<table>
<thead>
<tr>
<th>Time</th>
<th>Monday September 12, 2011 Preconference Workshop Schedule</th>
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<tr>
<td>10:00 AM-1:00 PM &amp;</td>
<td>Pre-Conference Workshop 1.1 (Day 1) LENS Foundation Training (3 day workshop) – Catherine Wills</td>
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<td>Pre-Conference Workshop 2.1 (Day 1) Advanced LENS Training (3 day workshop) – Len Ochs</td>
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<tr>
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<td>Pre-Conference Workshop 1.2 (Day 2) LENS Foundation Training (3 day workshop) – Catherine Wills</td>
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<td>Pre-Conference Workshop 3.1 (Day 1) Introduction to the Practice of Neurofeedback: Assessment leads to Appropriate Intervention (2 day workshop) – Michael Thompson &amp; Lynda Thompson</td>
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<tr>
<td>10:00 AM-1:00 PM &amp;</td>
<td>Pre-Conference Workshop 1.3 (Day 3) LENS Foundation Training (3 day workshop) – Catherine Wills</td>
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<tr>
<td>8:00 AM-12:15 PM &amp;</td>
<td>Pre-Conference Workshop 4 - Advanced Live Z-Score and Combined Protocols for Comprehensive Neurofeedback - Tom Collura,</td>
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<td>1:15-5:30 PM</td>
<td>Mark Smith &amp; Penijean Rutter</td>
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<tr>
<td>8:00 AM-12:15 PM &amp;</td>
<td>Pre-Conference Workshop 5 - Pain, Chronic Pain and the Nervous System - Stuart Donaldson</td>
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<tr>
<td>8:00 AM-12:15 PM &amp;</td>
<td>Pre-Conference Workshop 6 - Clinical Significance of Paroxysmal EEG Discharges: Location Links Pathology - Jay Gunkelman</td>
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<tr>
<td>1:15-5:30 PM</td>
<td>Ron Swatzyna</td>
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<tr>
<td>8:00 AM-12:15 PM &amp;</td>
<td>Pre-Conference Workshop 7 - Applied Neuroscience in Clinical Practice: What Can Event Related Potentials Add for Diagnostic and</td>
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<td>1:15-5:30 PM</td>
<td>Treatment Procedures? - Juri Kropotov</td>
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<tr>
<td>8:00 AM-12:15 PM &amp;</td>
<td>Pre-Conference Workshop 8 - Physiology, Clinical Outcomes, and Most Current Research on Audio-Visual Entrainment, Cranio-</td>
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<tr>
<td>1:15-5:30 PM</td>
<td>Electrical Stimulation, and Transcranial DC Stimulation - David Siever</td>
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Monday, September 12, 2011

Pre WS 1.1: LENS Foundations Training (Day 1 of 3)
(Lecture, Experiential, Demonstration)
Cathy Wills, CNS, MSN, Ochs Labs, cathywills@ochslabs.com

Credits: CME – 6.5, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences CE – 6.5, BCIA recertification – 6.5

Level of Difficulty: Basic

Abstract
This is a three day training in the LENS covering conceptual and practical elements that will allow the participant to:
1. Operate the software,
2. Make entry level clinical treatment decisions based on assessments and interview,
3. Evaluate the success of meeting treatment objectives,
4. Understand the elements of informed consent, communication with clients, and recommended and not-recommended early-experience clients.

Updated definitions of Perceptual Sensitivity, Reactivity, Behavioral Suppression, Hardiness, and Anxiety
Assessing Perceptual Sensitivity, Reactivity, Behavioral Suppression, Hardiness, and Anxiety
Relation between Traits and Dose considerations
Relation between Traits and Informed Consent considerations: anticipating the unexpected
Relation between Traits and Conversation with Client re what to expect during the LENS
LENS Topographic Mapping

The 10-20 International Classification of Sensor Sites and connect electrodes to the scalp

References
Learning Objective
List purposes of the LENS Evaluation.
Describe Sensitivity vs. Insensitivity.
Describe Hardiness vs. Fragility.
Describe Reactivity vs. Stolidness.
Describe Behavioral Suppression.
List the purpose of assessing the above four qualities.
Demonstrate the use of the Sensitivity Questionnaire in assessing the above qualities.

Outline
List purposes of the LENS Evaluation, including the CNS questionnaire (180 minutes)
Describe elements of the LENS Concepts, and their purposes (240 minutes)
Discussion (30 minutes)

Financial Interest: I am the President of OchsLabs, Inc.

Pre WS 2.1: Advanced LENS Training (Day 1 of 3)
(Lecture, Experiential)
Len Ochs, PhD, Ochs Labs, lochs@earthlink.net

Credits: CME – 6.5, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences CE – 6.5, BCIA recertification – 6.5

Level of Difficulty: Advanced

Abstract
This is an advanced integrated training updating past knowledge including new information on:
1. New information on traits such as perceptual sensitivity, reactivity, hardiness, behavioral suppression and anxiety,
2. Updates on the use of suppression maps
3. The New LENSWare 2 Interface and how it configures components of complex applications
4. New understanding of the LENS signals: hum, baseline, and feedback
5. Integration of standard, suppression, and other kinds of maps
6. New applications such as seizure management and wound applications

Marcus, L. (2001). EEG Amplitude and Variability Changes Following Low-Intensity Neurofeedback-Based Stimulation for Fibromyalgia. Palo Alto, CA, Western Graduate School of Psychology.
Ochs, L. (1997). EDS: Background and operation, EEG-driven pico-photic stimulation. Walnut Creek, CA, Flexyx, LLC.
To Review the latest in treatment of Traumatic Brain Injury – didactic (1 hour)
To Review topic of EEG Suppression: Diagnostic and Treatment implications (1 hour)
TBI treatment – discussion (1 hour)
Review Protocol Selection and strategies (1 hour)
Review of TBI treatment considerations from the point of view of a large treatment center (1 hour)
Review of concepts, suppression data via topographic mapping of
Coefficient of Variation, and the use of Suppression mapping in treatment. (1 hour)
Case presentations, reports of experience, discussion of evaluation and treatment considerations (1 hour)

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

Learning Objective
Enumerate the difference between the old and new views of EEG suppression.
List alternatives for opening and closing the LENS.
Demonstrate how to change the offset for each period.
Demonstrate how to turn off the feedback and leave hum signals.
Demonstrate how to turn on high efficiency feedback.
Demonstrate how 3 ways to turn the standard applications into complex applications.
Demonstrate how to change the filters from which the dominant frequency is extracted.

Outline
The New Interface, the features and the reasons for using and not using each of the parameters
Main Screen
Customization Screen
Saving Data
Setting Period Parameters
Duration
Feedback on/off

Financial Interest: While I am not an owner, employee, or stock holder of OchsLabs, Inc., I do use them as a conduit for the expression of my ideas. My meeting expenses are paid by OchsLabs, Inc. However, I am supported completely by non-OchsLabs, Inc. retirement funds.

Tuesday, September 13, 2011

Pre WS 1.2: LENS Foundations Training (Day 2 of 3)
(Lecture, Experiential, Demonstration)
Cathy Wills, CNS, MSN, Ochs Labs, cathywills@ochslabs.com

Credits: CME – 6.5, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences CE – 6.5, BCIA recertification – 6.5

Level of Difficulty: Basic

Abstract
This is a three day training in the LENS covering conceptual and practical elements that will allow the participant to:

1. Operate the software,
2. Make entry level clinical treatment decisions based on assessments and interview,
3. Evaluate the success of meeting treatment objectives,
4. Understand the elements of informed consent, communication with clients, and recommended and not-recommended early-experience clients.

Applying electrodes -1 hr
Information about the new LENSware2 user interface -1 hr
Running the user interface for mapping -1 hr
Applying the electrodes for mapping -1 hr
Estimating the offset for sensitive and less sensitive individuals -1 hr
Components of the LENS signals and stimulation vs. feedback paradigms -1 hr
Components of treatment and their relation to the assessment(s) -1 hr

References
Learning Objective
List the LENS evaluation protocols.
List the LENS applications.
List the relationship between the qualities assessed and the applications.
List the components of the applications: Hum, Baseline, Feedback.
List how the Hum, Baseline, and Feedback appear in each of the LENS applications.
Describe how Hum and Baseline appear in all standard neurofeedback applications, and why
Demonstrate Opening, Closing, Saving Data, and modifying the LENS applications.

Outline
Demonstrate the use of the Sensitivity Questionnaire in assessing the above qualities (180 minutes)
Demonstrate the use of the LENS Evaluation and Treatment protocols (240 minutes)
Describe the relationship between the qualities assessed and the applications (60 minutes)
Discussion (30 minutes)

Financial Interest: I am the president of OchsLabs, Inc.

Pre WS 2.2: Advanced LENS Training (Day 2 of 3)
(Lecture, Experiential)
Len Ochs, PhD, Ochs Labs, lochs@earthlink.net

Credits: CME – 6.5, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences CE – 6.5, BCIA recertification – 6.5

Level of Difficulty: Advanced

Abstract
This is an advanced integrated training updating past knowledge including new information on:
1. New information on traits such as perceptual sensitivity, reactivity, hardiness, behavioral Suppression and anxiety,
2. Updates on the use of suppression maps
3. The New LENSWare 2 Interface and how it configures components of complex applications
4. New understanding of the LENS signals: hum, baseline, and feedback
5. Integration of standard, suppression, and other kinds of maps
6. New applications such as seizure management and wound applications

Participants demonstrate placebo and nocebo issues related to the use of the LENS (1 hour)
Participants demonstrate interaction of glucose response curve and its implications for both the physiology underlying the EEG and aspects of functioning, as well as the implications for the use of the LENS with the consequences of aberrant insulin responses. (1 hour)
Participants demonstrate a grasp of different treatment options paired with different clinical pictures. (1 hour)
Review of the standard and advanced applications, including the extra-long applications, their components, and the effects of each of the components across our treatment spectrum (1 hour)
Participants are able to enumerate a range of clinical pictures associated with TBI, and discuss a range of treatment considerations for different clinical pictures (1 hour)
Participants demonstrate a grasp of different treatment options paired with different clinical pictures (1 hour)

References
Learning Objective
Demonstrate the site sort sequence for working with EEG suppression
Describe how frequency suppression is converted into amplitude suppression.
Describe how amplitude suppression is converted into frequency activity with varying degrees of variability
Describe the composition of Standard, 100% Duty cycle, Narrow Band applications
Classify elements of the LENS applications
Describe the purposes of Sensitivity, reactivity, Hardiness, and Behavioral Suppression in terms of their utility for conducting LENS sessions.

Outline
Broad/Narrow Band
Offset
Duty Cycle
Changes among the parameters
Band Filters
Number of sites

Financial Interest: While I am not an owner, employee, or stock holder of OchsLabs, Inc., I do use them as a conduit for the expression of my ideas. My meeting expenses are paid by OchsLabs, Inc. However, I am supported completely by non-OchsLabs, Inc. retirement funds.

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: CME – 8, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences CE – 8, BCIA recertification – 8

Level of Difficulty: Basic

Abstract
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 Brodman 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 centre 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
a. Knowledge: Understand the fundamental principles that underlie everyday work with clients and be able to define and discuss:
   i. Neurofeedback terminology including: 10-20 system, origin of the EEG, 5 characteristics that define every EEG waveform, types of waveforms, correspondence of bandwidth frequencies to mental states, LORETA, z-scores, Brodman areas, coherence, and very basic functional neuroanatomy.
   ii. How learning theory underlies every training session and be able to define key terms such as: operant and classical conditioning, shaping, generalization, etc.
   iii. Basic psychophysiological measures including heart rate variability (HRV), heart rate, respiration, electrodermal responses, peripheral skin temperature, electromyogram and, very briefly, an explanation of how NFB combines synergistically with BFB to optimize a client’s performance.
   iv. Other terms including: differential amplifier, impedance, optical isolation.

b. Assessment:
   i. Briefly discuss the common EEG, LORETA, and/or psychophysiological findings in ADHD.
   ii. Briefly explain the information provided by single channel EEG assessment and be able to state when this may be sufficient, and when a 19-channel EEG assessment should be carried out.
Outline
Participants will receive an overview of learning theory relevant to neurofeedback, the physiological basis of the EEG, how instruments measure the EEG, normal and abnormal wave forms, and the functional significance of parts of the brain that underlie neurofeedback interventions. Discussion of 19 channel QEEG and LORETA assessment findings will be touched upon. For each disorder, as they are used for demonstration, the functional neuroanatomy and research findings in imaging studies will be reviewed. Participants will also learn the fundamentals which underlie other biofeedback modalities which include skin conduction (EDR), peripheral temperature, respiration, heart rate (RSA) and EMG. (45 minutes)

Content will include electrode placement, impedance, recognizing and handling artifacts, and observing and distinguishing characteristic power patterns in the frequency range 2 to 61 Hz which may be observed in a number of disorders. Participants will be shown typical patterns for short attention span, impulsivity, learning disabilities, movement disorders (Parkinson’s, dystonia), Asperger’s syndrome, seizure disorders, anxiety, dysphoria with ruminations. Live demonstration of how to do the assessment will be carried out. To supplement the EEG diagnostic criteria, we will also give a description of ADHD as it is the most common condition dealt with using NFB. We will highlight the EEG differences between 2 distinct groups of adults and compare these EEG finding with those seen in children. We will include a list of the symptoms of ADD. Assessment procedures will be briefly reviewed (history, symptoms, computerized tests, QEEG, psychometric testing - intellectual and academic tests, questionnaires for children and adults and research evidence (PET, SPECT, EEG) will be discussed. A number of typical EEG patterns will be described and illustrated. (2 hours)

The components of a psychophysiological stress assessment will be listed and discussed. The measurements will include: skin conduction (EDR), peripheral skin temperature, respiration, heart rate, respiratory sinus arrhythmia (RSA) and electromyogram (EMG). These measurements will be evaluated and discussed in the context of likely correlated EEG findings. Typical findings in anxiety and stress will be reviewed with examples (e.g., executives, children with high anxiety and children with Asperger’s syndrome and others). A live demonstration of a stress assessment will be carried out and discussed (90 minutes)

A model for decision making for NFB training/treatment will be presented and discussed. Participants will be instructed in the appropriate placement of electrodes and the frequency bands to be enhanced or inhibited for each channel for the various conditions discussed in the assessment. In each case the training procedures are based on the EEG findings. The rationale behind the use of different kinds of display screens for different training/therapeutic purposes will be discussed. The presenters use instruments from 7 different manufacturers thus, the EEG discussion is not specific to any one system. QEEG assessments (usually 3 lead referential and/or bipolar but full cap is available as required) are combined with assessment of the autonomic nervous system as is appropriate for the client. The 10-20 electrode placement system will be reviewed and montages discussed in conjunction with specific protocols. In a full day workshop participants are able to participate in hands-on experience. (75 minutes)

With most clients a relaxed yet alert mental state is desirable. How to base BFB training on the stress assessment will be discussed and demonstrated. In ADD alertness when carrying out relatively routine or boring tasks is a primary issue. It is best addressed using NFB combined with EDR. This will be addressed and demonstrated. The neurophysiology underlying heart rate variability training and the importance of combining this BFB with NFB will be explained from a function Neuroanatomical systems viewpoint (75 minutes)

A balanced approach to training requires that most training sessions address the mental state of the client in a holistic manner by combining the appropriate mixture of NFB, BFB and strategies. This is as true for dealing with A.D.D as it is for handling the highly stressed executive or the professional athlete. How to include BFB in NFB training sessions will be discussed and demonstrated. Participants will learn ways to use both operant and classical conditioning by combining training with adjunctive techniques (such as metacognition) to enhance and generalize the learning from the NFB sessions. (75 minutes)

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 during the workshop.

Wednesday, September 14, 2011

Pre WS 1.3: LENS Foundations Training (Day 3 of 3)
(Lecture, Experiential, Demonstration)
Cathy Wills, CNS, MSN, Ochs Labs, cathywills@ochslabs.com

Credits: CME – 6.5, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences CE – 6.5, BCIA recertification – 6.5

Level of Difficulty: Basic
Abstract
This is a three day training in the LENS covering conceptual and practical elements that will allow the participant to:

1. Operate the software,
2. Make entry level clinical treatment decisions based on assessments and interview,
3. Evaluate the success of meeting treatment objectives,
4. Understand the elements of informed consent, communication with clients, and recommended and not-recommended early-experience clients.

LENS Treatment - 1 hr
The experiences of LENS assessment and treatment - 1 hr
Providing context and perspective to the experience of change using the LENS - 1 hr
The future course of learning - 1 hr
Special cases, which cases are appropriate and which are inappropriate to accept - 1 hr
The courses of healing and what to observe about the change processes - 1 hr
Test and workshop evaluation - 1 hr

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

Learning Objective
Demonstrate knowledge of the landmarks locating each of the 10-20 sites.
Demonstrate running a map.
Demonstrate doing an initial interview.
Demonstrate constructing an initial treatment plan based on the map and offset evaluation.
Describe how initial LENS evaluations and treatments are chosen on the basis of the initial evaluations.
Describe what problems may be accepted for use with the LENS during the first two years of use.
Describe criteria for making a referral and refusing to continue with the LENS.

Outline
Describe the relationship between the qualities assessed and the applications (60 minutes)
Define and contrast the components of the applications: Hum, Baseline, and Feedback (60 minutes)
Demonstrate providing Maps and Offset Evaluations (300 minutes)
Discussion (30 minutes)

Financial Interest: I am the president of OchsLabs, Inc.

Pre WS 2.3: Advanced LENS Training (Day 3 of 3)
(Lecture, Experiential)
Len Ochs, PhD, Ochs Labs, lochs@earthlink.net

Credits: CME – 6.5, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences CE – 6.5, BCIA recertification – 6.5

Level of Difficulty: Advanced

Abstract
This is an advanced integrated training updating past knowledge including new information on:
1. New information on traits such as perceptual sensitivity, reactivity, hardness, behavioral Suppression and anxiety,
2. Updates on the use of suppression maps
3. The New LENSWare 2 Interface and how it configures components of complex applications
4. New understanding of the LENS signals: hum, baseline, and feedback
5. Integration of standard, suppression, and other kinds of maps
6. New applications such as seizure management and wound applications

Participants demonstrate an understanding of electrophysiological characteristics of ADD/ADHD, changes in the EEG, and how the LENS treatment can be enhanced just LENS treatment, and by concurrent cranial-sacral work by reviewing a current study. (1 hour)
Participants demonstrate an understanding of electrophysiological characteristics of anxiety, changes in the EEG, and how the LENS treatment is used with Anxiety. (1 hour)
Participants review research methodology. (1 hour)
To provide multiple views of anxiety evaluation, treatment and re-evaluation with the LENS (1 hour)
Review and Discuss components of the LENS signals, applications, and paradigms (1 hour)
Review vascular physiology and infrared interventions (1 hour)
Complete Test, Workshop evaluation, and comments (1 hour)

References
Washington, DC, Bureau of Education for the Handicapped.


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


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


Learning Objective
List why the LENS involves more than just application selection.
Describe four qualities that lead to ceasing working with the LENS and making a referral out.
Define the meanings of EEG drift, and sharpening.
Give an alternative to finding a sweet spot for working with the LENS.
Demonstrate clocking the duration of reactions and why they are useful.
Define EEG hydraulics, and state why imagining liquid flow applies to problems.
Describe three ways to weaken LENS applications.

Outline
Customizing the Offset Evaluation
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: CME – 8, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences - 8, BCIA recertification – 8

Level of Difficulty: Intermediate

Abstract

First we will answer questions arising from Day 1. 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 with an emphasis 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 participant’s 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 may be able to cover other disorders in more advanced detail in another workshop. Efficacy guidelines will be noted for each of the disorders discussed in the workshop with reference to the joint ISNR/AAPB guidelines.

References


Goals/Objectives

b. Assessment:

iii. Describe appropriate data collection procedures: electrode placement for EEG, impedance, recognizing and handling artifacts, gathering accurate statistics during different conditions (eo, ec, reading, math, drawing).
iv. Recognize characteristic EEG patterns of ADHD including theta/beta power ratios and patterns that are missed by the published theta/beta power ratios (Monastra et al, 1998).
v. Identify basic psychophysiological responses to stress and patterns found during recovery from stress.
vi. Understand in broad general terms how 19 channel assessments are done, including use of normative databases and LORETA. and briefly which diagnostic categories of clients require 19-channel assessments and how they increase the variables that can be addressed.

c. Intervention:
   i. Use assessment data to develop a rationale for intervention using neurofeedback (NFB) and combine it with biofeedback (BFB) and strategies in a responsible manner for ADHD with anxiety.
   ii. List potential side effects of NFB and of BFB.
   iii. 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.

Outline (Expanded Information From Day 1)

Participants will receive an overview of learning theory relevant to neurofeedback, the physiological basis of the EEG, how instruments measure the EEG, normal and abnormal wave forms, and the functional significance of parts of the brain that underlie neurofeedback interventions. Discussion of 19 channel QEEG and LORETA assessment findings will be touched upon. For each disorder, as they are used for demonstration, the functional neuroanatomy and research findings in imaging studies will be reviewed. Participants will also learn the fundamentals which underlie other biofeedback modalities which include skin conduction (EDR), peripheral temperature, respiration, heart rate (RSA) and EMG. (45 minutes)

Content will include electrode placement, impedance, recognizing and handling artifacts, and observing and distinguishing characteristic power patterns in the frequency range 2 to 61 Hz which may be observed in a number of disorders. Participants will be shown typical patterns for short attention span, impulsivity, learning disabilities, movement disorders (Parkinson’s, dystonia), Asperger’s syndrome, seizure disorders, anxiety, dysphoria with ruminations. Live demonstration on how to do the assessment will be carried out. To supplement the EEG diagnostic criteria, we will also give a description of ADHD as it is the most common condition dealt with using NFB. We will highlight the EEG differences between 2 distinct groups of adults and compare these EEG finding with those seen in children. We will include a list of the symptoms of ADD. Assessment procedures will be briefly reviewed (history, symptoms, computerized tests, QEEG, psychometric testing - intellectual and academic tests, questionnaires for children and adults and research evidence (PET, SPECT, EEG) will be discussed. A number of typical EEG patterns will be described and illustrated. (2 hours)

The components of a psychophysiological stress assessment will be listed and discussed. The measurements will include: skin conduction (EDR), peripheral skin temperature, respiration, heart rate, respiratory sinus arrhythmia (RSA) and electromyogram (EMG). These measurements will be evaluated and discussed in the context of likely correlated EEG findings. Typical findings in anxiety and stress will be reviewed with examples (e.g., executives, children with high anxiety and children with Asperger’s syndrome and others). A live demonstration of a stress assessment will be carried out and discussed. (90 minutes)

A model for decision making for NFB training/treatment will be presented and discussed. Participants will be instructed in the appropriate placement of electrodes and the frequency bands to be enhanced or inhibited for each channel for the various conditions discussed in the assessment. In each case the training procedures are based on the EEG findings. The rationale behind the use of different kinds of display screens for different training/therapeutic purposes will be discussed. The presenters use instruments from 7 different manufacturers thus, the EEG discussion is not specific to any one system. QEEG assessments (usually 3 lead referential and/or bipolar but full cap is available as required) are combined with assessment of the autonomic nervous system as is appropriate for the client. The 10-20 electrode placement system will be reviewed and montages discussed in conjunction with specific protocols. In a full day workshop participants are able to participate in hands-on experience. (75 minutes)

With most clients a relaxed yet alert mental state is desirable. How to base BFB training on the stress assessment will be discussed and demonstrated. In ADD alertness when carrying out relatively routine or boring tasks is a primary issue. It is best addressed using NFB combined with EDR. This will be addressed and demonstrated. The neurophysiology underlying heart rate variability training and the importance of combining this BFB with NFB will be explained from a function Neuroanatomical systems viewpoint. (75 minutes)

A balanced approach to training requires that most training sessions address the mental state of the client in a holistic manner by combining the appropriate mixture of NFB, BFB and strategies. This is as true for dealing with A.D.D as it is for handling the highly stressed executive or the professional athlete. How to include BFB in NFB training sessions will be discussed and demonstrated. Participants will learn ways to use both operant and classical conditioning by combining training with adjunctive techniques (such as metacognition) to enhance and generalize the learning from the NFB sessions. (75 minutes)

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 during the workshop.

Pre WS 4: Advanced Live A-Score and Combined Protocols for Comprehensive Neurofeedback
   (Lecture, Demonstration with Case Studies)
Thomas Collura, PhD, BrainMaster Technologies, Inc., tomc1@brainm.com
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Credits: CME – 8, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences– 8, BCIA recertification – 8

Level of Difficulty: Intermediate

Abstract
This workshop will describe and demonstrate advanced techniques combining neurofeedback training with other methods, for comprehensive whole-head as well as body-oriented training. Additional techniques will include live z-scores and conventional neurofeedback (SMR, beta, theta, and alpha protocols), ILF (infra-low frequency) training, DC and Slow-Cortical Potentials, Beta Reset protocols, and the addition of peripheral modalities. The emphasis will be on principles, with specific systems used to illustrate implementation alternatives. Attendees will come away from this workshop with an understanding of how basic and advanced live z-score protocols can be designed on different platforms, and how conventional EEG as well as peripheral biofeedback protocols can be added with additional auditory and/or visual feedback. Continuity with proven methods will be emphasized, in a comprehensive approach. Despite their apparent complexity, live z-scores provide simple feedback, enabling demonstrable changes in both EEG and in cognitive functioning. Live z-scores can also be used as an “envelope” to guide EEG changes, when other modalities are in use. Limitations of this approach will be discussed, explaining the need for proper clinical and EEG assessment and the purposeful design of protocols. Protocols must be developed with an awareness of normal and abnormal brain processes, and the expected effects of brain normalization. While z-scores can provide a “GPS” for the brain, clinicians must still understand “where” they are guiding the brain, and “why.” Attendees are encouraged to bring their neurofeedback equipment, and take part in the practicum demonstration. Clinical Case studies will also be presented, demonstrating within-session and across-session changes in QEEG and related measures, along with clinical history and outcome data.

References

Goals/Objectives
Design and execute complex neurofeedback protocols including live z scores
Conduct neurofeedback using infra-low frequency protocols
Review QEEG data and use it to develop neurofeedback protocol plans
Interpret clinical outcome data resulting from QEEG-based neurofeedback training

Outline
Basics of neurofeedback and brain self-regulation (1 hour)
Basics of setting up neurofeedback protocols (1 hour)
Fundamentals of Live Z-Score training (1 hour)
Combining Live Z-Scores with conventional protocols (1 hour)
Infra-slow training protocols (1 hour)
DC and Slow Cortical Potential neurofeedback training (1 hour)
Combining EEG and peripheral biofeedback (1 hour)
Practicum and demonstration of QEEG and neurofeedback using live Z-Scores (1 hour)
Pre WS 5: Pain, Chronic Pain and the Nervous System
(Lecture, Demonstration)
Stuart Donaldson, PhD., Myosymmetries, myo@telus.net

Credits: CME – 8, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences– 8, BCIA recertification – 8

Level of Difficulty: Advanced

Abstract
It is estimated that between 10 and 15% of the total population in North America suffers from chronic benign pain. Despite these staggering numbers the success rate in the eradication of chronic benign pain is very low. The introduction of the concept of neuroplasticity offers the biofeedback practitioner a philosophical and practical basis for the assessment and treatment for chronic benign pain.

This workshop will focus on the utilization of numerous biofeedback procedures to document the activity of the three parts of the nervous system [a) central, b) peripheral and c) autonomic] as they are affected or involved with chronic pain. The workshop will start out with a discussion of pain and its relationship to anatomical injuries. This will lead into a discussion of how this affects the medical, legal and insurance industries and how it shapes our thinking as health care providers. NEUROPLASTICITY as a working model will be introduced and discussed not only as it affects the nervous system but all systems in the body.

The second part of the workshop will focus on the use of EEG, SEMG and stress profiling to study the electrical activity of the three parts of the nervous system. This data will then be discussed focusing on physiology and not diagnosis. The development of treatment plans and what to treat when will follow. Finally discussion will proceed to how to be a patient advocate and how to help the patient use the information gathered from our evaluation to enhance and improve their life.

References

Goals/Objectives
1. Understand the problems in defining pain and understand the implications this has for medical, legal and insurance industries. Particular emphasis will be placed upon the relationship of anatomical injury to reported pain and how this relationship (as poor as it is) restricts appropriate treatments.
2. Understand the differences between acute and chronic benign pain focusing upon a) biochemical comodulators and their effect upon the relationship between emotions (such as depression) and chronic pain, b) the differences in neural systems involved, c) the effect of mor-morbid conditions upon chronic benign pain and d) why it is so difficult to diagnose.
3. Understand neuroplasticity & how it impacts the 3 branches of the nervous system leading to neural excitability (allodynia & hyperalgesia). Also discussed will be the impact upon the human body starting at the cellular level. Finally, the ramifications of the introduction of this model has upon the medical model.

4. Understand the differences between anatomy and physiology and why the knowledge of physiology is important to understanding the concept of neuroplasticity.

5. Understand SEMG assessment and what it tells us about the muscles, the nerves, the joints and the kinesiological aspects of neuroplasticity

6. Understand the central nervous system, qEEG assessment and what this tells us about the central nervous system. Included will be a discussion of MTBI, absolute power, coherence and phase and how these impact the activity of the brain as it relates to chronic benign pain

7. Understand the autonomic nervous system & stress testing using Sue Wilson’s protocol. We will then discuss how this data directs the type of treatment conducted.

8. Understand the responsibilities of being a therapist to the chronic benign pain patient making sure the patient understands a) the neurophysiological basis of chronic benign pain, b) the need to reduce the level of excititation and c) give them control and thus hope.

Outline

1. Discuss acute pain tracing its history (starting with Descarte) to the present day complex definitions. We will then discuss how there is only a 20% match between anatomical findings and level of reported pain and the factors that create this poor correlation. What impact this has on the medical, legal and insurance industries and how it influences the thinking of the biofeedback field will be reviewed – 40 min.

2. We will discuss the problems in defining chronic benign pain using current models to illustrate the deficiencies in knowledge of the field. Issues to be examined will include: The nervous system (particularly as it relates to the sensory component; the brain), also to be discussed will be the impact of neuro-comodulators as it relates to depression and pain and PTSD – 30 min

3. Neuroplasticity will be discussed in detail focusing on activity in neuro system particularly in the brain. Also included with be a discussion of its impact upon cells, joints and neurotransmitters. The ramifications of this model upon the current medical model will be explored - 30 min

4. Discussion will focus on the need to shift from an anatomical basis to a physiological basis of the assessment and treatment. A case will be made how as biofeedback practitioners we should be talking about altering (increasing or decreasing) electrical activity and studying the interaction amongst the anatomical components – 30 min

5. SEMG assessment tells us about the interaction between muscles, joints, the peripheral nervous system, and all kinesiological aspects of the system. A live demonstration will illustrate how kinesiological systems become disrupted leading to the development of poor posture and dysponesis. Numerous illustrations of muscle dysfunction involving 2, 4 and 8 channel SEMG protocols will be included -90 min

6. EEG assessment – as part of the lecture we will focus on use of the qEEG documenting the absolute power in the different parts of the brain, coherence, phase, and further discuss trauma of the brain and its effect on neuronal regulation. Numerous illustrations of qEEG patterns as seen in chronic pain patients will be provided. A brief discussion of treatment protocols will conclude this section – 90 min.

7. Stress testing – discuss Sue Wilson’s protocol and what it tells us about the ANS. The use of this data in determining which of the numerous treatment modalities (including HRV, GSR, temp and emwave) will conclude this section – 90 min

8. The presentation will conclude with a discussion on how to develop treatment protocol using all of the above information. Not only will we talk about the use of numerous biofeedback procedures but we will talk about the importance of becoming a patient advocate. The latter part of this discussion will focus on making sure the patient understands the concept of neuroplasticity, how important it is for them to regulate their neurological systems finally give them control and thus hope in dealing with their condition.

Financial Interest: No financial interests.

Pre WS 6: Clinical Significance of Paroxysmal EEG Discharges: Location Links Pathology

(Lecture)

Jay Gunkelman, QEEG-D, Q-Pro Worldwide, qeejay@sbcglobal.net
Ron Swatzyna, PhD, The Tarnow Center for Self-Management, Ron@tarnowcenter.com
Level of Difficulty: Intermediate

Abstract

The overarching reason for doing an EEG is to find evidence of an organic brain abnormality (Hughes, 1994) 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 (Hughes, 1994). We have found that sharp paroxysmal discharges provide valuable supporting evidence of underlying pathology.

In this workshop, the qualification for those providing EEG interpretation will be established for qualified diagnosis (Neurologists, electroencephalographers, Psychiatrists, and Neuropsychologists, if they have proper licensure and board certifications). The difference between diagnostic and non-specific findings will be shown with specific reference to the literature (Niedermayer). We will demonstrate the presence of paroxysms in clients clinically symptomatic for seizures and in those without convulsions, but showing significant discharge related symptoms. The presentation is solidly grounded in the published EEG literature, and is not a new theory or approach, but merely suggests a clinical team approach with qualified individuals handling their specialty areas. The published literature being quoted lists many of the clinical presentations these paroxysms may take, from ischemia, cerebrovascular insufficiency, migraine ischemia, TIA, epilepsy, sensory/cortical processing disturbances including PDD/LD/ADD and others. Real clinical cases encountered within the last year will be used to illustrate this team approach.

Case 1
A 16 year-old female referred by her neurologist for chronic tension and migraine headaches. An EEG/QEEG revealed TMSSA in the right posterior temporal region. Asokan et al. (1987) also linked TMSSA to hippocampal ischemia, which in this case was the most likely source of her migraine headaches. The QEEG-Guided neurotherapy suppressed the TMSSA and in doing so, the migraines did not develop. The young patient was able to go on with her life no longer disabled by her headaches.

Case 2
A 55 year-old female presented with deterioration of language function. Visser et al. (1987) found that slight left-sided anterior temporal abnormalities are an early subclinical sign of temporal lobe pathology expressed in deteriorating language functioning. Although the MRI was negative, the identification of TMSSA prompted her neurologist to order a Magnetic Resonance Angiogram (MRA). The MRA identified a 9 mm aneurysm on her left interior carotid artery (paraclinoid area). Although the location of the aneurysm was physically outside of the brain (behind and below the left eye), the TMSSA was centered in the left anterior temporal lobe directly over the location. An endovascular stent and coil procedure stopped the progression of the aneurysm.

Case 3
Case three involved monozygotic twin females 8-years old. One of the twins was epileptic while the other was not but had several learning disabilities. This case demonstrates how a dramatically abnormal EEG can be without convulsion. Three EEGs will be shown and a discussion regarding medication and neurotherapy as an option. The critical nature of a working as a team with neurologists and psychiatrists are obvious in cases like these.

Conclusion
Subclinical EEG abnormalities require an examination of associated findings (Daly & Pedley, 1997). If the location of the paroxysmal EEG discharges correlates with observed symptomology, it is highly likely that discharges are responsible. Therefore, when the team agrees, a diagnostic leap can be proposed and efficacious treatment designed.

References

Goals/Objectives
1. Explain the rationale for identifying subclinical paroxysmal EEG discharges.
2. List the literature discussed in the workshop regarding subclinical EEGs.
3. Explain the link between ischemic migraine and paroxysmal discharges.
4. Know how to locate the source of the discharges.
5. Explain how an MRI can miss what an MRA can identify.
6. Theorize how an aneurysm may affect the EEG.
7. Explain how one twin could present with epilepsy and the other not.
8. How the workshop can help diagnosis difficult cases.
9. Know where to go to learn more about paroxysmal EEG discharges.

Outline
1. Introduction and literature review (Gunkelman)
2. Case 1 (Swatzyna)
3. Case 2 (Swatzyna)
4. Case 3 (Gunkelman)
5. Discussion (Swatzyna & Gunkelman)
6. Cautions and Clinical implications (Swatzyna)
7. Q & A (RS & JG)
8. Summary (Swatzyna)

Financial Interest: No financial interests.

Pre WS 7: Applied Neuroscience in Clinical Practice: What Can Event Related Potentials Add for Diagnostic and Treatment Procedures?
(Lecture, Demonstration)
Juri Kropotov, Institute of the Human Brain, jdkropotov@yahoo.com

Credits: CME – 8, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences- 8, BCIA recertification – 8

Level of Difficulty: Intermediate to Advanced

Abstract
Electrophysiological studies play important role in applied neuroscience. Because of high temporal resolution they are the only methods that allow neuroscientists to assess brain functions. Research shows that quantitative EEG (QEEG) and event related potentials (ERPs) reflect quite independent parts of brain functioning: QEEG reflect mechanisms of cortical self regulation whereas ERPs reflect information flow within cortical neuronal networks. The patient might have a normal self-regulation but abnormal information flow, and vice versa. Meta analysis of applied neuroscience literature within the frames of “diagnosis and treatment of brain dysfunction” shows that number of papers in ERP research is 10 times larger than the number of papers in QEEG research with this ratio dramatically increasing over the last five years. The effect size in ERP discriminant (patients vs. norms) analysis is usually much higher than the effect size in QEEG analysis. The workshop is intended to introduce the ERP field 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 and sLORETA imaging. Application of ERP for diagnosis ADHD, schizophrenia and TBI will be discussed. Biomarkers of these disorders will be presented. Finally, our own experience of using ERPs for constructing protocols of neurofeedback and tDCS will be discussed.

References

Goals/Objectives
Learn the basics of ERP recoding using Psytask as presentation software.
Learn averaging technique.
Understand how to preprocess the EEG record: artifact correction and artifact marking.
Explain independent component analysis on collection of ERPs.
Describe the physiological meaning of different ERP waveforms (literature analysis).
Understand the physiological meaning of ERP independent components.
Learn the discriminative power of some ERP independent components (such as P3b, sensory mismatch, P3 suppression, P4 monitoring) in diagnosis of ADHD, schizophrenia, TBI, OCD, dyslexia.
Practice assessment of brain dysfunction by means of comparing individual ERP with the normative data

Outline
Morning (4 hours): theoretical
a) Methods of recording, computation and analysis of ERPs (0.5 hour)
b) Extraction independent components associated with different psychological operation (0.5 hour).
c) Association of ERPs components with functioning of brain systems (1 hour).
d) Reflection of dysfunctioning of brain systems in ERPs components (1 hour).
e) What neurotherapy protocol can be suggested on the basis of ERP assessment. (1 hour)
The attendees are requested to bring laptops with them. The HBIdb software for analysis of ERPs will be provided together with EEG files recorded in healthy subjects and patients with ADHD, schizophrenia and TBI. During four hours the attendees together with the author will analyze the data, define what brain system is impaired and what neurotherapy protocol can be suggested for the patients.

Financial Interest: The NovaTech company and Mitsar company are paying for this trip. I am a co-owner of the HBImed AG company (Switzerland).

Pre WS 8: Physiology, Clinical Outcomes, and Most Current Research on Audio-Visual Entrainment, Cranio-Electrical Stimulation, and Transcranial DC Stimulation
(Lecture, Demonstration)
Dave Siever, Mind Alive, Inc., avedave@mindalive.com

Credits: CME – 8, American Psychological Association, NBCC, ASWB and CA Board of Behavioral Sciences – 8, BCIA recertification – 8

Level of Difficulty: Intermediate

Abstract
Stimulation modalities of Audio-visual Entrainment (AVE), cranio-electro stimulation (CES) and transcranial DC stimulation (tDCS) have been in clinical use for several decades. While CES is a fairly mature technology with over 130 studies demonstrating its effectiveness, AVE and tDCS, on the other hand, are seeing a new study emerging every few months.

With the publishing of the SAD & reduction of worry in college students last fall, AVE shows more excellent applications for college students in boosting GPA, concentration, memory and reduced worry. This new research builds on previous studies showing the effectiveness of AVE in promoting relaxation, cognition and hypnotic induction, treating ADD, PMS, SAD, PTSD, migraine headache, chronic pain, anxiety, depression, episodic memory and academic performance. Since the discovery of photic driving by Adrian and Matthews in 1934, much has been discovered about the benefits of brainwave entrainment (BWE) or audio-visual entrainment (AVE), as it is commonly known today. The first clinical applications of AVE are the credit of Sidney Schneider who developed the first photic stimulation device called the Brain Wave Synchronizer in 1958 and prompted the first research. AVE affects cerebral blood flow, neurotransmitters, dissociative states and brainwave activity.

Roughly 130 studies have been published on Cranio-electro Stimulation (CES). Most of the roughly 130 studies have shown CES as a reliable method to reduce anxiety, depression, pain, improve sleep, and improve cognition and IQ. Newly acquired research had demonstrated that CES is also effective in reducing pain and anxiety during dental procedures. Current interest in CES was initiated by Robinovitch, who, in 1914, made the first claim for electrical treatment of insomnia. In 1958, the book *Electro-Sleep* inspired research in Europe and in Eastern Block countries, as well as in South America, Asia and finally the US.

Transcranial DC Stimulation continues to thrive with new studies, two of which include enhancing mathematics ability and for treating Alzheimer’s. A major advantage of tDCS is that the cortical activity over a specific site on the brain may be enhanced or suppressed, much like NF with much greater ease and reduced risk of error. All that is needed is a brain functional diagram such as a Brodmann Area chart. A “new” form of stimulation is transcranial AC Stimulation, which may be nothing more than mislabeled CES by unsuspecting researchers. The differences between CES and tDCS will be explained. Over 80 studies involving tDCS have been published to date.

All maladies are the result of dysarousal on a physical or cortical level caused by genetics, life events and poor nutrition. This course covers both physical and cortical arousal issues and how stimulation technologies can restabilize one’s arousal. This course will teach the physiological mechanisms (neurotransmitter effects, brain waves, cerebral blood flow, dissociation, and neuronal activation) by which AVE, CES and tDCS act on, plus the clinical outcomes of each.

This course is particularly of benefit to those who have clinical experience and realize the need for some more innovative tools in their tool chest. This course applies to nurses, MDs, hypnotherapists, naturopaths, chiropractors, massage therapists, biofeedback and neurofeedback practitioners.

References


**Goals/Objectives**

1) understand a wider perspective on the causes of arousal dysregulation.

2) understand the physiological mechanisms of AVE/BWE and the studies that have shown efficacy.

3) learn about using AVE for heart-rate variability and ADD/ADHD.

4) see an AVE demo.

5) learn about the physiological mechanisms of CES and the studies that have shown efficacy. Experience the various forms of CES.

6) learn about the physiological mechanisms of transcranial DCStimulation and the studies which support tDCS.

7) experience tDCS.

8) learn how to use AVE to enhance Heart Rate Variability (theory and practice).

**Outline**

How genetics, life experiences, nutrition and prenatal factors affect arousal (80 minutes)

We will review QEEGs and observe how arousal is reflected in the brainwaves (25 minutes)

We will learn about the differences between arousal dysregulation caused by cortical inputs vs hypothalamic dysregulation (15 minutes)

We will learn about the physiological mechanisms of AVE (25 minutes)

We will review the previous research on AVE (30 minutes)

We will review research using AVE to treat ADD/ADHD (25 minutes)

We will see how AVE affects heart rate variability (20 minutes)

We will learn to breathe to the AVE breath pacer (20 minutes)

We will see a demo of AVE & CES (50 minutes)

We will learn the theory of operation of CES (10 minutes)

We will review the research on CES (15 minutes)

We will learn about the theory of operation of tDSCS (25 minutes)

We will review the research involving tDCS (20 minutes)

We will learn how to operate the equipment (30 minutes)

We will experience AVE, CES and tDCS (90 minutes)

**Financial Interest:** David Siever is the owner of by Mind Alive, Inc.