neurofeedback training is a treatment method that utilizes operant conditioning to reinforce specific EEG activity. In a recent meta-analysis, a large effect size (ES) was found for neurofeedback on impulsivity and inattention in controlled studies and pre- and post-designs (Arns, de Ridder, Strehl, Breteler, & Coenen, 2009). Studies indicated that ADHD children are able to self-regulate cortical activity (Drechsler et al., 2007; Leins et al., 2007; Strehl et al., 2006), which lead to changes in spontaneous EEG activity (Gevensleben et al., 2009; Monastra, Monastra, & George, 2002). However, limited research has investigated the efficacy of neurofeedback as a treatment for adult ADHD. This study will investigate changes in EEG following a course of 30 sessions of theta-beta neurofeedback.

Methods

Continuous 20-channel EEG was acquired from 12 adult participants that met DSM-IV criteria for ADHD (combined, inattentive, or hyperactive type) and 12 healthy matched controls, both groups without additional serious physical, neurological, or psychiatric disorders, and a full scale IQ > 80. The ADHD group received 30 sessions of neurofeedback training in which theta (4-8Hz) activity was inhibited and beta (13-21Hz) activity was augmented at CZ (referenced to A1, ground A2). Each
session consisted of a 2-minute baseline, (3) 7-minute blocks of continuous feedback of theta and beta frequency band amplitudes, and (1) 7-minute transfer block in which amplitude feedback was not presented. EEG recordings were collected pre/post treatment and included an EO, EC, and active listening task, in addition to ADHD behavioral questionnaires.

Results

This investigation is in progress. The EEG was Fourier transformed to provide absolute and relative power estimates for the delta, theta, alpha and beta bands. A baseline comparison of the ADHD participants and healthy controls, as well as, pre/post-training changes in behavioral and neurophysiologic parameters will be presented at the time of the conference.

Conclusion

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

References


**Learning Objective**
Understand and report protocol and disorder specific EEG and behavioral outcomes extracted from pre/post continuous 20-channel recording of EC, EO, active listening task following a course of theta-beta neurofeedback for adult ADHD; assess if adult ADHD patients identified as being able to learn to self-regulation neurofeedback parameters experience greater EEG and core symptom changes.

**Outline**
Background on EEG findings in adult ADHD; description of neurofeedback protocol/collection methods: (5 min)

Study population demographics, EEG/session data processing methods, and results: (7 min)

Discussion of treatment implications, study limitations, and future directions: (3 min)

**Financial Interest:** We have no significant financial interest or relationship with the commercial supporter(s) or manufacturer(s) of any commercial product or service that is discussed as part of this presentation.

**Combined Neuromodulation Method Aimed to Improve Frontal Functions in Autism (R,C)**

Estate Sokhadze, PhD, University of Louisville, tato.sokhadze@louisville.edu
Ayman El-Baz, PhD, University of Louisville
Allan Tasman, MD, University of Louisville
Lonnie Sears, PhD, University of Louisville
Manual Casanova, MD, University of Louisville

**Credits:** CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: .5

**Abstract**
**Introduction**

Among the emerging methods of neuromodulation such neurotherapeutic techniques as repetitive Transcranial Magnetic Stimulation (rTMS) and neurofeedback (NFB) are most promising for the treatment of core autism symptoms. TMS offers a noninvasive method for altering excitability of the neural circuits and induction of a short-term functional reorganization in the human cortex. Since effects of rTMS are not limited to the stimulated target cortex but give rise to functional changes in anatomically and functionally interconnected cortical areas, rTMS is a suitable tool to modulate neural plasticity within a distributed functional network. The rTMS may have therapeutic potential in some psychiatric disorders (e.g., depression, George et al., 1999) We reported recently positive therapeutic effects of low frequency rTMS in autism spectrum disorders (ASD) (Sokhadze et al., 2009 2010,2012; Baruth et al., 2010). The NFB is a form of operant conditioning of electroencephalographic (EEG) activity in which desired electrocortical activity is rewarded, while undesirable is inhibited. Positive effects of NFB training have been found and well documented for ADHD (Sherlin et al., 2010). Less is known about the effects of neurofeedback based intervention to sensory and cognitive functions in children with ASD (Coben et al., 2010). There are not any studies yet reported where rTMS and neurofeedback are used as a combined neuromodulation approach to treat core symptoms of autism.

**Goals**
Autism is a pervasive developmental disorder of childhood characterized by deficits in social interaction, language, and stereotyped behaviors and restricted range of interests. The study is based on an underlying neuropathology model of autism which emphasizes minicolumnar pathology (Casanova, 2007, Casanova et al., 2006) and lateral inhibition deficits resulting in behavioral, executive, and emotional dysfunctions. We propose that neuromodulation based on low frequency rTMS will enhance lateral inhibition through activation of inhibitory double bouquet interneurons and will be accompanied by improvements in prefrontal executive functions. The numerous studies of effects of TMS agree that most profound acute effects of magnetic stimulation last for approximately one hour, while effects of TMS session have one week-long washout period (George et al., 1999). It is an important goal to maintain and reward positive effects of individual TMS session during rTMS treatment course, which usually consists of 12 weekly sessions.

Method

In this study we used a novel approach by combining prefrontal rTMS sessions with prefrontal neurofeedback (NFB) to prolong and reinforce TMS-induced electrophysiological changes using operant conditioning paradigm. The pilot trial recruited children and adolescents with ASD. Outcome measures included behavioral and psychophysiological responses. In particular both active treatment groups (TMS only [N=20], NFB only [N=8], TMS with neurofeedback [N=6], and wait-list [N=20]) were assessed at (1) the initial baseline using clinical behavior questionnaires, i.e., ABC (Aman & Singh, 1994), SRS (Constantino & Gruber, 2005), RBS (Bodfish et al., 1999) and performed on visual oddball task with evoked EEG response recording (Kanizsa illusory figure test), and (2) post completion of 12 sessions of treatment (TMS, NFB, TMS+NFB), or 4-8 week long waiting period.

Discussion and Results

The project links behavioral, clinical, and electrophysiological (EEG and ERP) responses during cognitive tests and TMS-neurofeedback treatment outcomes with an underlying developmental neuropathology model derived from investigations in our laboratory. The study represents a new development in combining rTMS with EEG biofeedback and using functional outcome measures (cognitive ERP, EEG, and autonomic nervous system activity measures during rTMS sessions), where integrated TMS-neurofeedback trial represents a theory-guided psychiatric neurotherapy in autism. In this exploratory project we used active rTMS, wait-list and neurofeedback training combinations to examine effects of each intervention arm and their combination (TMS, neurofeedback, TMS with neurofeedback, wait-list) on EEG, ERP, autonomic physiology (heart rate, HV variability [HRV], skin conductance level, skin temperature), and other functional and behavioral clinical outcomes in autism. Collected preliminary data support our concept that rTMS induces decrease of sympathetic arousal and anxiety, improves executive functioning as evidenced by normalization of EEG/ERP responses during executive function tests and clinical behavioral evaluations, and that the combination of TMS with EEG neurofeedback may result in a synergetic outcome. We compared several clinical, behavioral, cognitive, and emotional measures to select those more sensitive to predicted changes resulting from the combined neurotherapy.

Conclusion

The results of our innovative pilot study were used to design a research project to explore clinical efficacy of developed novel integrated TMS/biofeedback neuromodulatory intervention for treatment of core autism symptoms. The grant proposal based on these concepts and pilot data has been submitted to a federal agency as an exploratory project.

References


Baruth, J.M., Casanova, M., El-Baz, A., Horrell, T., Mathai, G., Sears, L., Sokhadze, E.(2010b). Low-
frequency repetitive transcranial magnetic stimulation (rTMS) modulates evoked-gamma oscillations in autism spectrum disorder (ASD). J. Neurother., 14, 179-194


**Learning Objective**
Learn about executive frontal deficits in autism and understand how rTMS and neurofeedback can be used to improve these deficient functions in children with autism.

**Outline**
Introduction – 5 min

Methods – 5 min

Results and Discussion – 5 min

Conclusions and Future Directions – 10 min

Questions and Answers – 5 min

**Financial Interest:** No significant financial interests to report.
Neurofeedback Protocol for the Treatment of Phonetic and Expressive Speech Impediments: Report of Two Cases (C)

Jorge Julian Palacios, PhD, Biofeedback Center, jorgepalacios@biofeedbackcentemr.com

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: .5

Abstract

Background:

We will report the treatment results of two children, 6 and 5 years old respectively, who were diagnosed with severe Phonetic and Expressive Language Impairments (PELI) since they were two years old. The children failed to develop language with no diagnoses of slow development syndrome, physical abnormalities of the speech apparatus, autistic disorder, acquired or genetic brain or neural damage, hearing loss, oral motor deficits, and proper strength, coordination, range of movement, symmetry and speed of cranial nerves V, VII, IX, X and XII. Poor performance in the school, audition hearing test results of 25dB, nonverbal IQ of 90; lower verbal IQ, no emotional disturbances. No fluently speech, 20 words repertoire, short phrases, words omission poor comprehension of language in general.

Methods:

Evaluations results were with 85% Delta/Theta (1-7 Hz.) prevalence over 15% Beta1 (15-18 Hz) with eyes open. Patients receive 40 sessions Neurofeedback treatment, 3 sessions weekly, using an I-330 C2+6 Neuro/Biofeedback system with USE3 software, by J&J Engineering and trained to increased Beta1 and decreased Delta/Theta and EMG (40-360 Hz.) in T4/C4 and P3/F7 derivations simultaneously. Neurofeedback stimuli were auditory and visual.

Results:

Beta1 prevalence increased to 40% while decreased Delta/Theta to 60% and EMG to 10%, after second week treatment, school and parents reported general improvement in phonetic and expressive speech and language, increment in verbal repertoire and verbal IQ with more than 100 words, fluently speech, develop of accurate written language and improvement in school performance. Results persisted after 3 and 1 follow up years.

Conclusions:

Findings suggest that induction of brain activity integration T4/C4 and increment Beta1 in left hemisphere P3/F7 lead to appropriate speech development and strongly suggest to be replicated with a bigger group to develop standardized treatment protocols.

References


Learn the basic procedures to apply this protocol for the treatment of Speech Impediments and replicated in order to validate it.

Outline
Presenting and discussing the results of the Neurofeedback treatment for Speech Impediments and its implications for the development a new treatment protocol.

Financial Interest: No financial or commercial relationships with any individual or manufacturer or enterprise other than the Biofeedback Center.

Sunday, September 23, 2012

Plenary Room 2

On the Relation Between $\alpha$ and $\Theta$ in Specific Parieto-frontal Networks in Adult Attention Deficit/Hyperactivity Disorder (ADHD)

Rex Cannon, PhD, University of Tennessee, rcannon2@utk.edu
Debora Baldwin, PhD, University of Tennessee
Cynthia Kerson, PhD, Brain Science International
Tiffany Shaw, MS, University of Tennessee
Dominic DeLoreto, MA, University of Tennessee
Sherman Phillips, MA, University of Tennessee
Coleman Garner, BA, University of Tennessee

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: 1

Abstract
Introduction:

Several authors have proposed potential parietal foci for symptoms of attention deficit hyperactivity disorder (ADHD) involving interdependencies of the alpha and theta EEG frequency domains. We investigated these mechanisms across two studies. First we utilized intrinsic network connectivity of medial prefrontal cortex with other locations in the default network (DMN) using functional magnetic resonance imaging and standardized low resolution electromagnetic tomography. Second, we analyzed a larger sample of ADHD adults using sLORETA and evaluated changes initiated in the alpha and theta frequency domains between parietal and frontal regions using LORETA neurofeedback.

Methods:

Study 1 was conducted with 13 total participants, 7 normal controls and 6 adults with a current diagnosis of ADHD. We generated a PPI (psycho-physiological interaction) model for each individual using the SPM5 software package. We extracted the time-course from medial BA10 region of interest and assessed connectivity within the default network using fMRI and sLORETA. Study 2 assessed functional connectivity between parietal and frontal regions in the alpha and theta frequency...
domains in 14 participants (ADHD and control) for pre and post training LORETA neurofeedback.

Results:

Medial prefrontal regions do show significant correlations with many, not all regions in the DMN, with an emphasis on left parietal areas. Interestingly, the ADHD group shows increased correlations in the alpha frequency domain between frontal and parietal regions, and less connectivity strength for other frequencies. This pattern was also confirmed pre LNFB training. Changes in alpha and theta cross-frequency coupling between parietal-frontal regions shift as a result of training and increased scores in executive functions are noted. Clinical, research and theoretical implications are discussed.

References


Learning Objective
Gain important information about cross-frequency modulation between frontal and parietal cortices in ADHD and normal controls.

Outline
Functional connectivity and plasticity (10 min)

Inter-frequency and cross-frequency modulation between neuroanatomical locations (15 min)

Application for neurofeedback and diagnoses (10 min)

Potential implications in the etiology of ADHD (10min) Conclusions (10 min)

Questions (5min)

Financial Interest: No financial interests.

60 Minutes on the LENS Effects (R,C,T)
Len Ochs, PhD, Ochs Labs, lochs@earthlink.net
Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: 1

Abstract
This oral presentation introduces a variety of data-based topics on the LENS as well as tools used with the LENS. These topics range from:

Demonstrating the OchsLabs, Inc. Quality and Study Manager (QSM) and some of the capabilities of the QSM to serve as a research engine demonstrating results from incomplete studies under way,

A video-composite of the EEG from an 80-session course of treatment of fibromyalgia demonstrating a time-lapse view of the undulations of Alpha EEG,

A brief overview of both the helpful release of suppression as well as the helpful addition of suppression to a fragile and explosive young man,

A view of several different kinds of maps used for the LENS, and ends with

A data-based discussion of some of the typically unappreciated properties of many EEG amplifiers to generate stimulation, showing that the LENS is no more of a stimulation system than any other neurofeedback system.

References


Marcus, L. (2001). EEG Amplitude and Variability Changes Following Low-Intensity Neurofeedback-Based Stimulation for Fibromyalgia. Palo Alto, CA, Western Graduate School of Psychology. Ph.D.


Ochs, L (1997). EDS: Background and operation, EEG-driven pico-photic stimulation. Walnut Creek, CA, Flexyx, LLC.

Ochs, L (2006). Thoughts about EEG-Driven stimulation after three years of its uses: Ramifications for concepts of pathology, recovery, and brain function.


Ochs, L (2010) Underlying Treatment Issues in Neurofeedback as Exemplified by Treatment of
Learning Objective
Identify the OchsLabs, Inc. Quality and Study Manager.

Describe two underlying physiological events that contribute to the LENS effects.

Describe one advantage and one disadvantage of the presence of EEG suppression.

Describe two different ways that LENS Maps and qEEGs differ.

Explain two reasons why the LENS is not a stimulation system.

Outline
The Quality and Study Manager (QSMTM)

What's a Vascular vs. Neurological Effect: Vascular Trees and Vascular Responses of the LENS and EEG Rises and Falls during the Use of the LENS

EEG Suppression: Curse and Blessing

LENS Maps and Qs: Different Purposes, Different Tools

Areas for Research: The EEG Box, Itself, as a Trigger for Change

Financial Interest: Len Ochs is the inventor and developer of the LENS. I have no ownership, paid or unpaid position with OchsLabs, Inc. as my entire income is from Social Security retirement; I am not an employee of OchsLabs, Inc.

Role of QEEG Guided Neurofeedback in the Overall Treatment of Fetal Alcohol Spectrum Disorder (FASD) (C)

Ajeet Charate, MA, Illinois Centers for Fetal Alcohol Spectrum Disorders, acharate@trinity-services.org
James Kowal, PhD, The Center for Traumatic Stress, jkowal@traumaticstress.org

Credits: CME, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences Credits and BCIA recertification credits: 1

Abstract
Fetal Alcohol Spectrum Disorder (FASD) is an umbrella term used to describe the range of effects that can occur in an individual whose mother drank alcohol during pregnancy. Alcohol is a teratogen and therefore selectively toxic to the developing fetus, particularly to the Central Nervous System. Researchers estimate that some 2-5% of school age children have FASD. Most individuals lack the characteristic dysmorphologic features and/or a history of birth mothers use of alcohol during pregnancy, thereby making FASD one of the most underreported and under diagnosed conditions. Without a proper diagnosis, standard treatments are largely in-effective. The goal of this study is to
assess if QEEG analysis can assist in recognizing the neurological patterns, understanding the primary symptoms, and planning neuro-treatment. Ten children diagnosed with FASD received pre and post intervention QEEG analysis. No obvious pattern emerged but QEEG analysis showed a lot of variability based upon the amount of alcohol consumed by the birth mother and the stage of pregnancy. It helped in developing the protocols and deciding what neuro-treatment would be most beneficial. Some children received standard neurofeedback while a few received coherence training and LENS. The study concluded that pre-treatment QEEG analysis is absolutely essential in planning interventions for individuals with FASD. Additionally, behavioral consultation, skills coaching, parental support, and education about FASD resulted in greater improvements. Results of this study have been very promising. No significant side effects were reported. Future studies should include a larger sample size for studying neurological patterns, clinical trials with standard neurofeedback versus LENS treatment, and effects of nutrition and supplements.

References

Learning Objective
Be able to recognizing neurological patterns in persons diagnosed with FASD.

Identify types of treatment options available for FASD.

Identify other interventions to improve functioning and support within families with FASD.

Outline
1. Introduction to FASD (5-10 mins)
   Definition of FASD
   Prevalence & diagnostic criteria
   Primary and secondary symptoms
   Co-occurring conditions
2. QEEG Findings (10-20 mins)
   Symptoms mild, moderate, or severe
   Academic, Cognitive, Behavioral, Social, Executive Functioning
   Areas Effected: Frontal Slowing, Posterior, Temporal, Bi-Lateral, . . .
   Type of Neuro-abnormalities: Amplitude, Coherence, Phase Lag, . . .
3. Neuro-Treatment Options: (5-15 mins)
   Traditional Neurofeedback (Amplitude Training)
   Low Energy Neurofeedback System (LENS)
Coherence (Z-Score)

Phase Lag

4. Family-Treatment Options: (5 mins)

Skill Achievement Groups

Counseling

Educational Counseling

5. Future Areas of Investigation (5 mins)

Nutrition and Supplements

Early Dietary Interventions

6. (Optional: Questions and Answers 5 mins)

**Financial Interest:** There is no financial interest or relationship with any commercial supporters) or manufacturer(s) of any commercial product or service that is discussed in the presentation.
Effects of the Synchroton, An Electronic Device Pulsing at 7.8 Hz Schumann Fundamental Frequency, on the EEG (R,C)

Juan Acosta-Urquidi, PhD, Brain-Topos, jacostauui@yahoo.com

Guy Abraham, MD

Abstract

The adverse health effects of constant exposure to EMF fields, from ELF, RF to MHz bandwidth, is mounting, and has been a growing public safety concern for the past few decades. Electrohypersensitive subjects (EHS) are particularly affected, presenting symptoms like allergies, chronic pain, fatigue, agitation, nausea, brain fog, cognitive impairments, etc. A previous study has reported protective effects of an electromagnetic potential pulsed at the Schumann fundamental frequency of 7.8 Hz using a Synchroton against EMF effects on a population of CFS patients (Abraham et al., 2003). The Synchroton A-30 is a compact electronic device the size of a cigarette pack powered by 2 AA batteries (Abraham, 1999, Optimox Corp., Torrance, CA). We report that exposure to the Synchroton produced an entrainment effect on the EEG.

Methods

Volunteer subjects were recruited for this study (N=16), some meeting the criteria for EHS. Most QEEG data was recorded using Lexicor NRS-24 equipment and Mitsar 201 amplifier (St. Petersburg, Russia), 19 channel electrocap, International 10-20 system, referential linked ears, impedances ca. 5 Kohms. Sampling rate 256 Hz; the bandwidth studied was 0.3 to 40 Hz. The raw EEG files were visually edited to remove ocular and muscle (EMG) artifacts. Data analysis employed Neurolex (Lexicor), Neuroguide (www.appliedneuroscience.com), WinEEG (Mitsar). Spectral FFT power frequency graphs and brain topographic maps were generated. Peak Absolute power values (uV²) were compared before (10 min. control baseline, resting eyes closed) and during Synchroton exposure (10 min. eyes closed). Data was statistically analyzed (paired correlated samples t-test).

Results

Synchroton exposure produced a robust significant increase in Theta (T) and Alpha (A) absolute power values. A pooled data comparison of mean ± sem, P values, paired t-test, correlated samples) between baseline vs Synchroton yielded: frontal sites, T 90.7 ±16, A 121.8 ±28 vs T 125.9 ±26, A 163.4 ±37, P<0.005; Central, T 127.3 ±25, A 213.5 ±54 vs T 172.6 ±35, A 284.7 ±71 vs P<0.002; Temporal, T 71.6 ±19, A 168.3 ±47 vs T 100.6 ±27, A 220.5 ±55, P<0.005; Parieto-occipital T 109.1 ±32, A 327.2 ±79 vs T 167.8 ±53, A 466.13 ±105, P<0.002. Sham Synchroton exposure as control tests produced no consistent or significant trend in Theta or Alpha power. To further explore and confirm an entrainment effect, the Synchroton exposure was delivered simultaneously with photic stimulation (Polysync Pro). The following stimulation frequencies were employed: 5, 7.8, 9.6, 10 and 14 Hz. The results of these tests revealed a complex interaction between the Synchroton and photic stimulation at different frequencies. Specific to each subject’s baseline photic entrainment response, the Synchroton either potentiated or suppressed photic driving effects. Taken together, these tests further strengthened the evidence for a direct entrainment effect of the Synchroton on EEG.

Conclusion

The Synchroton device is an effective EEG entrainment tool that has a protective effect against EMF pollution and the Schumann frequency is also believed to have many beneficial health effects. Further
studies are underway.

References


C.E.N.T Computer Enabled Neuroplasticity Treatment (R,C)
Ben Cowley, PhD, University of Helsinki, ben.cowley@helsinki.fi

Abstract
The University of Helsinki brings Neurofeedback to Finland in a new study. Finland is heavily invested in cutting-edge brain science, yet it has never before had dealings with neurofeedback (NFB), either in research or clinical practice. However the research on ADHD in Finland has been strongly ongoing (e.g. Helenius et al., 2011, Gumenyuk et al., 2004) and thus provides a good ground for the introduction of neurofeedback into Finland.

On par with more global estimates (Polanczyk et al., 2007), the prevalence of ADHD in Finnish 8-year-olds is estimated at 4% (DSM-III) (Almqvist 2004), while among Finnish 16-18 year olds it rises to 8.5% (DSMIV) (Smalley et al., 2007). Indeed, given that medication therapy for ADHD is lowest in Finland among all Scandinavian countries (Zoëga et al., 2011), there may be a greater need in Finland. The CENT project will conduct a study on the effects of NFB on adult ADHD within Finland. Research is being conducted by the Cognitive Science Unit at the Institute of Behavioural Sciences, Helsinki University. Training will be conducted by trained technicians supervised by qualified psychotherapists, at the clinics of Mental Capital Care, Oy.

References


41(1), 30-6.


**LORETA Neurofeedback: Linking Self-regulation and Anxiety (R,C)**

Dominic Di Loreto, MA, University of Tennessee, ddilore1@utk.edu

Rex Cannon, PhD, University of Tennessee

Deborah Baldwin, PhD, University of Tennessee

Sherman Phillips, BA, University of Tennessee

Tiffany Shaw, MA, University of Tennessee

**Abstract**

Introduction: Anxiety is best conceptualized as a future-oriented cognitive-affective-somatic state, the prominent feature being “a sense of uncontrollability focused on possible future threat, danger, or other upcoming, potentially negative events.” It is characterized by a sustained hyperarousal or heightened apprehension and vigilance to temporally uncertain, usually distal, danger. Behaviorally, anxiety is associated with avoidance. We propose a hypothesis that implicates disruptions in functional integration of neural networks important to self-regulation. Methods: For the current study, six (4 male) participants with a prior diagnosis of anxiety or anxiety with a comorbid syndrome completed between 15 and 20 sessions spatial specific EEG operant conditioning (LORETA Neurofeedback) to improve self-regulation. Results: All participants were able to produce significant learning effects across sessions, including network convergent learning. Post training assessment discovered significant decreases in anxiety as measured by the Personality Assessment Inventory (PAI) and significant increases in executive functions as measured with subtests from the Delis-Kaplan Executive Function System (DKEFS). Functional correlations between neurological and behavioral data demonstrate specific network involvement in these symptom reductions and provide data to develop a potential intervention for anxiety disorders in 20 days or less.

**References**


**Use of a Simple BrainMaster EEG Training Protocol to Facilitate Cognitive and Physical Recovery of a 22 Month Old Child Suffering a SIDS-Related Anoxic Injury: A Case Study (C)**

Thomas Fink, PhD, Acorn Health Associates, tomefink@comcast.net

Teri Hagen, MS, Acorn Health Associates

**Abstract**

A clinical case study will be presented describing the use of a simple BrainMaster EEG Biofeedback training protocol to successfully treat a 22-month-old child suffering a SIDS-related anoxic injury. At the initiation of treatment, the child presented as agitated and developmentally impaired. He was unable to track movements with his eyes and he did not interact in any meaningful way with his environment. Physiologically, he had difficulty supporting his head and his resting heart rate was abnormally high. After
only six sessions, using a unipolar, single-site recording from CZ and a Delta/Theta/Gamma inhibit and an SMR/Low Beta augment, the child improved dramatically. For example, resting heart rate reduced from 120/min to 70+/min in an awake state. The child presented as more relaxed and composed. He gained control of his head movements, eye contact could be established and the child began to visually track, play with toys and initiate physical movement during physical therapy sessions. EEG amplitude measures, which all changed in the desired directions, will be presented for both within and across training session measures. A brief review of available research concerning recovery from anoxic injury in children will be provided, with attention given to typical recovery patterns found without the use of Neurofeedback therapy. Implications for future application, study and research will be discussed.

References


QEEG Evaluations of a Brain Enhancement Program Utilizing the Low Energy Neurofeedback System (LENS) (C)

D. Corydon Hammond, PhD, University of Utah School of Medicine, d.c.hammond@utah.edu
Stuart Donaldson, PhD, Myosymmetries Clinic

Abstract
The Low Energy Neurofeedback System (LENS) is a unique and passive form of neurofeedback which produces its effects through feedback that involves a microscopic electromagnetic field delivered down the electrode wire(s). This presentation will briefly describe LENS neurofeedback and the existing literature on LENS. We will then describe a “brain enhancement program” that utilized LENS, along with a description of the non-clinical subject sample (N = 40-50 subjects). Outcomes measures included subject ratings on variables (e.g., concentration, memory, quality of sleep) that subjects wished to see improved, and pre- and post-treatment quantitative EEGs. Significant changes were commonly found in QEEG measures of
absolute power, coherence, and phase-lag. The results of this study will be presented, along with several detailed case examples.

References


Heart Brain Synchronicity as a Candidate Neurofeedback Index (R,C)

Dae Keun Kim, MS, Institute of Complimentary and Integrated Medicine, boeun4@snu.ac.kr
Seung Wan Kang, MD, PhD
Jae Il Kim, PhD
Min Cheol Whang, PhD

Abstract
Electrophysiological changes in response to autogenic training were explored in 12 healthy volunteers who completed 8 weeks of a basic course in autogenic training. Heart coherence, representing the degree of ordering in oscillation of heart rhythm intervals, increased significantly (p < 0.0001) during the training. Relative alpha power averaged over 19 channels and alpha coherence averaged over 171 channel combinations also increased (p < 0.0001). Parietal peak alpha power increased(p < 0.0001) with increasing heart coherence during the training, but no such relationship was observed during baseline. Average alpha coherence also increased(p = 0.002) with increasing heart coherence during the training but no significant
relationship was observed at baseline. Relative alpha power increased with increasing heart coherence during both meditation and baseline period while coupling between relative alpha power and heart coherence was stronger during in training than in baseline. It is expected that increasing heart coherence and the accompanying EEG alpha activations, heart brain synchronicity, could be a candidate marker for a quality of training, and also would be helpful in recovering synchronization of chaotic human physiology of homeostatic depletion. The following study was designed to confirm validity of the heart brain synchronicity index for a training group of early dementia. At the turn of an aging society, dementia research among any other brain related diseases is very active. The dementia is developed from weakening of memory functions which is driven by emotion. The emotion is divided into positive and negative emotions. To strengthen the positive emotions would be directly be related to enhancement of cognitive function. Emotions, generated by both conscious and subconscious area, are considered to be initiated by brain(Top-down) or body(Bottom up). We argue that brain and body simultaneously interact each other to make emotions and the interactions could be evaluated quantitatively. In this study, 7 early dementia patients are evaluated before and after 8 weeks LENS trainings including the heart brain synchronicity and standard neuropsychological assessments. It is expected that LENS training facilitate balancing between consciousness and subconsciou and could change interactions between brain and body (especially, heart). The training is under process and final results will be presented at the conference.

References


Assessment of Memory Deficit and Malingering with a “Dual-Probe” Protocol, Using Incidentally Learned Information and Pictorial Stimuli (R,C)

Elena Labkovsky, PhD, Northwestern University, elenalabkovsky@yahoo.com
J. Peter Rosenfeld, PhD, Northwestern University

Abstract

Introduction:

Memory is one of the most vulnerable cognitive functions affected in cases of brain traumas, such as head injury, poisoning, radiation, viral infection, stroke, etc. Expectation to obtain monetary compensation increases motivation of “victims” to exaggerate and feign memory deficit. As estimated, about half of all the cases presented with “compromised memory” are actually cases of malingered psychological symptoms. Concerns raised by psychologists that the number of cases with feigned memory deficit continues growing led to increased interest in developing methods and techniques to identify malingers. One of the major challenges faced by researchers and practitioners utilizing neuropsychological tests and behavioral techniques to assess memory deficit is poor reliability of the conclusions of whether or not the patient has sustained real memory deficit or is malingering amnesia.

“A major roadblock to the study of malingered amnesia is the marked constraint on the verifiability of memory complaints. Unless an individual eventually admits that he or she has been intentionally deceptive, the clinician can never establish with confidence whether he or she has been malingering. Individuals willing to step forward and acknowledge their deceitfulness are rarely, under any circumstances, available. As a result, malingers usually cannot be identified independently of the outcome measures . . . being evaluated . . .” (Brandt, J. (1988) Malingered amnesia. In: R. Rogers (Ed.), Clinical Assessment of Malingering and Deception, Guilford Press, New York, NY, pp. 65-83).

Recently developed ERP-based tests reveal a high level of resistance to the effects of malingering
compared to neuropsychological/behavioral tests of memory (Rosenfeld, 2011).

Last year we demonstrated effectiveness of the “Dual-probe” ERP-based test to identify exaggeration of memory deficit (Labkovsky & Rosenfeld, 2011). The accuracy with the “Dual probe” protocol using personally-relevant (autobiographical) information reached 100% (with either one or both probes). The present study demonstrates effectiveness of the “Dual-probe” protocol utilizing incidentally-learned information and pictorial stimuli with 8 irrelevant stimuli in each of the two parts of a trial.

Methods:

In the “Dual-Probe” protocol each trial consists of two parts. There are about 400 trials in one block. Each of the two parts has one “Probe” (familiar to the subject item) and a few “Irrelevant” stimuli (unknown to the subject). In each part stimuli represent different domains. In the current study the first part of a trial consisted of pictorial stimuli. There was one probe (P1) and 8 irrelevants (I1.1-I1.8. All 8 irrelevants combined from part1 define Iall1). The probe was an image of the flash-drive (which subject was asked to steal and hide) and 8 irrelevants were pictures of items which were never shown to the subject before. In the second part of a trial the stimuli were names. The probe (P2) was name of the person from whose mailbox the flash-drive was taken by the subject and the 8 irrelevants were some random names (I2.1-I2.8, and Iall2= combined irrelevants in part2). There also was a target stimulus (Target) in the second part of a trial that was a task relevant name requiring a unique button press (with unique “assigned significance”, as in Johnson,1986). In each trial, the subject first saw an image (P1) or one of the 8 pictorial Irrelevants (I1.1-I1.8) followed by P2, or an “Irrelevant” from part2 (I2.1-I2.8) or a “Target.”

Subjects randomly pressed 1 of 5 buttons on one response box when they saw a pictorial stimulus (1st part), and they pressed 1 of 2 buttons on another response box to a name (2nd part).

Results:

For statistical analysis in the group with the Dual-probe protocol using incidentally learned information and pictorial stimuli (N=11), we implemented ANOVA and t-tests. ANOVA (2 parts of a trial x 2 stimulus types) showed significant stimulus type effect F(1,20)=39.363, p<.001. There was no significant group effect (between two parts of a trial) F(1,20)=1.192, p=.29 and no interaction (Pr vs.Iall x trial part).

Follow up t-tests revealed significant differences in the first part of a trial between P1 and Iall1 amplitudes t(10)=4.98, p<.001 and in the second part of a trial, between P2 and Iall2 t(10)= 3.83, p<.001. Hit rate was 100% with either P1(1 or 2) or P2(1 or 2) detected as recognizing the probes even if claiming a feigned stimulus recognition deficit.

Conclusions:

The “Dual-Probe” ERP-based protocol for assessment of memory deficit and malingering shows a high level of accuracy. When subjects try to feign cognitive impairment and, specifically deny recognition of familiar stimuli, the “Dual-Probe” (with pictorial stimuli) approach reflects the subject’s ability to recognize familiar stimuli. Thus, the “Dual-Probe” protocol can be used in situations where subjects are unable, or unwilling, to report their recollection for incidentally acquired or learned information.

Further research is required to investigate how introduction of countermeasures and changing number or irrelevants affect accuracy of the “Dual-Probe” protocol with pictorial stimuli and incidentally learned or rehearsed information.

References

Randomized Clinical Trial of Biofeedback in Patients with Multiple Sclerosis (R)

Michael McKee, PhD, Cleveland Clinic
Christine Moravec, PhD, Cleveland Clinic, Moravec@ccf.org
Elizabeth Grossman, BA, Cleveland Clinic
Gregory Bolwell, BA, Cleveland Clinic
Alison Reynard, PhD, Cleveland Clinic
Amanda Mills, BA, Cleveland Clinic
Dana Schneeberger, PhD, Cleveland Clinic

Abstract

Patients with multiple sclerosis (MS) have chronic symptoms, including unremitting fatigue, spasticity, bowel and bladder dysfunction, pain syndromes, cognitive impairment, mood disorders and gait disturbances. Underlying pathophysiology of this disease is complex, but includes dysfunction of the autonomic nervous system and activation of inflammatory cascades. We are testing the hypothesis that biofeedback-mediated stress management in patients with MS will improve autonomic balance, decrease the inflammatory state, reduce symptoms, and enhance quality of life. Patients between the ages of 18 and 90, with a complaint of fatigue interfering with daily activities, but with no recent disease exacerbation, are being enrolled in a randomized, controlled study. All patients receive an initial evaluation including psychobiologic stress reactivity to mental stress, manual muscle testing, numeric pain rating, the multiple sclerosis functional composite test, measurement of plasma norepinephrine, tumor necrosis factor alpha and C-reactive protein, and four questionnaires asking about overall health status, life engagement, anxiety and depression. After the initial evaluation, patients are randomized to the biofeedback (BF) group or the usual care (UC) group. Patients in the BF group return for eight weekly sessions of instruction in stress management, including finger temperature, skin conductance, respiration and heart rate variability (HRV) biofeedback training. Patients in the UC group continue as they normally would, with no additional sessions. After ten weeks, all patients return for a final evaluation, which duplicates the initial evaluation. Following the final evaluation session, patients in the UC group are offered a one hour educational session with a certified biofeedback therapist. They also receive the take-home aids which were provided to study patients (thermometer, relaxation CDs, stress management workbook). Data analysis seeks to determine whether MS patients are capable of learning self-regulation, and whether improved self-regulation results in enhanced quality of life, changes in plasma markers of autonomic nervous system activation or inflammation, and changes in the clinical condition, including pain perception, stress level, muscle strength,
walking, and memory.

References


An Interesting Finding

Mickey Ogan, Retired, oganicm@spro.net

Don Bars, PhD, Private Practice, dbars2001@yahoo.com

Abstract

Introduction:
The original goal of working with the Visual and Auditory Evoked Potential (VEP and AEP) data was to evaluate conscious vs. un/subconscious signals. Left vs. right electrodes from the International 10-20 system were evaluated. Initially, data from individuals of mixed gender and age were used. This data was initially processed with the assumption that the minds of each subject operated within System Theory and resulted in some comparisons being accurate to significant number of decimal places. At this point more consistent data relative to age and gender was acquired.

Methods:
Ultimately, the final data set analyzed consisted of sixty 13 year old males and forty 17 year old males. The data analyzed were from three test types. VEP from both pattern reversal and flash paradigms and an AEP odd ball paradigm. The AEP consisted of two files, both the standard tone and the target tone. The analysis, however, resulted in the data from both tones being precisely the same, therefore only the standard tone data was included in the final analysis. Preliminary attempts to analyze phase calculations were abandoned due to the data representing 256 time series points for 16 individual electrodes, each representing a quarter or half second time period. The time series data were converted to frequency bands of Delta (0-3.75 Hz), Theta (3.75-7.5 Hz), Alpha (7.5-15 Hz), Beta (15-30 Hz), Gamma1(30-60 Hz), Gamma2 (60-120 Hz), Gamma3 (>120 Hz). These frequency bands were then reconverted back to bands in a time series. A systems view of the normal "mapping" followed which caused the data from all 16 electrodes to be used to calculate the tilt variation of the 16 electrodes having identical values. Next, analysis was made utilizing auto and cross correlation of the Fast Fourier Transform data across all 16 active electrode signals. The data were then input to Microsoft Access 1A Data Base Management System (DBMS) for final analysis.

Results:
The Analysis separated two distinct types of data. The majority were in clusters that were relative to both individuals and coherence values. The clusters consisted of both auto and cross correlation values. Any data from individuals that did not match the cluster values were labeled as 'Outliers'. and were only considered in cross correlation.
functions. No outliers were identified in the autocorrelation portion of the data. While within the clusters the elements were similar, the ordering was not. Certain coherence types, specifically seven arbitrarily numbered pairs; 3&8, 4&6, 5&7, 13&22, 14&16, 17&20, plus 28&29 then the set of 18, 19, 25 and 26 always went together and were significant to fourteen decimal points. The autocorrelation elements were the numbers 2 - 8 while cross correlations types were numbered 11 - 29. Numbers 12 and 15 were always identically zero and were dropped from the analysis. The numbers 2, 11, 21 and 27 were individual and all these matches were geometrically related to the grid assigned the values. The "self similarity" within the clusters suggested a "fractal" view, therefore, the fractal dimension was assessed and, bottom line, all one hundred (100) boys mental processes were shown to be fractal. The dimensioning was done by summing all data for a given test type based on left side or right side then taking the logarithms of these data and calculating the ratio. The upper bound was limited to 1.0 by assigning the smaller of the two to the numerator of the ratio. Numbers less than or equal to 0.5 would have indicated the data was random while 0.5 to 1.5 was a fractal dimension, based upon criteria from, Fractal Structure in the Electroencephalogram, P.A. Watters, Complexity International, Vol. 5, 1998.

Discussion:
A primary significance of these results are that usual calculations based upon the "Bell shaped curve" would not be valid. The fractal dimensions are not an average but are related to each individual. This holds, however, only for this data set and therefore the individuals within the clusters. The similarity within the clusters combined with a variation in the ordering, as shown by the plotting of the data, was suggestive of the data being fractal. The meaning of the values within the clusters, which were unique to that cluster, or for that matter the values of individual records that didn't match the cluster values were beyond the scope of this study.

Logic: A Pain in the Anterior Cingulate (R)
Sherman Phillips, MA, University of Tennessee, sphill23@utk.edu
Rex Cannon, PhD, University of Tennessee
Debora Baldwin, PhD, University of Tennessee
Dominic Di Loreto, MA, University of Tennessee
Tiffany Shaw, MS, University of Tennessee

Abstract
Background:

The present study will utilize quantitative EEG and LORETA source localization, alongside hypothalamic-pituitary-adrenal (HPA) axis activity to facilitate real-time inquiry into active, cortical regions of interest (ROI) and stress reactivity associated with logic and deductive reason. To date, functional connectivity, neuronal and stress hormone cortisol activity underlying logic and deduction remain unclear.

Methods:

Eighteen study participants between the ages of 18 and 50 will participate in this study. Subjects will undergo continuous EEG recording in four conditions (eyes closed and eyes open baselines, learning (priming), and syllogism validation). Pre and post salivary cortisol sampling baselines will be collected before and after said experimental condition. Subject responses will be marked within the EEG record, extrapolated and compared for significance using standardized low-resolution electromagnetic tomography for 6,329 5mm3 voxels.

Results:

Previous research and statistical analyses, without cortisol measures, revealed current source density supporting evaluation processes in deduction were specific to left hemisphere, BA 30 parahippocampal gyrus, anterior cingulate and activity in right frontal lobe regarding beta frequency. Decisions compared to instruction (learning) produced increases in all frequency domains in various cortical regions. Delta frequency showed increase in BA 10, and a distributed pattern in the cingulate gyrus. Theta showed maximal increases at BA 10 and AC (BA 32), as well as right BA 18, 19, 37 and 40. Alpha frequency showed increase in left temporal and posterior cingulate (i.e., may reflect language processing and semantics). Beta showed increase in BA 19 (precuneus) and decrease in anterior regions.
Discussion:

Thus, this study will utilize a repeated measures design to analyze the underlying relationship between cortical activity and functional connectivity, along with HPA axis stress hormone cortisol reactivity associated with logic. Plausible interpretation of the data may denote the importance of low frequency bands and/or stress in information retrieval and network integration of syntax, semantics and other executive processes as a function of deductive inference decision making in the AC, PFC, and PCC.

References


On The Differences Between Topographical and LORETA Neurofeedback in Children and Adults

Tiffany Shaw, MS, University of Tennessee, tpetree1@utk.edu
Rex Cannon, PhD, University of Tennessee
Debora Baldwin, PhD, University of Tennessee
Dominic Di Loreto, MA, University of Tennessee
Sherman Phillips, MA, University of Tennessee

Abstract

Introduction:

This study distinguishes between the effects of a recently developed α-protocol designed for improvements in self-regulation and LORETA neurofeedback training of the α-frequency in the precuneus with the same function. Concentration of both protocols is in the left parieto-occipital cortex.

Methods:

This study consists of 6 children and 4 adults with ADHD who received 20 sessions of the alpha protocol 2 or 3 times per week contrasted with 2 age-similar children and age-similar adults who underwent the LNFB protocol over 15 – 20 consecutive weekdays.

Results:

There are similarities and differences specific to each protocol, with LNFB appearing to influence specific networks in a more definite fashion. Such disparities appear in fronto-parietal regions with global differences noted.

Discussion:

Neurofeedback, in general, operates under the auspices of neuroplasticity and a neural-efficiency model.
Differences between topographical and LORETA neurofeedback exist and should be investigated; yet, there are numerous similarities as well. Clinical and research applications will be discussed.

References


An Integrative Approach to High Performance Evaluation and Training: Illustrated by Data of a Professional Boxer (C)

Leslie Sherlin, PhD, Neurotopia, lesliesherlin@mac.com
Michael Gervais, Neurotopia
Chris Talley, Neurotopia
Noel Larson, Neurotopia
Andy Walshe, Neurotopia

Abstract

Introduction
In the clinical applications of mental health it is well recognized that there are multiple contributors to pathology and also multiple contributors to wellness. Largely in the past, athletic performance primarily has focused on training the body from an anatomical (e.g., Manning & Pickup, 1998), muscular (e.g., Anderson et al., 1991), and cardiovascular performance (Noakes, 2000) approach. As understanding and technology have emerged, the focus has broadened from simply being exercise physiology to a sports and high performance science including the interrelationships and collaborations between physiology, psychology, technology, coaching, biomechanics, and nutrition.

Methods
The model we employ addresses each of the core areas for maximum athlete performance. The body and brain need adequate nutrition in order to supply cells with fuel. In addition to assuring the intake of the optimal nutrients, it is critical to limit exposure to toxins and allergens that may be contained in food or other substances ingested. Physical capabilities also have to be measured and subsequently trained. Sports psychology techniques of measuring and training mental and emotional skills yield an understanding of the intrinsic belief systems of the individual that can be refined to enhance high performance attainment. The specific brain electrical activity measurements that can be acquired from quantitative electroencephalographic (QEEG) techniques have been well established to reflect levels of cognitive engagement and arousal regulation. Engagement and arousal are critical among the many variables that
contribute to brain states, and an athlete who can exercise volitional control of these aspects of brain state likely has a distinct advantage during training and competition. In combination, the areas mentioned contribute to a model that is designed to address each core aspect of the individual athlete to enhance the oft-allusive drive for maximum performance.

To illustrate the approach a case example was analyzed. The subject is a male professional heavyweight boxer. To measure the sport mindset The Attentional and Interpersonal Styles (TAIS) inventory was administered (Nideffer, 1976). TAIS is a 144-item self-report subjective assessment tool designed to measure concentration styles and interpersonal skills involved in effective decision-making in high-pressure situations. Quantitative electropherephalography (QEEG) was recorded from 19 electrode sites and spectral analyses were computed in classically defined frequency bands of delta, theta, alpha and beta in both absolute and relative power measures. Additionally a new metric of combined behavioral information and QEEG spectral data was utilized to evaluate the athletes “NeuroProfile.” Nutritional information was gathered from a blood and urine sample that in addition to a complete blood count (CBC), sex hormone panel, and basic lipoprotein screening, this analysis involves testing for a large number of common allergies, fatty acids and their derivatives, amino acids, vitamins/minerals, heavy metal toxicities, impaired detoxification indicators, and gut microbial imbalances.

Results
The TAIS data was used to help enhance identified psychological skills and was used in conjunction with neurofeedback protocols. The QEEG analysis demonstrated findings that included statistically significant deficits of alpha frequency and elevations of the beta frequency in the parietal and occipital cortex. These findings are consistent with a presentation consistent with cortical overarousal and could contribute to anxiety type presentation. Nutritional results showed serious allergies to milk, eggs, and mustard, a frank vitamin D deficiency, a nearly complete absence of omega-3 fatty acids, heavy metal scores that were well above normal levels, and a significant gut microbial imbalance. Correlation analysis were computed between these measures and interesting trends were found all consistent with the athletes subjective and objective presentation.

Conclusion
Our approach requires coordinating a group of specialists in a wide variety of performance disciplines (i.e., coaches, psychologists, nutritionists, psychophysiologists, biomechanists, and strength and conditioning experts) to deliver an integrated program of performance support. A clear understanding of the integration of the components of performance is becoming more and more critical. None of the components of performance can be considered in isolation any longer and the interrelationships of these core areas is the space in which many of the next strides in high performance training will be made.

References


Infra-low Frequency Neurofeedback: Results of a School-Based Program (R,C)
Mark Smith, MSW, Private Practice, marksmith50@verizon.net
Abstract
Infra-Low-Frequency (ILF) neurofeedback is a new paradigm in biofeedback training that is generating interest among practitioners due to clinical reports of its efficacy with a wide range of client presentations. This poster presentation reports on an infra-low frequency neurofeedback pilot program in a special needs school in New York City. Preliminary results from that program include pre/post CBCL and ATEC scales in additional to clinical reports.

Results of Pilot School Program:

In total, we have had sixteen students in the program. Thirteen of the sixteen students had a positive response that involved either: a significant reduction of tantruming behavior and/or a reduction or elimination of psychotropic medication and/or improved ability to sustain attention during class resulting in academic progress. Of the remaining three students: two have just begun the program and one had a positive response that is confounded by the initiation of an SSRI at the beginning of the training. This subject, a selectively mute child, achieved a remarkable improvement in symptoms after approximately one week on the SSRI and two weeks with neurofeedback.

In addition to presenting the behavioral data obtained from the school-based program, the poster will present pre and post QEEGs for individuals who have undergone ILF treatment. Recent equipment and software advancements have allowed for simultaneous 19-channel recording and ILF training. This innovation has provided a window on the mechanism of bipolar ILF training. The value of QEEG in predicting treatment responders, treatment planning, and determining treatment outcomes will be discussed. We will also outline some proposed mechanisms of action for ILF neurofeedback and will propose a research design for uncovering the mechanism.

References


Hughes SW, Lorincz ML, Parri HR, Crunelli V (2012) Infra-slow (<0.1 Hz) oscillations in thalamic relay nuclei: basic mechanisms and significance to health and disease states. Progress in Brain Research, 193C: 145-162.


Marshall L, Molle M, Fehm H, Born J (2000) Changes in direct current (DC) potentials and infra-slow EEG oscillations at the onset of the luteinizing hormone (LH) pulse. European Journal of Neuroscience,
Vol. 12 pp. 3935-3943.


**Insights Gained from Over 3 Years of Full-cap Z-score Neurofeedback: Towards a New Paradigm (R,C)**

Nancy Wigton, MA, Grand Canyon University, nwig@cox.net

Genomary Krigbaum, PsyD, Grand Canyon University

**Abstract**

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 (Collura, Guan, Tarrant, Bailey, & Starr, 2010). However in the initial application of ZNF the maximum number of channels that could be trained at one time was 4 and training was limited to the linked-ears normative database. In 2009 the use of full-cap ZNF (fcZNF) greatly expands the number of scalp locations and measures and included 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. Since the introduction of fcZNF various approaches within this modality have been developed. Also, given that this is a relatively new approach to NF, to-date there have been no multi-year follow-up data presented or published which addresses the question of longer-term benefit of this new technique.

This will be an overview presentation of multiple case histories with emphasis on the practical applications for the NF clinician. As one of the first clinicians in private practice to incorporate surface fcZNF the presenter will share what has been learned after three years of using four primary approaches to fcZNF, on two different platforms. The presenter’s application of fcZNF is an eclectic approach resulting in a blended matrix which has led to significant advances in terms of shortened treatment time. As a result of observations over the years, the presenter believes certain elements of a changed paradigm are now possible.

**Method:**

Treatment records of a private neurofeedback practice are reviewed. Pre and post QEEG comparisons, case examples with pre and post outcome measures, and where possible, multiple year follow-ups from this new neurofeedback technique are examined.

**Results:**

As of the writing of this abstract this investigation is still in process. At the time of the conference applicable statistical analysis will be presented. The different fcZNF approaches to be discussed will be the Linked Ear montage with and without symptom checklist, Laplacian montage, and PZOK-19channel. Suggestions for a framework of a new paradigm will be presented.

**References**


WS 1: Fundamentals in Research Methodology: An ISNR Research Foundation Workshop

(Lecture)

David Trudeau, MD, trude003@gmail.com
Estate Sokhadze, PhD, University of Louisville, tato.sokhadze@louisville.edu
Rex Cannon, PhD, University of Tennessee, rcannon2@utk.edu

Credits: 3

Level of Difficulty: Intermediate

Abstract
This workshop, presented by top researchers in neuromodulation, will focus on fundamental research design principles, and show their usefulness in researching neuromodulation, which is an operant conditioning and behavioral modality, which is a highly applied field. It will compare common flaws and successful study designs in recent research, including specific examples of both flaws and successful designs. Some questions to be discussed are: Is it possible to do RCT of operant conditioning that is truly double blinded and the active condition is therapeutic? What can we learn from comparison studies? From physiologic outcome measure studies? This is a very important workshop for those interested in clinical trials and students embarking on their theses and/or dissertations.

References


Goals/Objectives
Discern the problems inherent in randomized controlled trials and blinding issues in researching operant conditioning and behavioral modalities.

Know common flaws in published research in the field and learn how to avoid them.

Utilize basic research methodology in neuromodulation such as rTMS and neurofeedback.
Outline
This workshop, presented by top researchers in neuromodulation, will focus on fundamental research design principles (.75 hr), and show their usefulness in researching neuromodulation (.75 hr), which is an operant conditioning and behavioral modality. It will also discuss common flaws (.75 hr) and successful study designs in recent research (.75 hr)

Financial Interest: No financial interests.

WS 2: Autism Spectrum Disorders: Integrating Clinical Knowledge and Individual Symptoms and Neurophysiology in the Formation of Neurofeedback Treatment Plans
(Lecture, Demonstration)
Robert Coben, PhD, Private Practice, drcoben@gmail.com

Credits: 3

Level of Difficulty: Advanced

Abstract
The prevalence of Autism Spectrum Disorders (ASD) continues to rise at an alarming rate (CDC, 2012). Providing effective treatment options is becoming more and more crucial for this population. While some treatments have shown promise and have preliminary empirical support (i.e., Neurofeedback (Coben & Wagner, 2011), Hyperbaric Oxygen Therapy (Rossignol, 2007)), the most pressing clinical issue is how to individualize treatment to the needs of the individual.

This workshop will focus on enhancing knowledge about ASDs, treatment options, symptom constellations, and neurophysiological mechanisms so participants will be able to start integrating such information in the formation of neurofeedback protocols and treatment plans. Empirical data will be presented from neurofeedback trials to form the basis for effective treatment protocols. This will include a focus on the following:

Review of ASD symptoms, diagnoses, epidemiology, potential causes and neurophysiological findings.

Current therapies and their empirical support.

Using neurofeedback to treat ASD: Empirical data regarding EEG, neuroimaging and neuropsychological research will be integrated in order to maximize the evaluation and therapy course and avoid common pitfalls while working with this population.

Discussion and questions.

The true value of this workshop is an understanding for a neuroscientific level what leads to autistic symptoms and how to remedy them. Individualization of treatment is at the heart of this approach, which has led to an overall success rate in approximately 90 – 95% of the cases.

References


Goals/Objectives
Discuss the symptoms and neurophysiological findings associated with autistic disorders.

Review available treatments, especially alternative one’s.

Be able to start to integrate knowledge of symptoms and neurophysiology in the formation of neurofeedback protocols.

Outline
Review of ASD symptoms, diagnoses, epidemiology, potential causes and neurophysiological findings.

Available treatments and their current empirical support.

Using Neurofeedback to treat ASD: Integrating knowledge of ASD with individual symptoms and neurophysiological findings.

Discussion and questions.

Financial Interest: There is no financial relationship with any software or product discussed in this workshop.

WS 3: Video Games As Exceptional Learning Environments (Lecture, Discussion, Demonstration)
C. Shawn Green, PhD, University of Wisconsin, csgreen2@wisc.edu

Credits: 3

Level of Difficulty: Basic to Advanced

Abstract
Those who develop off-the-shelf video games have generally done so with one goal in mind – to make a product that is incredibly entertaining and which is thus a product that consumers will want to buy. However, in accomplishing exactly this, whether intentionally or otherwise, they have exploited principles that have been the focus of more than one hundred years of research in neuroscience and psychology to produce an experience that changes the brain and behavior unlike any training paradigm that came before (Gentile & Gentile, 2008; Green & Bavelier, 2008).

In this workshop we’ll examine the basic neuroscientific and psychological principles that are known to affect the rate, depth, and generality of learning. This includes topics such as the effects of arousal and motivation - mediated by the cholinergic basal forebrain system (Kilgard & Merzenich, 1998), reward - mediated by the mesocortical dopaminergic pathway (Bao, Chan, & Merzenich, 2001), reward scheduling (Ferster & Skinner, 1957), feedback (Roelfsema, van Ooyen, & Watanabe, 2010), active learning (Gee, 2003), level of difficulty (Ahissar & Hochstein, 1997), variety (Schmidt & Bjork, 1992), hierarchical learning (Harlow, 1949), and so-called “spiral curricula” (Bruner, 1960). More specifically, we’ll consider how commercial video games embody and/or implement these principles to produce efficient learning and perhaps just as importantly, we’ll also discuss the myriad ways in which most games designed for practical purposes (e.g. “brain trainers” or educational software) neglect these principles (thus resulting in the derisive nickname of “chocolate covered broccoli”).
Finally, using the video game implementations as a guide, we'll attempt to devise ways (using video games or otherwise) to employ the basic learning principles to augment learning in the domains of most interest to the workshop participants (whether it be cognitive therapy, education, rehabilitation, etc).

**References**


**Goals/Objectives**

Understand many of the principles of effective learning regimens including arousal, reward and reward scheduling, feedback, and variety as well as the neuroscientific bases for this principles.

Appreciate how these principles are effectively implemented in modern commercial video games (and neglected in many modern games designed for practical purposes.

Have a framework for utilizing the knowledge in their own domain of interest.

**Outline**

For each topic: psychology of XX, neuroscience of XX, implementation of XX in good games, implementation of XX in bad games, how to utilize XX in own domain of interest:

- arousal and motivation
- reward
- feedback
WS 4: Neurofeedback Intermediate - Advanced (BCIA Review Course) (Lecture)

Lynda Thompson, PhD, ADD Centre, landmthompson@gmail.com
Michael Thompson, MD, ADD Centre, landmthompson@gmail.com

Credits: 3

Level of Difficulty: Intermediate

Abstract
This workshop covers areas from the BCIA blueprint of knowledge and skills, information relevant to all neurofeedback practitioners. Basic definitions and descriptions will be discussed. It will cover the highlights concerning the history of neurofeedback, research criteria for determining efficacy, efficacy levels of various disorders treated with NFB, basic neurophysiology & neuroanatomy (very brief) as these apply to assessment for biofeedback interventions, source of the electroencephalogram (EEG), instrumentation, procedures for assessment and intervention. It additionally comments on adjunctive techniques, including biofeedback and relaxation.

Method:
This course is a didactic presentation that provides a very brief review of basic knowledge and will cover selected topics from the areas that comprise the Blueprint of Knowledge for specialty certification in EEG biofeedback developed by the BCIA. Goals are that participants will be able to answer questions on material that could legitimately be covered in a BCIA examination on EEG Biofeedback (that is, material that has been published, as contrasted to ideas based on clinical impressions). For example, they will be able to answer questions regarding EEG data collection and instrumentation including: impedance versus resistance, differential amplifier, sampling rates, filters and so on and understand EEG assessment (one, two and 19 channels, brain maps, LORETA, data bases, EEG artifacts, normal and abnormal waveforms, common findings in disorders where neurofeedback is used). Methods for obtaining accurate data and interpreting this information will be covered. Additionally, they will be able to demonstrate an understanding of how learning theory (especially operant conditioning) applies to EEG biofeedback, discuss basic neurophysiology relevant to interventions that use the EEG, and briefly relate basic information on other related topics including: HRV, ERPs, ethics, statistics, and so on. Blueprint areas include:

Section I Section II Section III Section IV

Overview of Biofeedback, Neurofeedback and Learning Physiological Basis of the Electroencephalogram and basic neuroanatomy. Measuring The EEG: Instruments & Electronics Brief Overview of Statistics and Research Design with an emphasis on criteria for evaluating efficacy

Section V Section VI

Section VII
Psychopharmacology Overview as it relates to assessment and training. Fundamentals of Intervention: Choice of Electrode Placement, Channels, Bandwidths and Adjunctive Techniques Professional conduct: very brief review

Results & Conclusions These headings do not have equal emphasis in this workshop. Feedback concerning the workshop has been that it increases the confidence level and successful outcomes for people taking the BCIA examination.

BCIA REVIEW Section I Overview of Biofeedback, Neurofeedback and Learning Section II Physiological Basis of the Electroencephalogram and basic neuroanatomy. Section III Measuring The EEG: Instruments & Electronics Section IV Brief Overview of Statistics and Research Design with an emphasis on criteria for evaluating efficacy Section V Psychopharmacology Overview as it relates to assessment and training. Section VI Fundamentals of Intervention: Choice of Electrode Placement, Channels, Bandwidths and Adjunctive Techniques Section VII Professional conduct: brief review

References


Goals/Objectives
Discuss EEG data collection and interpretation using common terms including: referential, sequential, and laplacian montages; active, reference, and ground electrodes, digital versus analogue recording, QEEG, LORETA.

Discuss instrumentation including: impedance versus resistance, differential amplifier, sampling rates, high and low pass filters.

List and describe common artifacts including: eye movement, muscle tension, cardiac, cardioballistic, electrode movement.

Describe normal and abnormal waveforms.

Describe common findings in disorders where neurofeedback is used including: Seizure disorders, ADHD, anxiety, depression.

Outline how learning theory (especially operant conditioning) applies to EEG biofeedback.

List three Brodmann Areas that are common targets of NFB intervention and name a disorder where that area is important.
Describe levels of efficacy for NFB.

Define event related potential (ERP) and describe the importance of latency and amplitude with one example (e.g., ADHD or dementia).

List and describe four basic ethical principles.

Define basic statistical terms.

Name five structures that comprise the Basal Ganglia.

Name four neural networks.

Define SDNN and describe how heart rate variability training can improve this measurement.

**Outline**

First Hour

1. History, Theory & Assessment
2. TERMS: Frequency (Hz), Amplitude (µV), Magnitude, Power (pW)
3. 10:20 System; Montages
4. EEG waves: Delta, Theta, Alpha, Beta, Gamma
5. Other Terms: QEEG, Phase, Coherence, LORETA
6. EEG Band Widths correspond to functions (ADHD examples)
7. Operant & Classical Conditioning
9. Instrument: High pass & Low Pass Filters
10. Impedance & Differential Amplifier; Optical Isolation

Second Hour

11. Basics of EEG Assessment
12. Brodmann Areas & Limbic System
13. Data Bases
14. Neurotransmitters
15. Evoked Potentials, slow cortical potentials
16. Psychophysiological Variables & Biofeedback
17. Artifacts & Medication Effects
18. Side effects NFB
19. Side effects BFB

Third Hour

20. Disorders with illustrations of Assessment EEG findings
21. Levels of efficacy
22. Training Paradigms using both NFB & BFB
WS 5: A Clinician’s Guide to Understanding Recent Developments in Neurofeedback: Amplitude, Live Z-score and sLORETA Training Explained
(Lecture, Demonstration)
Thomas Collura, PhD, BrainMaster Technologies, tomc1@brainm.com
Penijean Rutter, Stress Therapy Solutions, Penijean@gmail.com
Ronald Bonnstetter, University of Nebraska, rjb@unl.edu

Credits: 3

Level of Difficulty: Intermediate

Abstract
With EEG technology and training software developing at blistering speeds, clinicians new to the field of neurofeedback are expressing confusion at the differences between the available training options, and even experienced practitioners are struggling to understand and incorporate recent technological innovations in a clinical practice.

This workshop will provide a brief analysis of the literature, history and development of conventional amplitude neurofeedback, live Z-score methods, and sLORETA training, while evaluating differences and similarities between the highlighted approaches. A review of the relevant scientific terms and principles will include remarks on the emerging role of QEEG in protocol selection, an examination of the available models for neurofeedback training, a discussion of the differences between standard types of acquisition and feedback, and an overview of how to make educated choices regarding clinical interventions appropriate to the client presentation based on an introduction of the technical information critical to such a process.

The intent of this workshop is to equip the attendee with a working knowledge of the scientific paradigms that underlie different theoretical approaches to neurofeedback, to increase the skill with which protocols are chosen or created through improved understanding of the conceptual framework behind the design of available training software, and to enhance individual clinical competency through technical education.

References


Goals/Objectives
Describe neurofeedback paradigms and their published clinical results.
Interpret QEEG results of neurofeedback therapy in clinical terms.
Demonstrate combined methodologies using selected cases of live Z-score, amplitude, and sLORETA methods.

Outline
Overview of neurofeedback paradigms (1 Hour)
Principles of protocol combination: conventional + innovative (1 Hour)
Case studies with QEEG pre and post outcome measures (1 Hour)

Financial Interest: Dr. Collura has a financial interest in BrainMaster Technologies Inc. Part of the workshop will describe products provided by BrainMaster Technologies, along with other providers. "As part of StressTherapy Solutions, Inc. faculty, Penije Rutter has no financial gain or interest in BrainMaster Technologies. Dr. Bonnstetter is a Senior Vice President of Target Training International.

WS 6: Psychopharmacology of Depression
(Lecture)
Fredric Shaffer, PhD, Truman State University, fredricshaffer@gmail.com

Credits: 3

Level of Difficulty: Intermediate

Abstract
This 3 contact-hour workshop is designed for biofeedback/neurofeedback practitioners, psychologists, clinical counselors, clinical social workers, marriage and family therapists, nurses, physicians, and other
health care professionals and academicians interested in pharmacological and biofeedback and neurofeedback treatment of depression. This workshop will examine the neurogenic theory of depression, review the mechanisms, efficacy, side effects, and EEG effects of first-generation, second-generation, and dual-action antidepressants, summarize the lessons of the STAR*D study, and assess the promise of HRV biofeedback and neurofeedback as alternatives to antidepressants. Attendees will learn that antidepressants may relieve depression by increasing levels of neurotrophins like BDNF and restoring neurogenesis in the hippocampus. They will discover that while successive generations of antidepressants have reduced toxicity and improved side effect profiles, they have not increased the onset of action or reduced the number of treatment-resistant patients. Finally, they will realize that HRV biofeedback and neurofeedback are possibly efficacious in the treatment of depression.

Topics


Efficacy guidelines for pharmaceutical and behavioral treatments (10 min) 1. Efficacy levels 2. Effect size

First-generation antidepressants (30 min).
7. Tricyclic antidepressants A. Mechanism of action B. Pharmacological effects C. Side effects D. EEG effects

8. Monoamine oxidase inhibitors A. Mechanism of action B. Pharmacological effects C. Side effects D. EEG effects

Second-generation antidepressants (30 min). 1. Selective serotonin reuptake inhibitors
A. Mechanism of action B. Pharmacological effects C. Side effects D. EEG effects

Dual-action antidepressants (30 min) 1. Serzone, Savella, Effexor, Cymbalta, Remeron
A. Mechanism of action B. Pharmacological effects C. Side effects D. EEG effects

2. Dopamine-norepinephrine reuptake inhibitor (Bupropion) A. Mechanism of action B. Pharmacological effects C. Side effects D. EEG effects

3. Selective norepinephrine reuptake inhibitors (Vestra, Strattera) A. Mechanism of action B. Pharmacological effects C. Side effects D. EEG effects

Lessons from the STAR*D study (15 min) A. Study description B. Study conclusions

HRV biofeedback and neurofeedback for depression (35 min) 1. HRV biofeedback for depression A. Treatment protocol B. Mechanism of action C. Efficacy

2. Neurofeedback for depression A. Treatment protocol B. Mechanism of action C. Efficacy
References


Saarrelainen, T., et al. (2003). Activation of the TrkB neurotrophin receptor is induced by antidepressant drugs and is required for antidepressant-induced behavioral effects. Journal of Neuroscience, 23, 349-357.


**Goals/Objectives**

Explain the neurogenic theory of depression.

Describe the mechanism, efficacy, and side effects of first-generation antidepressants.

Describe the mechanism, efficacy, and side effects of second-generation antidepressants.

Describe the mechanism, efficacy, and side effects of dual-action antidepressants.

Summarize the major findings of the STAR*D study.

Discuss the behavioral alternatives of HRV biofeedback and neurofeedback for depression.

**Outline**

Neurogenic theory of depression (30 min).

First-generation antidepressants (30 min)

Second-generation antidepressants (30 min)

Dual-action antidepressants (30 min)

Lessons from the STAR*D study (15 min)
Abstract
Day 1: For professionals who have not had extensive experience in assessment, differential diagnostic work-up, and basic interventions, for Asperger’s (AS) or Autism (AD).

Day 2: For professionals experienced with autistic spectrum disorder (ASD), an emphasis on how QEEG and psychophysiological assessments lead to effective intervention.

Goal: Attendees for both half days of this workshop will become familiar with how symptoms differ between Asperger’s and autism. They will be able to outline, on the basis of functional Neuroanatomy (which includes discussion of Brodmann Areas, neural networks and connections, including vagal inputs to the medulla and brain stem connections to basal ganglia, thalamus, and cortex), why a combination of NFB + BFB + Strategies improves social functioning in addition to resulting in significant improvements in scores on academic, intelligence, and attention measures (Thompson, Thompson, & Reid, 2010). The participants will learn how autistic spectrum disorders (ASDs) have major difficulties in at least three major networks: executive, affect, and the default network. Every patient will have a different “balance” of involvement or difficulties related to these networks. These three networks can be influenced by Neurofeedback at various sites over the central midline structures (CMS). They are also altered by means of biofeedback and, in particular, by Heart Rate Variability (HRV) training that influences the same CMS. The neuroanatomy and connections of the CMSs, and in particular of areas of the anterior cingulate and how they are involved in the networks, is highlighted.

Abstract for Day 1

Presenters will outline, with case examples, the major symptoms of Autism and Asperger’s Disorder. The workshop will include a basic over-view of cortical areas that are dysfunctional in the autistic spectrum disorders (ASD) with LORETA examples of how these are seen in the QEEG assessment, which is expanded in day 2. Functional significance of cortical areas is partially elucidated in the Brodmann Areas booklet (Thompson, Thompson, & Wu, 2008) and those findings will be mentioned. Psychophysiological stress assessments include respiration and heart rate to provide a baseline for heart rate variability (HRV) training. The EEG plus peripheral measures reflect the anxiety components of ASDs. Breathing, heart rate, electrodermal response, and temperature can all show the negative effects of even minor stressors on all aspects of the functioning of those with ASD. Neuroanatomical connections suggest how HRV training may influence neural networks. Regarding assessment, presenters discuss how high tactile sensitivity in some clients means you begin with only a single channel QEEG assessment (emphasized in day 1) and follow-up later with a 19-channel QEEG once SMR up-training has modified the tactile sensitivity and the client has learned to minimize EMG artifact. EEG interpretations used to illustrate findings range from raw EEG data to quantitative analysis with LORETA and, when possible, event related potentials. (ERPs are mentioned but not dealt with in detail in this workshop). This first day will include a review of methods and results of researchers and clinicians...
working with neurofeedback who have published in the area of ASD, such as Coben, Kouijzer, Linden, Pineda, Sichel & Fehmi, Thompson & Thompson. Similarities and differences between the methods described in these publications will be noted. For example, a similarity is that these authors all use careful QEEG assessment as the basis for their interventions. There were differences in training parameters, with some, such as Coben, placing an emphasis on coherence, while Pineda targeted mu waves measured across the sensorimotor strip, and others emphasized different frequency bands and initial sites for training. The Thompsons combine neurofeedback (NF) with biofeedback and strategies, publishing a case series of 159 cases. Note that it is beyond the scope of this workshop to discuss more traditional interventions and research concerning interventions such as ABA, IBI, speech and language therapy, special diets, etc., though use of medications (stimulants, anti-depressants, anti-psychotic medications as discussed in the Sloman review) will be touched on briefly, since drugs are sometimes combined with NF interventions.

Assessment results will lead to discussion of how interventions are initially focused on decreasing anxiety and dealing with the ADHD symptoms found in these patients. A multimodal treatment approach will be outlined that addresses the four key groups of symptoms: (1.) anxiety and affect modulation, (2.) ADHD symptoms of inattention and impulsivity, (3.) empathy, affect interpretation and expression and maintaining social interactions, and (4.) executive functioning difficulties. These interventions typically combine neurofeedback (NFB), biofeedback (BFB), and strategies.

References


Bojana Knezevic, Lynda Thompson, and Michael Thompson (2010) Using the Tower of London to assess
The impact of Neurofeedback training in clients with Asperger’s Syndrome, Journal of Neurotherapy.


**Goals/Objectives**
List the primary symptoms observed in Asperger’s Syndrome.

List differences between Autism and Asperger’s.

Relate these symptoms to the Neural Networks that are most likely to be involved.

List reasons why initial NFB training over central midline structures, based on QEEG and LORETA assessment findings, is likely to produce improvement in some of the core symptoms of people with Asperger’s.

State why up training of high frequency alpha may be contraindicated in some patients.

State why up training of 14 Hz may be contraindicated in some patients.

Outline, with reference to: affect, executive, and distress networks, and the hypothalamic-pituitary-adrenal axis (HPA), why HRV training may have a positive influence on the outcomes of patients with Asperger’s.

**Outline**
1st hr – Discussion of symptoms of Asperger’s and Autism.

2nd hr – Describe the assessment including QEEG and Psychophysiological Assessment peripheral biofeedback variables.

3rd hr – Outline basic interventions to decrease symptoms of: Anxiety, inattention, impulsivity, and begin on appropriate social interactions.

**Financial Interest:** Lynda Thompson is co-author of THE A.D.D. BOOK. Michael and Lynda are co-authors of SETTING UP FOR CLINICAL SUCCESS. Michael and Lynda Thompson are co-authors of THE NEUROFEEDBACK BOOK. It is likely that these books may be on sale at the meeting. The authors will state their interest in these books at the workshop.

**Friday, September 21, 2012**

**WS 8: Ethics and Neurofeedback: Thoughtful Discussions**
*(Lecture, Experiential, Discussion)*

Rex Cannon, PhD, University of Tennessee, rcannon2@utk.edu

**Credits:** 3

**Level of Difficulty:** Basic to Advanced

**Abstract**
This workshop provides an open-discussion into the ethical principles involved in neurofeedback and brain mapping. In recent years the technological advances in quantitative EEG, source localization methods and operant conditioning of the EEG have increased at an exponential rate. As such it becomes prudent to
discuss potential ethical dilemmas that arise and steps we might take to guide our clinical and research outcomes. We will cover neuroanatomy and potential issues associated with spatial specific training. We will engage in the process of decision trees to facilitate the best clinical and research decisions. We will identify key concepts and evaluate our current referral procedures and develop individual plans for ethical practice and networking. As a profession we follow ethical guidelines maintained by other professional organizations. Neurofeedback, however, as it advances presents many issues that may not fall within these particular auspices.

References
Koocher & Keith-Spiegel (2008). Ethics and the Mental Health Professions: Standards and Cases

Goals/Objectives
Attendees will understand ethical dilemmas that may arise in the clinical and research setting and gain resources to aid in formulating ethically sound decisions.

Attendees will gain education in many of the terms and modalities in use today and as such gain experience in communicating technologically advanced terms to patients and peers (e.g. what do we tell the patient about anomalies and how do we minimize potential damage?)

Attendees will engage in discussion to practice ranking ethical problems and resource building to find ethical solutions.

The group and attendees with engage in the process of developing individual and group ethical solutions to potential problems that may arise in the clinical and research setting.

By the end of the workshop attendees will be able to describe a problem, determine whether there is an ethical issue or dilemma, identify and rank key values and principles, gather and process information, review applicable codes or cases, select a course of action, enact the plan of action and critically evaluate the results.

Outline
Neurofeedback: History, challenges and future 30 min

Ethical dilemmas, fundamental processes (APA, AMA, ACA) 30 min Potential issues and ethical dilemmas in clinical and research settings using neurofeedback and brain mapping 40 min Ethical decision making and evaluating ethical dilemmas 30 min Planning and taking action 30 min Critical evaluation of plans, actions and resource effectiveness 20 min

Financial Interest: Nothing to disclose.

WS 10: Breaking Down Barriers to Peak Performance Brain Training™ in Elite Athletes
(Lecture, Experiential)
Leslie Sherlin, PhD, Neurotopia, lsherlinphd@neurotopia.com
Noel Larson, MA, Neurotopia, Noel.Larson@gmail.com

Credits: 3
Level of Difficulty: Intermediate

Abstract
Self-regulation of attention, arousal, and motor control are favorable skills for athletes, thus performance training directed at improving these specific abilities or related talents is a valuable pursuit (Vernon, 2005).
Due to the growing interest in the application of neurofeedback training among athletes (Harung et al., 2011), the scientific team at Neurotopia began collecting quantitative electroencephalography (QEEG) data alongside various psychological testing (questionnaires, CPT, etc) from elite athletes. This led to the development of a new database, the BrainBankTM, that is comprised of data from elite athletes of varying developmental levels in many sports, e.g., baseball, track and field, basketball, football, action sports, etc. Over the past three years we discovered that this population is unique and should not be approached with the traditional model typically employed with clinical populations when using neurofeedback. We had now overcome the barrier of understanding the elite performer, yet had a barrier of effectively communicating this information to the athlete.

The BrainBankTM data was used to develop a new format for presenting sport relevant QEEG and brain performance results to athletes and their support staff. The NeuroPerformance ProfileTM provides a standardized report and communication tool that integrates brain performance outcomes into comprehensible language and constructs of sport psychology. The NeuroPerformance Profile provides the basis to training protocols designed to impact sport performance variables. Subsequently research was carried out to test validity of the Performance Brain TrainingTM protocols to sport specific outcomes.

Finally, the practical barriers of ease of use, invasiveness, opportunity for training and quality control concerns (capability of the athlete to carry out training accurately and effectively) required the development of a new platform for the training. This is implemented in a newly designed dry sensor headset integrated with software on portable iOS devices. This workshop will provide the attendee with the presentation of these barriers encountered while developing an elite athlete brain training program. Theoretical models, validation research and applications will be presented.

References


Goals/Objectives
Attendee will understand constructs necessary for implementing neurofeedback in an athlete population.

Attendee will understand barriers to entry for peak performance populations.

Attendee will be familiar with the background literature in neurofeedback and sports performance.

Outline
Model for understanding athlete population’s psychophysiology.

Constructs for evaluating athlete brain performance.

Interpretive elements for athlete brain performance data.

Platform for Performance Brain TrainingTM application for athletes.
Financial Interest: Both presenters are employees of Neurotopia, Inc the company that has sponsored all research and products resulting from the research presented in this workshop.

WS 11: Infra-low Frequency Training in Clinical Practice
(Lecture, Experiential)
Mark Smith, MSW, Private Practice, marksmith50@verizon.net

Credits: 3

Level of Difficulty: Basic

Abstract
Infra-low frequency (ILF) oscillations, first identified by Russian researchers in the 1950's (Aladjalova, 1957, 1964) and later corroborated by Joe Kamiya (1973) and others, have become a signal of significant interest to researchers recently. Research suggests that the infra-low signal underlies the excitability dynamics of cortical networks (Vanhataloo, 2004). The phase of infra- slow fluctuations is robustly correlated with the amplitudes of 1-40 hertz oscillations. Further, it appears to be a direct electrophysiological correlate for slow fluctuations in human psychophysical performance (Monto 2008). Marshall(2000) demonstrated a coupling of hypothalamic-pituitary activity with an increase in the magnitude of the infra-low frequency signal. Most recently, research has suggested that very slow oscillations are associated with the Default Mode Network of the human cerebral cortex and appear to be related to ADHD symptom status (Broyd, 2011).

It was almost fifty years ago that Aladjalova (1964) proposed a role for the infra-low frequencies in hypothalamic functioning. His animal research discovered that stimulation of the ventromedial nucleus of the hypothalamus resulted in high-amplitude slow waves with a long latent period appearing in the EEG of both hemispheres. Aladjalova established that the ILF became intensified by agents that elicit a defense reaction similar to the response to "stress." More recently, Marshall (2000) supported this association between infra-slow oscillations and hypothalamic function by demonstrating the coupling of increased ILF power and hypothalamo-pituitary hormone release.

The hypothalamus plays an integral role in affective response, as well as, playing a vital role in maintaining homeostasis. It is the control center for many autonomic functions of the peripheral nervous system. Hypothalamic hormones control pituitary hormone secretion which in turn manages adrenal secretion of Epinephrine and Norepinephrine, the hormones that organize sympathetic nervous system response. Known as the Hypothalamic/Pituitary/Adrenal Axis (HPA), this organ system has feedback loops that promote reparative, parasympathetic nervous system, response as well.

ILF neurofeedback training may be efficacious because it addresses the energy that regulates this organ system.

Recent developments in commercial amplifiers available to neurofeedback practitioners have produced an instrument that is Direct Current (DC) coupled. This seamless integration of the lower (DC) and higher (AC) energies has produced a superior instrument for infra-low training. These DC coupled amplifiers produce enough "bounce" in the low alternating current domain, riding as they do on the DC off-set, to filter and train energies below .1 hertz with a high signal to noise ratio.

This workshop will demonstrate the process of infra-low frequency training on a DC amplifier in clinical practice. The process pivots on the determination of an optimum frequency (OF) that is trained for each individual client. In the didactic portion of the workshop the OF determination process will be demonstrated along with a discussion of the equipment and optimal signal processing requirements necessary to accomplish effective training. The value of QEEG in predicting treatment responders, treatment planning, and determining treatment outcomes will be established. Recent equipment and software advancements have allowed for simultaneous 19 channel recording and ILF training. This innovation that has provided a window on the mechanism of bipolar ILF training, will be discussed. Case studies with pre/post-QEEG analysis will be presented.
**References**


Hughes SW, Lorincz ML, Parri HR, Crunelli V (2012) Infra-slow (<0.1 Hz) oscillations in thalamic relay nuclei: basic mechanisms and significance to health and disease states. Progress in Brain Research, 193C: 145-162.


**Goals/Objectives**

Be familiar with the special equipment and signal processing requirements for low frequency training.

Grasp the process of categorizing client presenting problems that determine the appropriate infra-low frequency intervention.

Understand the process of optimum frequency tuning.

Decide whether infra-low frequency training is appropriate for the participants practice.

**Outline**

Definition of terms frequently used in low frequency work. 10 min

Signal processing requirements, montages, amplifier capabilities, and electrode specifications. 20 min
Starting sites and frequencies as determined by presenting problems. 30 min

Demonstration of the process for determining a client’s optimum reward frequency in the first session. 60 min

The use of QEEG analysis to predict treatment responders, aid in treatment planning, and determine client response. 30 min

Case presentation. 30 min

Financial Interest: No conflicts of interest.

*(Lecture)*

Richard Soutar, PhD, New Mind Neurofeedback Center, [drs@newmindcenter.com](mailto:drs@newmindcenter.com)

Credits: 3

Level of Difficulty: Intermediate

**Abstract**

It has been observed by clinicians for over a decade and a half that pre post qEEGs do not often demonstrate a linear and consistent pattern of change toward normative standards. Clients often appear to regress in some areas while improving in others. This process can be explained in part using Alvaro Pascual Leone’s Theories (2005) regarding compensatory aspects of brain functioning that affect factors such as transcollosal inhibitory control. The work of Alstott (2009) with respect to lesion modeling is also supportive of this perspective as well as work on adaptive mechanisms relating to TBI by Turner et al (2011). Compensatory theory also helps to explain many of the features of change observed in summary and trend screens during training. Utilizing this paradigm can greatly assist practitioners in analyzing qEEGs and trend lines as well as selecting best fit protocols and adjusting them dynamically during training. This workshop will develop the ideas of horizontal and vertical integration from the perspective of Brain Rate (Pop-Jordanova, 2005) and Cortical coupling (Schutter et al, 2005) as well as Compensatory mechanisms to account for and explain changes in the brain due to neurofeedback. It will be integrating Sterman’s arousal theory (1996) with Davidson’s (2000) asymmetry theory in a manner that can be directly applied to clinical data analysis

**References**


Nada Pop-Jordanova1, Jordan Pop-Jordanova2 (2005). Spectrum-weighted EEG frequency “Brain-Rate” as a quantitative indicator XXVI/2, 35–42.


**Goals/Objectives**
Analyze trend screens in terms of horizontal and vertical integration of brain networks.

Recognize normative EEG patterns of horizontal and vertical integration.

Use trend screens to determine changes in reinforcement rates.

Use trend screens to evaluate need for changing protocols.

Use trend screens to determine whether metabolic confounds are present.

Help clients understand their progress in terms of trend screens.

Relate symptoms changes to EEG patterns in trend screens.

**Outline**
Hour 1

Compensation

Transcollosal Inhibition

Adaptive Mechanism

Adaptive Networks

Affect Regulation and Symmetry Theory

Horizontal Integration

Hour 2

Brain Rate

Cortical Coupling

Arousal Theory

Sterman's EEG Arousal Theory

Vertical Integration

Hour 3

EEG Trend Distributions: Normal and Abnormal

Component Band Change Parameters

Component Band Scaling
Abstract for Day 1

For professionals who have not had extensive experience in assessment, differential diagnostic work-up, and basic interventions, for Asperger’s (AS) or Autism (AD).

Day 2: For professionals experienced with autistic spectrum disorder (ASD), an emphasis on how QEEG and psychophysiological assessments lead to effective intervention.

Goal: Attendees for both half days of this workshop will become familiar with how symptoms differ between Asperger’s and autism. They will be able to outline, on the basis of functional Neuroanatomy (which includes discussion of Brodmann Areas, neural networks and connections, including vagal inputs to the medulla and brain stem connections to basal ganglia, thalamus, and cortex), why a combination of NFB + BFB + Strategies improves social functioning in addition to resulting in significant improvements in scores on academic, intelligence, and attention measures (Thompson, Thompson, & Reid, 2010). The participants will learn how autistic spectrum disorders (ASDs) have major difficulties in at least three major networks: executive, affect, and the default network. Every patient will have a different “balance” of involvement or difficulties related to these networks. These three networks can be influenced by Neurofeedback at various sites over the central midline structures (CMS). They are also altered by means of biofeedback and, in particular, by Heart Rate Variability (HRV) training that influences the same CMS. The neuroanatomy and connections of the CMSs, and in particular of areas of the anterior cingulate and how they are involved in the networks, is highlighted.

Abstract for Day 2

For professionals experienced with autistic spectrum disorders (ASD), this day has an emphasis on how QEEG and psychophysiological assessments lead to effective intervention. The emphasis is on more complex assessments with QEEG & when required, evoked potentials (overview only of ERP work). Assessment information is combined with knowledge of Brodmann Areas and knowledge of functional and anatomical neural networks to develop hypotheses regarding how the EEG findings correlate with the Asperger’s symptoms and with symptoms of comorbid disorders (inattention and impulsivity, anxiety, obsessions and compulsions). This is demonstrated using case examples. This EEG assessment is combined with psychophysiological stress profile assessment and psychological and/or psychoeducational
testing to develop an individualized treatment approach. This multifaceted assessment leads to an integration of interventions including: NFB, peripheral biofeedback (BFB), HRV training, transcranial direct current stimulation (tDCS), metacognitive strategies, and perhaps other treatments, such as passive infra-red (pIR) feedback. Although EEGs from several cases are shown because they best illustrate individual aspects of the assessment, we will attempt to describe one case in more detail to walk participants through the diagnostic and treatment prescription process. These interventions usually require NFB + HRV training and, more recently, LORETA neurofeedback has been added to the mix for selected clients. Networks may account for the observation that initial training over central midline structures (CMS) at Fz or Cz, may have effects on broad functional networks (affect, attention, executive, salience, and default networks). Central Midline Structures (CMS) and basic inhibitory linkages that depend on the cortical-basal ganglion-thalamic-cortical links will be reviewed to help the participants understand why and how neural networks are involved in the symptom patterns of these disorders (Thompson, 2011). It follows that interventions are focused on improving a combination of symptoms including: anxiety, social difficulties and executive functioning. Participants will see commonly observed EEG and QEEG patterns including a very common presentation of excess frontal slow wave activity, a dip at Pz in the low alpha (8-10Hz) range, and higher than expected beta activity. Correlation of findings to symptoms and networks is made and exceptions are noted. The QEEG findings are the basis for setting NFB parameters for training and common initial settings will be described. This is complemented by a discussion of the functional neuroanatomical basis for doing BFB, particularly heart rate variability (HRV) training, along with NFB. A rationale will be provided regarding why coaching in metacognitive strategies related to both cognitive and social skills can provide added value. The training addresses the symptoms that interfere with a child patient being able to interact constructively with caregivers including, in order: anxiety, impulsivity, attention span, executive functions, and finally, understanding and responding to social cues. Evidence of producing changes in these areas is provided with statistical analysis of changes in pre-post measures. The p results for NFB, done over CMSs and combined with BFB + Metacognitive strategies, includes data showing changes in EEG ratios TOVA and IVA continuous performance tests, Wechsler Intelligence Scale (WISC & WAIS) scores, academic measures (WRAT3 and WRAT4), and questionnaires for 150 patients with Asperger’s and 9 with Autism (Thompson & Thompson, 2010).

In summary, highlights of Day 2, include hypotheses about EEG correlates of the symptoms found in people with ASD, such as EEG markers for attentional problems, anxiety, and sensory and motor aprosodia. Neuroanatomical differences as compared to a normative database, especially as observed using LORETA, will be discussed and examples shown. Participants will be able to outline, on the basis of functional neuroanatomy why a combination of NF + BF + Strategies improves social functioning in addition to resulting in significant improvements in scores on academic, intelligence, and attention measures (Thompson, Thompson, & Reid, 2010). The neuroanatomical underpinnings include discussion of Brodmann Areas, neural networks (as described in publications relating to work with NFB by de Ridder and the Thompsons) and connections, including vagal inputs to the medulla, and brain stem connections to basal ganglia, thalamus, and cortex. The participants will learn how those with autistic spectrum disorders (ASDs) have major difficulties in at least three major networks: the executive, affect, and default networks. (These three networks will be described, as will the proposed mechanisms by which these networks can be influenced by Neurofeedback at various sites over the central midline structures (CMS). They are also altered by means of biofeedback and, in particular, by Heart Rate Variability (HRV) training that influences the same CMS. The neuroanatomy and connections of the CMS, in particular, areas of the anterior cingulate, and how they are involved in the networks, is highlighted. There will be research support cited, such as Porges’ polyvagal theory and how it supports the HRV intervention, which influences vagal functioning.

Hand-outs that show all power points and references will be provided to participants.

References


**Goals/Objectives**

List the primary symptoms observed in Asperger’s Syndrome

List differences between Autism and Asperger’s

Relate these symptoms to the Neural Networks that are most likely to be involved.

List reasons why initial NFB training over central midline structures, based on QEEG and LORETA assessment findings, is likely to produce improvement in some of the core symptoms of people with Asperger’s.

State why up training of high frequency alpha may be contraindicated in some patients.

State why up training of 14 Hz may be contraindicated in some patients.
Outline, with reference to: affect, executive, and distress networks, and the hypothalamic-pituitary-adrenal axis (HPA), why HRV training may have a positive influence on the outcomes of patients with Asperger’s

Outline
1st hr – Discussion of symptoms of Asperger’s and Autism.

2nd hr – Describe the assessment including QEEG and Psychophysiological Assessment peripheral biofeedback variables.

3rd hr – Outline basic interventions to decrease symptoms of: Anxiety, inattention, impulsivity, and begin on appropriate social interactions.

Financial Interest: Lynda Thompson is co-author of THE A.D.D. BOOK. Michael and Lynda are co-authors of SETTING UP FOR CLINICAL SUCCESS. Michael and Lynda Thompson are co-authors of THE NEUROFEEDBACK BOOK. It is likely that these books may be on sale at the meeting. The authors will state their interest in these books at the workshop.

WS 13: ADHD and Learning Disabilities: Integrating clinical knowledge and individual symptoms and neurophysiology in the formation of Neurofeedback Treatment Plans

(Lecture, Demonstration)
Robert Coben, PhD, Private Practice, drcoben@gmail.com
Anne Stevens, Private Practice, annestevensphd@sbcglobal.net

Credits: 3
Level of Difficulty: Intermediate

Abstract
Current estimates suggest that anywhere from 3 – 9% of school-aged children have been diagnosed with some form of Attention Deficit Hyperactivity Disorder (ADHD). In addition, about 10% of the childhood population has been diagnosed with some type of Learning Disability (LD). Taken together, these major neurodevelopmental disorders impacts approximately one out of every five children (Pastor & Reuben, 2008). The economic costs to society based on these figures has been estimated at at least $42 billion per annum (Pelham, Foster, & Robb, 2007). It has been estimated that about 66% of the children diagnosed with ADHD are treated with medication (CDC, 2005). Considering there are no medications FDA approved for the treatment of learning disabilities, this would suggest that of the 20% impacted by ADHD and LD 13.33 % are treated non-pharmacologically. Neurofeedback has shown promise in the treatment of these neurodevelopmental disorders (Arns, de Ridder, Strehl, Breteler, & Coenen, 2009) as they are neurophysiological in nature.

This workshop will focusing on enhancing knowledge about major neurodevelopmental disorders, treatment options, symptom constellations, and neurophysiological mechanisms so participants will be able to start integrating such information in the formation of neurofeedback protocols and treatment plans. The various forms of ADHD and Learning Disabilities will be discussed. This will include a focus on the following:

1. Review of symptoms, diagnoses, epidemiology, potential causes and neurophysiological findings related to major neurodevelopmental disorders commonly seen in clinical practices. This will include a focus on the various forms of ADHD and Learning Disabilities.

9. Available treatments and their current empirical support.
10. Using Neurofeedback to treat ADHD and LD: Integrating knowledge of these neurodevelopmental disorders with individual symptoms and neurophysiological findings.

11. Discussion and questions.

The true value of this workshop is an understanding from a neuroscientific level what leads to symptoms of these common disorders and how to remedy them. Individualization of treatment is at the heart of this approach, which has led to an overall success rate in approximately 90 – 95% of the cases.

References


Goals/Objectives
Discuss the symptoms and neurophysiological findings associated with major neurodevelopmental disorders.

Review available treatments for these childhood difficulties.

Be able to start to integrate knowledge of symptoms and neurophysiology in the formation of neurofeedback protocols.

Outline
Review of symptoms, diagnoses, epidemiology, potential causes and neurophysiological findings related to major neurodevelopmental disorders commonly seen in clinical practices. This will include a focus on the various forms of ADHD and Learning Disabilities.

Available treatments and their current empirical support.

Using Neurofeedback to treat ADHD and LD: Integrating knowledge of these neurodevelopmental disorders with individual symptoms and neurophysiological findings.

Discussion and questions.

Financial Interest: There is no financial relationship with any software or product discussed in this workshop for either presenter.

**WS 14: Biofeedback and Neurofeedback with Professional and Olympic Athletes**
*(Lecture, Demonstration)*
Michael Linden, PhD, Private Practice, drmike49@aol.com
Penny Werthner, PhD
Credits: 3

Level of Difficulty: Intermediate

Abstract
Interest in the use of Neurofeedback in Sport Psychology is increasing in both professional and Olympic sports. Neurofeedback is usually integrated with biofeedback and other techniques and skills to achieve the greatest application.

QEEG Patterns in athletes with ADD/ADHD and Aspergers will be presented and implications of Neurofeedback protocols with these athletes will be discussed. Biofeedback and Neurofeedback applications will be presented for several Olympic sports (skiing, speed skating, volleyball) and professional sports (tennis, baseball, golf). Demonstration of several general and specific assessment methods and protocols will be provided during this workshop.

References


Goals/Objectives
Understand how to use QEEG to help identify characteristics of ADD and Aspergers in athletes.

Learn Neurofeedback & Biofeedback techniques to improve attention and performance in sports.

Review how other supplementary techniques combine with Neurofeedback & Biofeedback with athletes.

Outline
QEEG patterns in athletes with ADD and Aspergers – Michael Linden -30 minutes

Neurofeedback techniques with athletes – Wes Sime, Michael Linden, Penny Werthner, Sandy Silverman -60 minutes

Biofeedback techniques with athletes – Wes Sime, Penny Werthner, Sandy Silverman, Michael Linden –
Supplementary techniques to combine Neurofeedback with athletes – Wes Sime, Penny Werthner – 30 minutes

Financial Interest: Wes Sime & Michael Linden are editors of a book on Biofeedback & Neurofeedback in Sport Psychology. No conflicts of interest for other presenters.

WS 15: sLORETA and Z Score Neurofeedback: A Clinical Symbiosis (Lecture, Demonstration)
Mark Smith, MSW, Private Practice, marksmith50@verizon.net

Credits: 3
Level of Difficulty: Intermediate

Abstract
This course will demonstrate the use of Z-score/sLORETA training in clinical practice. Stand alone sLORETA training will be shown. Integrative approaches that combine simultaneous sLORETA and Z-score training will be covered. This new training innovation will be discussed as a first order intervention, as well as, for use with non or partial treatment responders. The effective use of QEEG and the Key Institute's LORETA Analysis software, to focus neurofeedback training, will be established. We will demonstrate the integration of recent resting state functional network research as a means to improve clinical decision making. The use of Brainmaster/Avatar Live LORETA Projector will be demonstrated as an effective feedback mechanism. It will also be taught as a real-time analytic tool to aid treatment decisions based on activation patterns and source analysis. Clinical strategies will be taught via a series of case studies from the instructor's private practice. The course practicum will utilize Brainmaster/Avatar training software and Neuroguide's LORETA/Z Score training module. Analytic tools will include Neuroguide QEEG software and the Key Institute’s LORETA analysis software. Thought Technology, Nexus, Deymed, and EEGer users are encouraged to attend as much of the information presented is easily transferable to these platforms.

References


**Goals/Objectives**

Use the LORETA and sLORETA analytic software to choose ROI's for training.

Combine the results of QEEG and LORETA analysis as a means to decide the kind of training, Z Score/sLORETA/traditional inhibit-enhance training, that would be most effective for the client.

Utilize recent functional network research to develop neurofeedback treatment strategies.

**Outline**

How does QEEG analysis, recent neuroscience research, current neurofeedback research, LORETA analysis, and client presentations lead to treatment strategies. 60 min

Explanation and demonstration of sLORETA/Z Score intervention strategies and software applications. 60 min

Demonstration of live sLORETA and sLORETA/Z score training with one or more workshop participants. 60 min

**Financial Interest:** No conflicts of interest.

**WS 16: HRV Biofeedback Training Strategies (Lecture, Demonstration)**

Fredric Shaffer, PhD, Truman State University, fredriesshaffer@gmail.com

**Credits:** 3

**Level of Difficulty:** Intermediate
Abstract
This 3 contact-hour workshop is designed for biofeedback/neurofeedback practitioners, psychologists, clinical counselors, clinical social workers, marriage and family therapists, nurses, physicians, and other health care professionals and academicians interested in utilizing heart rate variability (HRV) biofeedback in their practice or research. This workshop will examine HRV time domain and frequency domain measurements, discuss how to monitor HRV and respiration, demonstrate how to correct breathing mechanics, explain how to design an HRV biofeedback training session, and review the clinical efficacy of HRV biofeedback.

Topics

HRV time domain and frequency domain measurements (30 min).

HRV definition

Time domain measures of HRV (HR Max – HR Min, pNN50, RMSSD, SDNN, and SDRR)

Frequency domain measures of HRV (VLF, LF, HF, LF/HF)

How to monitor HRV and respiration (30 min).

ECG method of monitoring HRV

Sensor placement

Identifying and controlling major artifacts

ECG tracking test

BVP method of monitoring HRV

Sensor placement

Identifying and controlling major artifacts

BVP tracking test

Respirometer method of monitoring breathing A. Measurement of respiration rate and depth B. Warning signs of breathing effort

How to correct breathing mechanics (60 minutes).

Breathing basics

Posture

Clothing

Dysfunctional breathing

Hyperventilation
Thoracic breathing
Clavicular breathing
Reverse breathing
Apnea
Effortless breathing
Explanation of effortless breathing
Effortless breathing training protocols
Computer and smartphone breathing programs
How to design an HRV biofeedback training session (30 min).
Resonance frequency measurement
Lehrer and Gevirtz resonance frequency protocol
2-week test-retest reliability data
HRV Training Protocols
Effective HRV biofeedback displays
How to structure an HRV biofeedback session
The HRV biofeedback learning curve
Important training elements
The effects of drugs on HRV
Innovative resources for home practice
The clinical efficacy of HRV biofeedback (30 min)
Probably efficacious applications: asthma
Possibly efficacious applications: heart disease, heart failure, hypertension, COPD, fibromyalgia, PTSD, and unexplained abdominal pain.

References


biofeedback as a strategy for dealing with competitive anxiety: A case study Biofeedback, 36(3), 109-115.


**Goals/Objectives**

Explain the time domain measures (HR Max – HR Min, pNN50, RMSSD, and SDNN) and the frequency domain measures (VLF, LF, HF, LF/HF) of heart rate variability.

Describe the blood volume pulse (BVP) and electrocardiogram (ECG) methods for monitoring heart rate variability, to identify common sensor placements, to describe tracking tests, and to recognize common artifacts.

Describe the respirometer method for monitoring breathing, and to recognize signs of excessive breathing effort.

Identify dysfunctional breathing mechanics and explain strategies for correcting them.

Describe how to design an HRV biofeedback training session.

Evaluate the clinical efficacy of HRV biofeedback.

**Outline**

HRV time domain and frequency domain measurements (30 min).

How to monitor HRV and respiration (30 min).

How to correct breathing mechanics (60 minutes).

How to design an HRV biofeedback training session (30 min).

The clinical efficacy of HRV biofeedback (30 min).
Financial Interest: No significant financial interests.

WS 17: Validating Emotionally Charged Ipsative Assessments Using Prefrontal EEG Gamma Asymmetry (Lecture)
Ronald Bonnstetter, University of Nebraska, rjb@unl.edu
Thomas Collura, PhD, BrainMaster Technologies, Inc., tomcl@brainm.com

Credits: 3

Level of Difficulty: Intermediate

Abstract
The process of self reported forced-rankings by an individual, as a description of behaviors and beliefs, is a standard approach for many psychological assessments. While these self-perception tools are commonly used and in many cases possess abundant statistical validation, including internal validity, correlation data and means comparisons, until now virtually no research exist that links these self-reports to actual gamma brain activity.

The GIVE process uses asymmetric gamma wave bursts in the prefrontal cortex to validate the underlying subconscious decisions behind these self reported responses, at the very moment of decision-making. This report provides evidence that an evoked emotionally laden response results in corresponding brain activity that documents both the intensity of human emotional response as well as the directionality of the response. While a great deal of research has been done to show how prefrontal cortex baseline alpha wave activity is altered by an evoked stimuli the real source of these subconscious decisions are based on short bursts of gamma activity. The GIVE process not only provides the intensity of a person’s emotional responds to a stimuli by measuring voxel activation, but also provides emotional directionality by differentiating approach/withdrawal responses. The workshop will present the theoretical and experimental foundations, experimental design, results, and application data relating to this new technique. The presentation will be relevant to researchers, educators, clinicians, and others interested in emotional responses and the brain, and emerging technology.

References


Goals/Objectives
Articulate the principles of emotion responses in the brain based on published research Interpret live brain activity recordings showing emotional responses in the brain

Describe experiments using live emotional cues and simultaneous brain recordings showing emotional responses in the brain.

Outline
Brain emotional responses and the published literature (1 Hr)

Design of experiment showing emotional responses in the brain (1 Hr)

Experimental results showing live changes in brain activity during emotional responses (1 Hr)

Financial Interest: Dr. Collura has a financial interest in BrainMaster Technologies Inc. Part of the workshop will describe products provided by BrainMaster Technologies, along with other providers. Dr. Bonnstetter is a Senior Vice President of Target Training International.
ISNR 2012 Conference Vendor Seminars

**VS 1: BrainMaster Basic and Advanced Atlantis, Discovery, and BrainAvatar Workshop**

Tom Collura, PhD, BrainMaster Technologies, tomc1@brainm.com
Bill Mrklas, BrainMaster Technologies
Penijean Rutter, LMHC, Stress Therapy Solutions

This vendor seminar will review the basic functions of the BrainMaster hardware and software, and quickly advance to the new features and functions in BrainAvatar. Live Z-Score (LZT) training using our exclusive multivariate proportional percent z-ok (MVP/PZOK) will be covered, as one of the foundations of our innovative neurofeedback capabilities. The Atlantis and Discovery systems provide from 1 to 24 channels of EEG biofeedback plus peripheral biofeedback. Ease of use, customizability, flexibility, and new functionality are the important features of our new software. Multiple tabbed screens, easy to use desktop shortcuts, new 3-D and contour displays, and the exclusive LLP Live sLORETA Projector are the important new capabilities of this new software. With a response time of 30 milliseconds and a resolution of 6,239 voxels, the LLP provides the unprecedented ability to image and feedback real-time brain activity. More recently, the optional Kaiser Brodmann sLORETA imaging and training has been added, plus access to the SKIL normative database. The use of LLP event-related potentials for client response analysis and interpretation will also be covered. Peripheral biofeedback using skin conductance, heart-rate, EMG, respiration, temperature, and HEG will be included. Our new live TOVA (Test of Variables of Attention) continuous performance task interface will also be described and demonstrated.

**VS 2: The LENS Vendor Workshop**

Len Ochs, PhD, Ochs Labs, lochs@earthlink.net

This Vendor Workshop gives an overview of the LENS,

- how it is different from traditional neurofeedback,
- why it has an overall average of 20 sessions treatment duration,
- why it can have a 6-session average treatment duration for mild traumatic head injury,
- what makes treatment have an either shorter or longer duration of treatment.

Also covered are the concepts underlying it, the practical elements of the LENS.

It is also the goal of this presentation to clear up misinformation and incomplete information about the LENS as a neurofeedback system.

Ample time will be devoted to answering questions.

**VS 3: Zukor’s Grind & Next-Generation Feedback Games**

Samuel Turcotte, President and Chief Technical Officer of Zukor Interactive, turcotte@zukor.com

Allen Novian, PhD, Chief Clinical Advisor to Zukor Interactive

**Overview**

This workshop will demonstrate and explain the next-generation feedback game features
of Zukor’s Grind for clinical neurofeedback and biofeedback. This game, the first of a series from Zukor Interactive, has dozens of features, from basic to advanced, never before seen in the industry.

**Live Demos**

After the presentation, live demos of the game will be conducted on four major neurofeedback systems: BrainMaster, EEGer, Mind Media and Thought Technology.

**VS 4: The Many Applications of the Thought Technology Infiniti Product Line**

Thought Technology, Ltd.

Marc Saab, M.Eng, marc@thoughttechnology.com
Didier Combalalade, D.C., research@thoughttechnology.com
Jane Arave, janearave@msn.ca

**Abstract**

Attend our vendor seminar and learn about the use of Thought Technology Ltd. Products in various clinical environments. This year’s special guest is Jane Arave, MA, LPC, BCB, BCN. Jane is a Licensed Professional Counselor and is BCIA certified in both Biofeedback (BF) and Neurofeedback (NF). She has been in private practice at Northeast Counseling & Learning Center in Columbia, SC since 2002. In 2006, she designed, developed and implemented a 12-session optimum performance program for golfers. “The Mental Game of Golf” is a combination of cognitive strategies, mindfulness exercises, breathing techniques, and psychophysiological BF and NF protocols. Jane began working with the Biofeedback Foundation of Europe (BFE) in 2008. As a software developer and on-line instructor for the BFE, she worked closely with Dr. Paul Swingle to develop the QuickQ Suite for the ProComp Infiniti. Her most recent accomplishment for the BFE was the development of the ProGolf Suite using her golf program as her main source of reference. Jane contributed a golf case study chapter for *Case Studies in Applied Psychophysiology* (W. Alex Edmonds and Gershon Tenenbaum, editors) which was published in 2012. In addition, to maintaining her private practice, Jane is currently contributing to research at the William Jennings Bryan Dorn VA Medical Center in Columbia, studying the use of Heart Rate Variability (HRV) training with patients diagnosed with Post Traumatic Stress Disorder (PTSD).

Jane will be presenting the specialized BFE software applications she helped develop, namely Dr. Swingle’s QuickQ and BrainDryver Suite and the ProGolf Suite.

For more information on these and other suites available from the BFE, visit [http://www.bfe.org/buy/advanced_search_result.php?keywords=francois+dupont&x=48&y=8](http://www.bfe.org/buy/advanced_search_result.php?keywords=francois+dupont&x=48&y=8). Register for Thought Technology’s vendor seminar for a chance to win one of these BFE suites!

In addition, come and see why BioGraph Infiniti is the best and most widely used bio and neurofeedback platform in the world. See a sneak preview of Biograph Infiniti v 6.0 and receive a chance to win a free upgrade! With features like true real-time artifacting, enhanced zscore biofeedback features and amazing new event marking tools, BioGraph Infiniti v 6.0 is clearly in a league of its own!