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New technologies are emerging so fast today that it is difficult to keep up. It seems like only yesterday I recall the big controversy over whether LENS was neurofeedback or not. We had a pretty robust discussion about it during a board meeting back when Jon Walker was president of the neurofeedback division at AAPB. Today, it is pretty well accepted, and now we have other technologies, “newbies,” being discussed in this issue. This, too, has sparked a large amount of controversy and some fairly dark back and forth on the list serves. It often becomes personal. People’s fortunes, reputations, and cherished beliefs become intertwined with scientific validity and reliability assessments. Some are afraid of losing their audience and of seeing their favorite technology lose the spotlight. Others are afraid of not ever getting into the spotlight.

What technologies should we let in the door to be associated with neurofeedback? Not long ago, neurofeedback was the newbie in biofeedback and garnered a fair amount of suspicion and concern. Then look what happened. They say people in glass houses should not throw stones. Today's dubious technology may be tomorrow’s dominant technology. The naysayers, however, do perform an important service. They challenge the newbies to make a “good” case. This is part of a larger universal process in all fields involving acceptance. After all, we have to filter some of it or we will be overwhelmed with chaos. The difficult task is to challenge without too much attachment. Science is always in transition. There are no “hard” facts because they melt away into the next dominant paradigm just as Newton gave way to Einstein. With this in mind, many authors recommend a healthy skepticism guard-

Continued on page 10
Letter from the ISNR President

Robert Coben, PhD

It is my pleasure to serve as the president of ISNR this upcoming year, after having served as member at large on two different occasions and as president-elect last year. Over the past year, we have sought to modernize ISNR by moving towards online publications. This has included the online publication of *NeuroConnections*, our newsletter. We have also transitioned our print journal to an online, open-access journal called *Neuroregulation*. This now enables anyone in the world to access our publication information so that more people can learn about neurofeedback and neuromodulation. Lastly, we have now sponsored two special issues in the open-access journal *Frontiers in Human Neuroscience*. These topics have focused on aspects of seizure disorders and autism. Again, this enables more researchers and consumers to learn about what we do and the science behind it.

The fields of neurofeedback, neuromodulation and applied neuroscience are growing, with their empirical support becoming more widely disseminated. More and more people are learning about us and from us. However, we still have some concerns. As a result, we as a board of directors have set several goals for the upcoming year. This, of course, includes the financial security of ISNR. We are looking to grow our organization so that we may serve our members in more diverse ways, including the provision of education and training which we believe is crucial. Our second goal is to provide unbiased and high level education to our members and others throughout the world. ISNR University was started this past year with the goal of doing just that, in an online format.

Any evidence-based practice and profession must be guided by ethics and sound science. We will be taking a new look at both of these aspects on an ongoing basis. We are forming and revising a new ethics committee and will look to update our ethical code of conduct. This committee will serve as a resource for professionals and work with those whose ethical behavior does not match our code of conduct. Lastly, we continue to strive towards encouraging sound science addressing our work and how it can become even more effective. As such, we have formed a new scientific council that will promote research and guide *NeuroConnections* and *Neuroregulation* to be the best they can be. It is our goal to then spread the word of our work and how it can help people all around the world.

We look forward to the upcoming year and all the opportunities that it may present. As always, we wish to include all our members and are open to input at any time.
Letter from the AAPB Editor
Roger Riss, PsyD

“Always listen to experts. They’ll tell you what can’t be done, and why. Then do it.”
—Robert A. Heinlein

Counting horses’ teeth

In the year of our Lord 1432, there arose a grievous quarrel among the brethren over the number of teeth in the mouth of a horse. For thirteen days the disputation raged without ceasing. All the ancient books and chronicles were fetched out, and wonderful and ponderous erudition such as was never before heard of in this region was made manifest. At the beginning of the fourteenth day, a youthful friar of goodly bearing asked his learned superiors for permission to add a word, and straightway, to the wonderment of the disputants, whose deep wisdom he sore vexed, he beseeched them to unbend in a manner coarse and unheard-of and to look in the open mouth of a horse and find answer to their questionings. At this, their dignity being grievously hurt, they waxed exceeding wroth; and, joining in a mighty uproar, they flew upon him and smote him, hip and thigh, and cast him out forthwith. For, said they, surely Satan hath tempted this bold neophyte to declare unholy and unheard-of ways of finding truth, contrary to all the teachings of the fathers. After many days more of grievous strife, the dove of peace sat on the assembly, and they as one man declaring the problem to be an everlasting mystery because of a grievous dearth of historical and theological evidence thereof, so ordered the same writ down.”
Attributed to Francis Bacon, 1592 (Mees, C. E. K., 1934).

As young students, many of us were first exposed to the story of the “horses’ teeth,” as an allegorical teaching tale about the rise of the scientific method and the dangers of over-reliance upon the entrenched opinions of the past. Five hundred years after the rise of the age of science, it may come as a rude awakening when we are confronted with evidence that we are not immune to the same blind spots in reasoning and powers of observation as were our “pre-scientific” ancestors.
How do you lose an important part of the brain for 100 years?
Jason Yeatman and colleagues at Stanford University recently announced their “discovery,” via diffusion tensor imaging, of a large vertically-oriented fiber-bundle in the visual cortex, which they labeled the vertical occipital fasciculus (VOF). That they were the first to observe this structure was perplexing; not only was this pathway readily visible on diffusion tensor imaging, but it also appeared to play a prominent and unique role in visual processing, as the only major fiber bundle connecting the dorsolateral and ventral lateral vision systems of the brain. How had it been missed by neuroanatomists? They searched in vain for previous references to the VOF in the anatomical literature but found no mention of it, until their detective work led them to anatomy texts from the late 1800’s.

There they learned that their “discovery” was in fact first described more than 100 years ago, by none other than famed German neurologist Carl Wernicke, author of the classic studies of language deficits in stroke patients who, at the time, was a graduate student studying neuroanatomy in Theodor Maynert’s laboratory at the University of Vienna. Wernicke noticed the VOF in slices of monkey brain, and included it in his 1881 brain atlas, naming it the senkrechte occipitalbündel, or ‘vertical occipital bundle’. Unfortunately, this discovery ran counter to the entrenched orthodoxy of the time, which was championed by none other than Theodor Maynert, Wernicke’s mentor, and a highly influential neuroanatomist of the time. Maynert held that long distance white matter tracts of the brain run only in a horizontal, and never in a vertical direction. Therefore, Wernicke’s discovery simply had to be wrong. Maynert remained influential and continued to refuse to acknowledge Wernicke’s discovery up until his death in 1892. While the VOF appeared from time to time in subsequent publications, it was largely ignored, eventually falling into obscurity, until it’s recent rediscovery by Yeatman and his Stanford team.

Neurofeedback researchers and practitioners may be forgiven if they sense obvious parallels between this saga and their own struggles to gain recognition for an intervention whose arrival seems, at times, to elicit responses from critics ranging from disbelief, to indifference, to the constantly shifting bar of “if only you had just one more well-controlled study”. If so, perhaps they can find some comfort in the recognition that, with time, old viewpoints, along with their defenders, do eventually pass away, making way for new sets of eyes, ready to take a fresh look at evidence which has been in front of them all along.

In the following pages readers will find part 2 of our thematic issue devoted to neuromodulatory interventions which are increasingly finding their way into neurofeedback practice. The concept that neurostimulation as an adjunct to traditional therapies (not limited to neurofeedback) has potential to synchronistically potenti-
ate therapy outcomes, is a growing part of the conversation, yet much has still to be learned about this emerging class of tools. In the pages within this issue, our contributors continue to share their observations and clinical experiences in the hope of informing dialog regarding the strengths and limitations of this emerging class of interventions. Like counting horses’ teeth, their observations and case histories should be understood as accounts of clinical experience, and a small but important contribution to larger research efforts underway.

References


Getting Started with Pulsed Electromagnetic Field (pEMF) Therapy

John N Demos, MA, LCMHC, BCN

Introduction

Many will be surprised to learn that pulsing magnetic fields have long been acknowledged by the medical community for fracture healing and inflammation reduction. Currently, a variety of devices have been either registered or approved by the FDA and Health Canada; also, they have been widely accepted in Europe and among veterinarians for many decades.

Low intensity pulsed electromagnetic Field (pEMF) devices are becoming more common in neurofeedback clinics. Magnetic coils are placed strategically on the scalp. Each coil outputs electromagnetic pulses at a preset frequency or frequency pattern. Reports from neurofeedback providers indicate that pulsing magnetic therapies often reduce the total number of sessions.

In general, pulsing magnetic therapies do not fall under the heading of biofeedback self-regulation or operant conditioning. However, designs may be created that emit pulses which are contingent upon or in sync with the flow of the EEG. Hence, the magnetic pulses become part of, or are in a loop with, the biofeedback process. Additionally, magnetic pulsing may well influence EEG frequency. And there is overwhelming evidence that it promotes positive changes at the cellular level.

Pulsing Magnetic Fields: High Intensity vs. Low Intensity

Magnetic fields are measured as follows:

- 1000 milliGauss = 1 Gauss
- 10,000 Gauss = 1 Tesla

Low Intensity pEMF devices for the brain range from a mere 0-to-2 Gauss or 0-to-2000 “milli”Gauss. They should not be confused with high intensity devices for the body that may range anywhere from 1-to-20,000 Gauss. High intensity devices are used to relieve bodily pain or to heal fractures. High intensity pulsing near the brain may be harmful and should be avoided by mental health practitioners.

However, some medical doctors employ high intensity pEMF which is also known as Transcranial Magnetic Stimulation (TMS). For example, one commercial product that is approved by the FDA is Neuronetics NeuroStar TMS system™ which states the following:
“The peak magnetic field strength achieved with each pulse in the cortex is approximately 0.5 Tesla.” (User Manual, 2006) (Note: .5 Tesla = 5000 Gauss).

Pulsing at 5000 Gauss or (.5 Tesla) causes a hyperpolarized state at the neuronal membrane. Hyperpolarization and depolarization are related to the chemical/electrical activity of nerve cells and action potentials. Hence there is a rapid impact on cellular processes. (Stade, 2011)

Neuronetics NeuroStar TMS System™ is approved by the FDA for the treatment of drug resistant depression. One research study testing NeuroStar TMS Therapy® indicated the following: “Transcranial magnetic stimulation is effective in treating major depression with minimal side effects reported. It offers clinicians a novel alternative for the treatment of this disorder” (O’Reardon, 2007). Machines such as the NeuroStar are costly: $75,000. Treatment costs: $6,000-to-$12,000 for the four-to-six week treatment. Coil placement (or targeting) is typically based on Davidson’s research articles consequently pulsing is limited to the DLPFC (Davidson, 1988).

Neuronetic NeuroStar’s output is in sharp contrast to low intensity devices commonly used by neurofeedback providers that do not generate rapid changes to action potentials in nerve cells. Low intensity devices are known to stimulate cellular growth and repair and enhanced electrical flow. Interestingly, low intensity pulsing may influence EEG frequencies.

One extensive study in the Journal of Neurofeedback reviewed the topic of pulsing magnetic fields (Mehran, et al., 2013). The research and literature review in this article came to the following possible conclusions about pEMF and EEG frequencies:

1. Magnetic pulsing “causes the reinforcement of brain signals in the same frequency as the exposure field.” Note, this statement was limited to wide fields of exposure pulsing at a specific frequency that may well result in greater amplitude of that same frequency.

2. Magnetic pulsing with narrow fields of exposure promotes a variety of frequency responses. Hence, 10 Hz pulsing with a single coil at T4 cannot be relied upon to increase 10 Hz amplitude at T4.

3. pEMF pulsing may increase epileptiform activity in patients who are at risk for seizure.

According to a review of the literature incorporated into the 2009 TMS application guidelines, using high intensity stimulation, the risk of seizure in healthy subjects is <1% with either LF rTMS or HF rTMS (Rossi S, Hallett M, Rossini PM, Pascual-Leone A., 2009). However, the risk of inducing seizures is controllable because it is a function of frequency and field strength. Importantly, the field strength of low intensity pEMF devices, such as those used as an adjunct to neurotherapy practice, fall well below
the field strength utilized in rTMS: the field strength of these devices is not 100–220% (Wassermann, 1998) of the motor evoked potential threshold, but in the order of 0.05%, significantly reducing likelihood that induction of epileptic seizures is a risk with this class of devices (Kortekaas, et al, 2013).

Additionally, there is some concern about how to measure the intensity (Gauss) of pulsing magnetic signals: The output of magnetic coils are best measured by commercial analog milliGauss meters or high-end digital gauss meters such as Trifield’s “DC Gauss Meter Model GM2,” because it measures both DC and AC magnetic pulses. Avoid inexpensive digital MilliGauss meters which cannot accurately measure pEMF pulsing signals.

When it comes to measurement, pulsed electromagnetic fields should not be confused with LENS signals in which “The EEG signals traveling back to the client are profoundly weak, a million times weaker that the signals from the Alpha stim.” (Ochs, 2011). Nor should they be confused with non-pulsing invasive Transcranial DC stimulation (tDCS) that requires an anode and a cathode to deliver a weak but measurable electric current directly into the scalp. (Siever, 2013).

Benefits of pulsing magnetic therapies (literature review)
Before any new therapy is introduced into a neurofeedback clinic it is essential to know how it is viewed by the scientific community. Health benefits and statements made to patients should be supported by peer reviewed research. The following quotes are consistent with countless (perhaps thousands) of research articles referring to pEMF (emphasis added):

“Fueling this recent interest is the fact that extremely low-frequency and low-intensity pulsed electromagnetic fields (PEMFs) have been shown to be innocuous, possibly even beneficial, to normal cell types. On the other hand, certain malignant cell classes have been shown to be particularly vulnerable to their effects.” (Crocetti, 2013)

“Previous papers have reported that PEMF exposure could act modulating cartilage and bone metabolism, stimulating chondrocyte and/or osteoblast cell proliferation and the synthesis of extracellular matrix components. The stimulation of chondrocyte and/or osteoblast cell proliferation induced by PEMFs has been shown to have a positive effect in the treatment of fracture healing.” (Vincenzi, 2013)

“Numerous clinical studies have reported that pulsed electromagnetic fields (pEMF) are able to modify some parameters of nerve function in diabetic patients, and a voluminous amount of literature has suggested that pEMF can stimulate nerve growth, regeneration, and functional
The aforementioned articles are consistent with volumes of research studies and literature. Pulsed electromagnetic fields promote healing in various cell types. The following is a partial list of conditions that have responded:

- inflammation (Kaszuba-Zwońska, 2008)
- bone-fractures (Satter Syed, 1999)
- migraines (Sherman, 1999)
- insomnia (Moore & Kube, 2013)
- chronic stroke (Avenanti, 2012)
- Parkinson's (Sandyk, 1992, Dogris, 2012)

pEMF is not a new treatment; its origins date back to the work of Nikola Tesla (120 years ago). Pulsing magnetic therapy is firmly grounded in research. It is a serious therapy and knowledge of its proper use is required.

**pEMF Frequency Selection Hypothesis: Basic Terms and Considerations**
Pulsed electromagnetic therapy has established its clinical value; however, efficacy is related to application. On the one hand, it improves cell functioning and reduces inflammation which means that positive clinical changes can occur with or without EEG changes. On the other hand, when EEG changes are desired, how can optimum pulsing frequencies be chosen?

Mehran’s (2013) research implied that a wide field exposure pEMF may result in a corresponding frequency enhancement. This effect may be the result of ions that resonate with weak magnetic fields. Changes to EEG amplitudes within a specific frequency domain would thus be mild.

Another form of wide field exposure is photic stimulation, which is an entrainment therapy. pEMF is not an entrainment therapy. However, if frequency pattern selection can be modeled after photic stimulation, then three principles apply: stimulation, inhibition and random frequency output (disentrainment).

For an example of wide field photic stimulation, notice the change in the EEG due to photic pulsing at 1 Hz and then at 14 Hz in Figures 1 & 2:

Photic pulsing impacts the visual cortex found in the occipital lobes: changes to EEG amplitudes within a specific frequency domain are significant! Photic pulsing activates brain potentials which resonate at the same frequency of photic pulses. Next, the EEG is influenced by the pulsing brain potentials. Consequently, evoked potentials that pulse at 1 Hz or 14 Hz promote higher amplitudes of the exact same

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1 See website links in bibliography
EEG frequency as shown in Figures 1 & 2. Clearly, the above figures show the effect of photic “stimulation.”

An example of inhibition comes from a study by Collura & Siever (2009). Notice in Figure 3 the trainee has elevated theta amplitudes, likely ADHD. For the first 20 minutes, neurofeedback training alone had little effect on theta reduction. Next, a new feedback component is introduced: 14 Hz photic pulsing was triggered each time theta (4-8 Hz) exceeded threshold conditions. Hence, in this neurofeedback design\(^2\) photic stimulation was contingent upon EEG activity. Results: 14 Hz pulsing set to react to EEG activity significantly reduced theta.

The example at left sets forth a principle: 14 Hz stimulation (contingent on EEG activity) tends to inhibit 7 Hz EEG amplitude. How far reaching is this principle? To answer that question, Dave Siever was consulted as an authority on photic pulsing. His response, “The 2:1 principle has been observed between 10 and 20 Hz.” In other words, 10 Hz stimulation results in 5 Hz suppression or 20 Hz stimulation results in 10 Hz suppression.

Even though pEMF is not the same as entrainment, protocol designs may well benefit from the above concepts.

Random frequency patterns constitute the third pulsing principle. It has its roots in equipment such as the Roshi, NeuroField,\(^3\) and it is now available on MicroTesla by BrainMaster. Random frequency outputs can be altered every few seconds; they may continue for 10–40 minutes as a stand-alone therapy. Or, random pulses can be output each time EEG training goals are no longer being met. The purpose of random pulsing is to set the stage for change; shifts in training frequency output likely result in shifts in entrenched EEG frequencies. For example, anxious clients who are “stuck”

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\(^3\) Patent pending
in a diffuse beta pattern may find relief from the anxiety often reflected by high amplitudes of beta. Furthermore, those same clients may now respond to subsequent EEG biofeedback interventions designed to suppress diffuse beta patterns.

**Note:** pulsing at low frequencies may promote greater epileptiform discharges in patients at risk. Therefore, random pulses from 10 Hz–100 Hz may well be safer than random pulses from 1 Hz–100 Hz because the wave component of “spike and wave” discharges is a slow frequency, e.g. 3 Hz.

Another principle of note that has been utilized by photic stimulation devices has been labeled “dissociation” by Dave Siever. Again we note his observation: When photic pulsing is set to generate frequency patterns in the delta range (e.g. 1, 2, 3 and 4 Hz) as a part of the training regime then entrenched patterns may be weakened and the potential for change is enhanced. (Care is always observed when working with patients who have a history of seizure.). Low delta z-scores are common.

**Pulsing Within a Set Range**

Pulsing devices do not use bandwidths such as delta, theta, alpha and beta; rather, they output single frequencies within bandwidths. For example, if a brain map indicates weak alpha in the dorsal posterior of a client, pulses are chosen within the range of 8–12 Hz. Likely, multiple coils (wide field exposure) would be placed near Pz. In general, pulsing “steps” can be set in any one of the following ways. For the sake of simplicity, pulsing (frequency as measured in Hertz) is set to change every five seconds although this is not a rule:

- Repeating steps: 8, 9, 10, 11, 12, 8, 9, 10, 11, 12
- Reversing steps: 8, 9, 10, 11, 12, 11, 10, 9, 8
- Random steps: 8, 10, 12, 9, 11, 10, 8, 11, 12, 9, etc.
- Fine step: 8, 8.1, 8.2, 8.3, 8.4, 8.5, 8.6, 8.7, 8.8, 8.9, 9.0, 9.1, 9.2, etc
- Very fine step: 8.0, 8.01, 8.02, 8.03, 8.04, etc.
- Low frequency steps: .31, .32, .33, .34, .35, .36, .37, etc.

**Protocol Example:**

Working the above principles, the following “automated” BrainMaster MicroTesla z-score protocol has been used successfully. The components (Event Wizard) of this protocol are as follows:

- Z-score training is set to auto adjust.
- Random pulses are output for almost 45 seconds until calculations are made
- Z-score measurements determine which one of the following bandwidths are the furthest (maximum) from the mean: delta, theta, alpha, beta, hi-beta.
- Pulsing is auto driven based upon the principles of stimulation and inhibition.
For example, if the highest z-score is theta (4-8 Hz) then pulsing is automatically output from 12-16 Hz based upon the principle of inhibition.

- As long as z-scores exceed dynamic threshold requirements, pulsing continues; however, if z-scores drop below dynamic thresholds, then random pulsing is output.
- Pulsing changes output if a new bandwidth takes the lead as the furthest from the mean of the database.

The above protocol requires no operator intervention. Coils should be placed strategically on the scalp. Pulsing selections come from BrainMaster's Session Wizard, including: repeating, reversing, fine or very fine steps.

**Note:** in general, more than one coil is used (2-4 coils) to generate a wide field exposure, rather than a narrow field exposure. When using a narrow field it would be useful to observe changes to qEEG outputs to determine if the specific frequency intervention at a specific location produces the desired result. QEEG and LORETA will continue to be very useful tools in the future to determine best coil location and optimum intensity.

What are the results from (earlier versions of) the above protocol?
Encouraging Words Counseling Center, Lewisburg, WV: Terry Lusher, director, offers the following report, “We have been doing neurofeedback for four years. Twelve months ago BrainMaster’s MicroTesla (MT) automated training was added to every session. (Approximately 40-60 sessions per week, or 2500 sessions) Clients are more apt to report relaxation with reduced symptoms of anxiety (also, PTSD), racing thoughts, as well as depression, since MT has been added. Overall, we have a 10–15% reduction in the total number of sessions. Each client fills out depression and anxiety inventories and is re-mapped every 10 sessions; positive changes to qEEG are significant. Clinical effectiveness has improved over the past 12 months using 2 coils (Type 3) of BrainMaster’s MicroTesla pEMF, guided by z-score data. (Encouraging Words Counseling Center uses an earlier version of the automated protocol described above, [http://www.encouragingwordscounselingcenterwv.com/](http://www.encouragingwordscounselingcenterwv.com/)).

**Conclusion and suggestions:**
For more information on getting started with NeuroField’s low intensity pEMF please consult N. Dogris at [http://www.neurofield.org/](http://www.neurofield.org/).

For those getting started with BrainMaster’s MicroTesla sub-threshold pEMF, consider the following possible ways to commence with coils:
- Use a multiple coil design
- Start clients with stand-alone, random pulsing for 15-30 minutes. Consider following this treatment with basic z-score or power training protocols. Place
two coils at C3 C4 or four coils at F3F4 & P3P4. Do not hesitate to move coil positions if goals are not met.

- Start with the automated protocol as described above.
- Start by pulsing within the alpha range in posterior locations alone or in combination with EEG biofeedback. Employ different combinations of frequency output steps. Pay special attention to finer steps. When training for EEG power enhancement or inhibition create designs that are contingent upon EEG output.
- Show caution when patients have a history of seizure.
- *Set pulsing intensities low for new trainees*. For example, during the first five minutes pulse as low as 25–50 milliGauss. If the trainee tolerates this low level of intensity, increase to 100 milliGauss, and so on. Clearly some trainees can tolerate as much as 2000 milliGauss (or 2 Gauss) whereas others cannot. *Assess all new trainees for hypersensitivity to medications and environmental changes before using pEMF.*

Pulsed electromagnetic fields promote healing at the cellular level. Pulsing that is contingent on EEG output can be very powerful. Random pulsing promotes EEG flexibility. Reports indicate that neurofeedback outcomes are often enhanced with the addition of pulsed magnetic therapy. For further information about protocol development please send inquiries to: eegvemont@gmail.com.

**Suggestions for further research:**

- One commercial website that provides a list of pEMF interventions as well as references to research articles: [http://drpawluk.com/updates/](http://drpawluk.com/updates/).
- Additional resources can be found at: [http://www.pemf.us/docs/MedicalPEMF-Studies2.pdf](http://www.pemf.us/docs/MedicalPEMF-Studies2.pdf).
- Consult the NeuroField software manual.
- Also, consult BrainMaster Technologies knowledge base at [http://www.brain-master.com](http://www.brain-master.com)

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For What the Bells Toll (with apologies to Earnest)
Feedback vs. Driving
Paul G. Swingle, PhD, RPsych

My research has focused on developing techniques for increasing the efficiency and accelerating the process of modifying brain functioning. One such harmonic (OMNI) is a blend of several carrier frequencies providing a 10 Hz overriding frequency that is imbedded in a filtered pink noise at between −15 and −25 dB(C). The effect of the OMNI harmonic is that it suppresses EEG theta (3–7 Hz) amplitude and has been found to markedly accelerate the neurotherapeutic treatment of common attention deficit disorder (CADD) (Swingle, 2001).

The theta suppression is about the same with males and females (provided the sound pressure levels are presented at gender specific levels (see Swingle, 1992), but differs with age. For clients over 18, the suppression of theta amplitude is about 30% whereas for young children the suppression is about 15%.

It is not surprising that sound influences brain activity; further research has identified a number of harmonic blends that have specific effects on the EEG, such as reducing beta amplitude or increasing theta amplitude and thus can be very useful as adjunctive treatments for sleep or anxiety difficulties. Harmonics have also been developed to enhance the Sensory Motor Rhythm (SMR) and slower frequencies, suppress high frequencies (28–40Hz) and to speed up alpha.

Regarding the speeding of alpha, the following copy of a slide sent to me by the late Tom Budzynski (Figure 1) shows the effects of the OMNI harmonic. Of particular interest is the effect on the alpha band in which slow alpha is suppressed and fast alpha is enhanced.

The use of a stimulus like...
OMNI as an adjunctive treatment procedure, for example, to help an ADHD child focus while doing homework, is static in the sense that it is applied to have a specific effect on autonomic and/or central nervous system functioning. The OMNI harmonic, for example, is prescribed for home use by an ADHD child because at intake it has been determined that this stimulation will reduce theta amplitude for this child.

My research into braindriving technologies was stimulated by a long acquaintance with Len Ochs. As many readers know, Dr. Ochs is one of the pioneers in the development of stimulated EEG treatment procedures. Dr. Ochs demonstrated that making supraliminal light stimulation (Light Emitting Diodes (LEDs) mounted on eyeglass frames) contingent on EEG activity could be an effective neurotherapeutic treatment for a variety of disorders.

Contingency is the important concept, in that the light stimulation is contingent on brainwave training parameters such as amplitude, peak frequency, and the like. It has been demonstrated that the effects of AVS stimulation do not persist into post-stimulation baselines (Frederick et al., in press). Hence, these static procedures are not effective as stand-alone treatments other than in the context of relaxation or arousal for short term effects.

They can be effective, however, if coupled with some task. For example, when the OMNI harmonic is used while a child is doing homework, stable, albeit small, changes in theta amplitude are observed because of the increased efficiency of the brain while engaged in the homework. The cumulative beneficial effect over an eight week period was about 0.5% in one study (DuPont & Swingle, 1996).

Given the theta-suppressing effect of the OMNI harmonic, it seemed logical to use that harmonic to modify brainwave amplitudes by making the sound contingent on EEG events in a manner similar to that introduced by Len Ochs. This procedure, “braindriving,” is simply using the classical conditioning paradigm for neurotherapy. Make the unconditioned stimulus contingent on EEG activity.

That brainwave activity can be classically conditioned was demonstrated in the 1940s by Jasbir & Shagass at McGill University, who demonstrated that contingent light, an unconditioned stimulus for alpha suppression, will classically condition sound to suppress alpha.

Neurofeedback is said to be instrumental conditioning, and to some extent this is correct. If a child likes to see Pac-Man moving across the screen then it is a reinforcing stimulus for the child’s maintaining focus as measured by reductions in the theta/beta ratio.

Likewise, when doing an eyes closed protocol, the client hears a sound when the
brain is doing what we want. If the sound is not unpleasant then it is providing information to the brain and the client will often comment that the sound is “comforting.” One can think of this as classical conditioning, in that the relaxed state of increased slow frequency amplitude is associated with the tone. In this case, the tone itself may gain strength to increase slow frequency amplitude before it extinguishes.

However, if the client finds the “reward” tone annoying, then the instrumental conditioning may have some classical conditioning properties of blunting theta, for example. Clients often will comment that at some point in the session the tones have an irritating property if for no other reason than they can be taken as a signal for failure.

Providing the brain with information regarding state changes is a fundamental component of the neurofeedback paradigm. Screen-size changes while watching a video that reflect analogue changes in brainwave activity have also been reported to result in EEG changes. Likewise, magnetic stimulation in the milligauss range has been reported to be too weak for neuronal effects, and thus is simply providing the brain with information. However, this view has recently changed somewhat: “…these tiny magnetic fields have the potential to affect subthreshold trans-membrane potentials, producing minute but useful changes in brain activation” (Brainmaster website).

But of course one can use my favorite for providing information to the brain for desired EEG changes as shown in Figure 2. Smaller vibrating soft cuddly toys are for kids whereas the large vibrating toy is for adults needing comforting of their “inner child.” Is the vibrating bear a “reward” in instrumental terms or an unconditioned stimulus in classical conditioning terms? As soon as a distressed adult unashamedly cuddles with “Homer” (the name of one of our therapy bears) one sees immediate EEG and peripheral changes and the vibrations with EEG contingencies show unmistakable learning/conditioning.

Cuddly bear vibration feedback is very effective for treating infants. As shown in Table 1, a child with West syndrome became symptom free after three months
of intensive braindriving treatment. These clients came to the clinic for intensive daily treatment for their child and stayed in Vancouver for three and one half months. The child has remained symptom free.

Realtors are adamant that the only issue of importance regarding real estate investment is “location, location, location.” In neurotherapy there are two cardinal factors: 1: “valence, valence, valence” and 2: “contingency, contingency, contingency.”

For braindriving one needs to establish the valence of the stimulus. Is it negative or is it positive or somewhere in-between? Now, by negative I do not necessarily mean unpleasant but rather direction. For example, does the stimulus increase or decrease alpha? As pointed out earlier, light suppresses alpha amplitude. The OMNI harmonic suppresses theta amplitude. The parameters associated with this valence are hard data based, in this case on EEG measures.

Contingency is straightforward. How close to an event is the presentation of the stimulus? If the event is a training threshold crossing of a brainwave band, does the stimulus go on, or off? If one is using lights to suppress alpha amplitude then the lights come on immediately upon alpha amplitude crossing threshold. If one is using OMNI to enhance fast frequency alpha, then the OMNI stimulus would come on when alpha peak frequency dropped below threshold.

However, given that some stimuli have entraining properties, they can be used to “grab” or sustain an event. For example, if one wanted to increase theta amplitude then having a 7.8 Hz flashing light come on when theta amplitude was above threshold might help to strengthen and sustain (“grab”) the theta response.

It would perhaps be useful at this point to offer a few examples of stimulating or braindriving the EEG. The most straightforward example is a child with CADD in which the only remarkable feature of the ClinicalQ (more about this below) is high amplitude theta activity over the sensory motor cortex (location Cz). The usual treatment for this condition is theta inhibit, beta enhance neurofeedback over location Cz. The number of sessions required to treat this disorder using “conventional” neurofeedback is between 40 and 80 (Lubar, 1991). One can reliably and permanently remediate this simplest form of ADD in 15 to 20 sessions using braindriving technology (Swingle, 2001). We typically include brain driving in between one-third and one-half

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**Table 1:** Treatment of West Syndrome with 9-month-old male

<table>
<thead>
<tr>
<th>Theta/SMR</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-Treatment:</td>
<td>&gt; 10 @ C3, Cz, C4</td>
</tr>
<tr>
<td>Post-Treatment:</td>
<td>M=5.12, Best 3.23</td>
</tr>
</tbody>
</table>

**SEIZURES**

<table>
<thead>
<tr>
<th>WEEK SFD (%)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Start</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>45.0</td>
</tr>
<tr>
<td>8</td>
<td>57.1</td>
</tr>
<tr>
<td>10</td>
<td>87.7</td>
</tr>
<tr>
<td>11</td>
<td>100.0</td>
</tr>
</tbody>
</table>
of the neurotherapy sessions when treating this disorder. When theta amplitude is below the training threshold, the game icons move and the child hears the reward tone. When the theta amplitude goes above the training threshold, then the game icons stop moving, the child does not hear the reward tone, but the theta suppressing harmonic (in this case, OMNI) is presented, which suppresses theta amplitude.

A more complicated example is in the treatment of seizure disorders. Again, the conventional treatment for epilepsy is to enhance the amplitude and/or frequency of SMR operant responses over the sensory motor cortex (locations C3, Cz, C4). One should also set an inhibit on theta, because if theta amplitude increases when the SMR amplitude increases, there is a likelihood that seizure activity will remain unchanged or become worse even though SMR amplitude is increasing (Lubar and Bahler, 1976).

Using braindriving technology, one can cascade the units so the theta-suppressing harmonic is presented when theta amplitude increases above threshold, and the SMR-enhancing harmonic is presented when SMR amplitude drops below threshold. The braindriving technology can be used alone (i.e., no visual feedback) or with visual feedback displays. In most cases, braindriving is not used exclusively in the treatment of any condition, but is combined with conventional neurofeedback. This is a practical clinical decision, since the method of researching this technology has been to add it to the neurotherapy and observe the changes in the EEG, then determine if the enhancements are sustained in the ongoing neurotherapy treatment sessions. We have been systematically increasing the percentage of sessions in which braindriving technology is used. There have been cases in which braindriving has been used exclusively, but these have been cases in which there were circumstances mitigating conventional neurotherapy. One of these cases will be discussed in detail later in this article.

In keeping with the philosophy of rapid and efficient neurotherapeutic treatment, I have developed a rapid intake brain assessment—the ClinicalQ (Swingle, 2014). This rapid intake assessment requires about 6.5 minutes of recording at 5 brain sites (O1, Cz, F3, Fz, F4). At locations Cz, O1, F3 and F4, three brainwave bands are recorded: Theta (3–7Hz), Alpha (8–12Hz) and Beta (16–25Hz). At Cz and O1 we measure Eyes Open (EO) and Eyes Closed (EC) and also we test the harmonics to be used in the braindriving protocols, to verify that they affect brainwave amplitude as expected. At the frontal locations all recordings are EC. It should be noted that the ClinicalQ is not used in cases where a full nineteen-site brain map is warranted.

The following cases all proceeded from the ClinicalQ in which, aside from identifying areas for treatment, the effectiveness of the harmonic sounds for modifying brainwave activity had been established.
CASE 1 Joan: This young woman was under treatment for a severe anxiety disorder that manifested in eating difficulties and poor immune functioning, as evidenced by incessant colds and flus. Of several areas requiring treatment, one prominent brainwave feature was a markedly deficient theta/beta ratio at location O1. Her ratio was .54 whereas normative would be around 2.00. The neurotherapeutic treatment for this condition is to enhance theta amplitude and/or decrease beta amplitude at location O1.

Generally, one does not commence treatment with these brainwave bands, nor at that exact location, but gradually approach the training bandwidths and locations starting in areas and with bands that are easier for the client to master. However, this is beyond the scope of this paper; suffice it to say that the following example of braindriving occurred at the time when the client was ready for theta amplitude enhancement. In keeping with the strategy stated above of approaching the treatment frequency with more manageable (for the client) frequencies, we started with brain-driving alpha (8-12Hz). The potentiating harmonic for alpha amplitude enhancement is 6 to 8 cycles per minute, that is presented to the client any time alpha amplitude drops below the training threshold.

The baseline alpha amplitude was 3.2 microvolts (mv) that increased to 8.4 mv after 20 minutes of braindriving. Consistent with what one finds with alpha/theta neurofeedback training, when alpha amplitude increases, theta tends to increase as well. In this case the theta amplitude increased by 15.4% (from 5.2 to 6.0 mv) that resulted in an increase in the theta/beta ratio of 14.3%.

CASE 2 Karl: A man in his 50s who was under treatment for post-traumatic diffuse body pain and severe sleep quality difficulties. His initial ratio of theta to SMR (13–15Hz) was 4.40, whereas a normative range is below about 3.00. At the session to be reported here, his starting theta/SMR ratio was 3.29. The braindriving protocol was to present the OMNI theta-suppressing harmonic when theta amplitude exceeded the training threshold, and to present the SMR-enhancing harmonic when the amplitude of the SMR dropped below the training threshold.

Baseline measurements at the start of the session indicated theta amplitude of 5.6 mv and SMR amplitude of 1.7 mv. At the end of the session, the theta amplitude remained unchanged at 5.6 mv but the amplitude of the SMR had increased to 4.0 mv for a ratio of 1.40. It is unusual to have changes this large, but this case nicely shows that even with driving techniques, the brain “knows what it needs,” a concept most neurotherapists embrace, in that theta remained unchanged while SMR increased.
even though both were driven. Karl reported a marked improvement in the diffuse body pain at the next session.

**CASE 3 Susie:** This little girl was under treatment for a serious learning disorder. One of the things we noticed in her intake ClinicalQ was that the anterior cingulate gyrus was hyperactive. Her ratio of hibeta (28–40Hz) to beta was .88 at intake, whereas normative is .55 to .65. Hyperactivity of this structure is related to obsessive/compulsive forms of behavior, including stereotypy of thought, problems “letting go” of thoughts, stubbornness, and of particular concern in situations of learning disorders, often resistance to accepting different approaches to learning.

Braindriving with young children usually is integrated into conventional biofeedback procedures because braindriving alone is rather boring. One simply sits there while the computer delivers sound stimuli about thirty percent of the time. As described above, braindriving can be integrated into conventional biofeedback with visual icon displays. In this case, when the icons were not moving the braindriving sound stimuli were presented. This particular session with Susie was rather late in treatment. Her hibeta/beta ratio was down to .59 at the start of this session. The suppressing harmonic was 24.5 Hz and the feedback game display was Pac-Man. The braindriving harmonic was presented, on average, 30% of the time. Susie's end session hibeta/beta ratio had dropped to .53, that is well within normative range.

**CASE 4 Grant:** One of the most exciting applications of braindriving is with clients who have limited capacity for volitional biofeedback. Although it is an axiom of neurotherapy that the brain learns even if the client is not paying attention, nonetheless neurofeedback is compromised when the client has such limited capacities. Such clients include the more severe autistic spectrum disordered, psychotic, and brain injured. We have used braindriving with such clients, many of whom have become capable of fully cooperative volitional neurofeedback. Braindriving protocols for such clients include:

- Suppression of hibeta and beta amplitude over the anterior cingulate gyrus with autistic spectrum disordered children with a “hot midline”.
- So-called “squash” protocols, suppressing the amplitude of all frequencies from 2 to 25Hz, for developmentally delayed and severe FAS children.
- Slow frequency suppress and “speed-up” alpha protocols for stroke clients.

Grant spent the first 45 minutes of his first appointment screaming and thrashing on my office floor, despite heroic efforts of his parents. Fortunately, one of my staff members is a most talented young woman who works magic with these seemingly
unapproachable children. She was able to habituate Grant to tolerate electrodes on his head and to remain relatively quiet for a few minutes at a time watching videos of children’s animated cartoons. This habituation took several sessions, after which we started the braindriving protocols and obtained a ClinicalQ. The braindriving protocols included suppression of hibeta and beta over the frontal midline (Fz), “Squash” over the frontal (F3 and F4), and central (Cz) areas, and suppression of theta amplitude over the occiput (O1 and O2), as well as centrally and frontally.

There have been some remarkable changes in Grant. He converses in sentences, albeit awkward and clipped, interacts with peers, and, importantly, is capable of volitional neurofeedback where we are now addressing the anomalies found in his full 19-site qEEG. We started with the ClinicalQ after Grant was able to tolerate a single electrode, and this ClinicalQ guided our braindriving protocols. Once Grant was able to tolerate the full cap, we preceded to the full qEEG, which is guiding his current treatment.

With braindriving, we often find that the major effect occurs within the first few minutes and that prolonged treatment (20 to 30 minutes) yields little further gain, although may be important to stabilize initial changes. The following data are from a session with a severely traumatized woman in which the purpose of the session was to increase theta amplitude in the back of the brain (location O1).

**Case 5 Margie:** Her theta amplitude was 3.6 when she started. The data for the first 20 seconds of treatment indicate that the theta amplitude increased, at five-second intervals, as follows: 4.1, 4.6, 5.8, and 8.1. Thus, after 20 seconds of braindriving, her theta amplitude increased from 3.6 to 8.1 mv. After an additional 20 minutes, her theta amplitude increased to 10.1 mv, indicating that the amplitude had increased 125% in twenty seconds and an additional 24.7% after an additional 20 minutes.

**Case 6 Glenn:** The final case is a man who was physically beaten and later developed what was diagnosed as fibromyalgia. The data on treatment of fibromyalgia seem quite clear. Medications are not identified by patients as very helpful (other than treating comorbid depression), are identified by patients as a source of side-effects, and exercise and cognitive therapies are rated as effective treatments of all.

Glenn was a 67-year-old male who was physically attacked and beaten about the head, shoulders, and neck. Pain started in head, neck, and shoulders, and then moved to other body locations. Major complaints: sleep disturbance, poor concentration, memory loss, inability to organize tasks and to concentrate, depression, fatigue, irritable, loss of libido, right hand neuropathy.
Often neurotherapy is adjunctive to other treatments as a method for increasing the efficiency of those treatments. The treatment of fibromyalgia is generally a longer-term therapy, and one in which neurotherapy potentiates other treatment methods. In Glenn’s case, the major therapeutic goal was to improve his sleep, as is generally the case with all fibromyalgia patients.

Second, exercise and muscle exercises have been found to be more effective than medications for helping clients with this condition, so Glenn was given several home treatment procedures for this purpose. For sleep, Glenn was prescribed a Cranial Electrical Stimulator (CES) (which has been approved for treatment of sleep [also depression and anxiety]), which he was to use daily for at least 20 minutes.

He was also provided a pedometer to record the number of steps he walked every day. His preliminary activity was about 4,200 steps per day at the outset of treatment. He understood that an inactive person walks less than 5,000 steps per day and that the target for an active person is over 10,000 steps per day.

Finally, Glenn was also given a recording for guided Progressive Muscle Relaxation (PMR) and a second exercise version (cognitive tension reduction), which essentially is guided mental imaging of tightening and releasing the same muscle groups as in the PMR exercise.

Figure 3 shows the results of Glenn’s record of sleep and pain. Treatment started in March and continued for about 9 months. As the data indicate, Glenn’s sleep improved by about one hour per night and he went from no pain free days per month to approximately 20 pain free days per month. Note consolidation of gains by month 3 of program.

In summary, braindriving has been found to be an extremely effective method for increasing the efficiency of neurotherapy. Combined with the very rapid and efficient ClinicalQ intake procedure, neurotherapy can be a remarkably cost effective treatment option for a very wide range of disorders. Braindriving is simply applied
learning theory in which stimuli with known and measurable effects on the central nervous system are made contingent upon a response, following a classical conditioning paradigm.

This classical conditioning protocol can be combined with the operant conditioning properties of neurofeedback. One nice feature of the Braindryvr Cascade is that it can also be used to reinforce an operant, in addition to presenting stimuli in classical conditioning format. For example, the instrument can be programmed so that if the child produces an SMR response every few seconds, an electric train can be kept moving for a few seconds even though the SMR response is a brief operant. In addition, a second channel can deliver a second unconditioned stimulus such as 13 Hz light with look-through eyeglass frames or an SMR-enhancing sound, contingent on SMR amplitude dropping below training threshold. In essence, one is using a neurofeedback (instrumental conditioning) protocol that is being augmented with braindriving (classical conditioning methodology). In my opinion, the combination of volitional and non-volitional procedures is going to dramatically accelerate the development of neurotherapy as a primary treatment option for many disorders.

About the Author

Dr. Swingle was professor of psychology at the University of Ottawa from 1972 to 1997, prior to moving to Vancouver. He was lecturer in psychiatry at Harvard Medical School from 1991 to 1998, and during the same time period was associate attending psychologist at McLean Hospital (Boston,) where he also was head of the clinical psychophysiology service. Professor Swingle was clinical supervisor at the University of Ottawa from 1987 to 1997 and was chairman of the faculty of child psychology from 1972 to 1977. Dr. Swingle is a registered psychologist in British Columbia and is board certified in biofeedback and neurotherapy.

References


This paper examines the theory and use of four non-drug, non-invasive modalities; Phototherapy, the LENS, SCENAR/Cosmodic, and NeuroField, for various symptoms and two case studies involving mild to moderate and severe traumatic brain injury.

The four modalities and their conceptual basis for use are:
1. Phototherapy: To increase overall cellular energy and efficiency and speed up rate of cellular healing.
2. LENS: To optimize brain functioning and initiate changes in the cortex that flow to the body.
3. SCENAR/Cosmodic: To reduce pain and inflammation, increase mobility, and boost the body’s innate immune system.
4. NeuroField: To break up cortical phase lock and encourage shifts toward beneficial frequencies.

Phototherapy

Light may be produced in two ways; as coherent light (such as that emitted from LASERS) where the light’s diameter remains essentially constant with distance, or, as Incoherent light (such as that emitted from LEDs) that spreads out with distance from the source.

While light therapy is shown to increase the healing rate of wounds (Whelan, 2001), there isn’t any definitive proof that healing rates of one particular type of light, coherent or incoherent, is best. There are however, attributes of light that affect healing efficiency, associated to the total amount of energy (dosage) and/or the wavelength(s) used during a session.

The most efficient total exposure or dosage of light may relate to a biphasic quality. This biphasic nature is observed as the therapeutic benefit peaks, then decreases with continued exposure time, increases again, then continues to decrease, and can go negative with prolonged exposure time. (Hamblin, 2009). This dose response relates to the total power given to the treated area. (Figure 1).
Red wavelengths are used for issues located near the surface of the skin and approach optimal doses in the region of 4 J/cm² with a range of 1–10 J/cm². Doses in the near infrared wavelength region used for deeper disorders, can be higher, in the 10–50 J/cm² range. Phototherapy is usually repeated either every day or every other day, with a course of treatment of around two weeks. (Hamblin, 2006).

Prime wavelength ranges for cellular repair were found to depend on an “optical window” property. If one looks at all the available wavelengths, research showed that certain ranges penetrate deeper into cellular tissues than others. This optical window range is limited primarily by absorption, due to either blood, at short wavelengths, or water, at long wavelengths. Further, the first law of photobiology states that for low-power visible light to have any effect on a living biological system, the photons must be absorbed by electronic absorption bands belonging to some molecular photoacceptors, or chromophores (Sutherland, 2002). This makes proper wavelength a key consideration. Wavelengths of 810, 630, 660, 780, 904 and 940 nm have been routinely used in phototherapy.

Once light reaches the cell, it can interact with mitochondria, tiny structures inside each cell which represent our body’s “cellular power plant” because of a process called oxidative phosphorylation that occurs within the mitochondrial respiratory chain. This process converts food into energy in the form of, adenosinetriphosphate (ATP), supplying the body with energy it needs to thrive (Karu, 1989). Light may also be involved with Cytochrome c oxidase (COX) and nitric oxide (NO) release. Nitric oxide produced in the mitochondria can inhibit respiration by binding to COX and competitively displacing oxygen (Brown, 2001), and work by photodissociating NO from COX, thereby reversing the mitochondrial inhibition of respiration due to excess-
sive NO binding. (Lane, 2006).

Light reacts with tissues and mucous membranes located close to the skin’s surface, and with immune cells moving through superficially-located capillaries (Martin, Leibovich, 2005). Light directly affects the secretion of soluble protein mediators (SPMs) by these cells and helps the resolution of inflammation and repair if inflammation is delayed (Dyson, Young, 1986), (Dyson, 2007). “Deeper cells can be affected indirectly by SPMs released from peripherally-located cells that have absorbed photons, so that Phototherapy has both local and systemic effects.” (Dyson, 2009).

Phototherapy’s local and global effects make it a reasonable starting point for any given session, before the other modalities are implemented as it may increase the available healing energetics for use by subsequent modalities.

**The LENS (Low Energy Neurofeedback System)**
The LENS was developed in 1990 by Dr. Len Ochs and tends to increase self-regulation for several issues such as ADD/ADHD, anxiety, depression, migraines, and especially, mild to moderate traumatic brain injury. Mild-to-moderate traumatic brain injury responds especially well to LENS therapy. (Hammond, 2007) (Hammond, 2010).

The LENS appears to increase clarity and ease of functioning, as well as reduce the amplitude and variability (including spiking) of the EEG activity across the 1—0 Hz spectrum. The LENS can encourage increases in amplitude and variability of the EEG when there is too little variability. This process may result in an initial unmasking of the full extent of hidden EEG pathology; over the course of continued LENS training, amplitude and variability may again diminish as functioning improves. (Ochs, 2006). The signs of physical relaxation that typically occur during a LENS session may be accompanied by warming due to the parasympathetic shift and increased vasodilation.

Similarities exist between the LENS and conventional biofeedback/ neurofeedback systems in that the same amplifiers and 10–20 sites may be used. However, in contrast to methods training just a few sites, LENS protocols use most of the cranial surface, and may involve systematically training up to 21 sites, depending on the protocols.

The major differences between the LENS and conventional neurofeedback relate to the LENS construct of a faint, very low energy feedback signal that is based upon which dominant frequency patterns the brain is producing at that moment in time. This ever-varying signal is sent over the sensor wires back to the brain to gently disrupt dysfunctional patterns. The feedback signal changes as the brain changes, and discourages accommodation. Another difference is the active session time; conventional brain training requires a person be attached to the sensors for 40 to 60 minutes per session, whereas the active time for a LENS session may last only a few seconds.
or minutes.

The LENS’ disentrainment approach may be the reason the number of sessions required to get a sustainable positive outcome seems to be much less for LENS compared to conventional methods for many symptoms.

In a six-patient case study, Dr. Hammond presented data (Figure 2) at an ISNR annual conference, indicating that the average number of sessions to obtain a positive, lasting result with OCD was 32.3 for the LENS compared to 71.7 sessions with conventional neurofeedback. (Hammond, 2010).

Dr. Nicholas Dogris performed a small LENS study of 10 children with ADHD. Prior to the study, the patients were taken off all medications. After 15 sessions the improvements were measured. The effects of the therapy were sustained, so that subsequent sessions were not required. (Figure 3). (Dogris, 2008).

NeuroField, invented by Nicolas Dogris, PhD, delivers specific frequencies to the
client via a pulsed electromagnetic field. This pulsed field interacts with the person’s electromagnetic field and tissues. Applications for NeuroField written by this author were used to support stroke recovery, reduce inflammation, and encourage autonomic nervous system improvements. The LENS and NeuroField’s Dehabituation application can be combined in an attempt to break up phase lock so that the body’s system might be more open to change from other modalities.

Sara Harper and Jill O’Brien reported that symptoms were significantly reduced or eliminated within 1–6 sessions when a group of treatment-resistant depression patients received a combination of 2-channel LENS and NeuroField (utilizing the De-hab procedure) (Harper and O’Brien 2011). Ochs has reported significant resolution of symptoms of mild to traumatic brain injury in as few as 1 to 5 LENS sessions. (Ochs, 2008).

The case of a female athlete and “straight A” student is illustrated in this 1 to 5 session range for recovery from post-traumatic headache triggered by a sports concussion. During a soccer match the student experienced a head-on collision with another player. The impact occurred on the right side of her head, she blacked out, fell to the ground, hit her right temporal region and later experienced severe migraines while studying. She stopped attending school due to migraines and came in for therapy two weeks later.

Her LENS map (figure 4) shows all twenty one of the LENS 10–20 EEG sites. The bar graphs represent individual sites that relate topographic maps to their left. Clinicians use this information in subsequent sessions to help select the best electrode placement of the scalp’s sensor sites.

The therapy plan in this case consisted of using a Low Level LASER to increase overall energy and promote healing, followed by the LENS to restore optimal cortical functioning. The total course of therapy was completed in three sessions performed within one week. Her migraines resolved and she returned to classes without any further intervention.

The LENS application was selected based upon her sensitivity, reactivity and resilience (hardiness). These three characteristics, and reactions to the last session, assist
the clinician in selecting which LENS applications best fit subsequent sessions and how many sites to use during those sessions. In this case the application lasted 3 seconds per site. Her total exposure to LENS therapy was 63 seconds (3 seconds/site x 21 sites = 63 seconds) spread out over the three sessions.

The LENS has also been used for more severe neurological insult, which generally requires more sessions to affect a sustainable positive outcome, as in the following case of a sixty-five-year-old medical doctor.

This individual suffered a severe stroke that paralyzed his entire right side, resulted in extreme cognitive impairment, muscle cramping, inflammation, and severe slurring of speech. Therapy began four months post-stroke. Initially, he was lying in bed at a nursing home and his family was told not to expect much improvement going forward. Each session began with Phototherapy followed by LENS and SCENAR/Cosmodic. NeuroField was added after the third session.

The therapeutic summary notes are included to illustrate the rate of symptom improvement.

**KEY DATES in 65YOM MD Stroke Case:**

7/23 Stroke  
11/11 Initial Therapy Session (4 months Post-Stroke)  
November 11: Session #1  
Client is supine in bed during entire session, eyes slightly dull, no motion of right hand, arm, foot (except slight plantar flexion right foot ~ 4°. Stuttering is very noticeable, with word finding & speech lags, very groggy, forced focus.  
November 16: Session #3  
Client is sitting up in wheelchair. “It is much easier to focus. Speech pathologist said I’m doing much better, I wake up at 7:30 and I’m ready.” More energy and better sleep.  
December 3: Session #7  
“I’m constantly getting better, it seems.” Movements are more fluid; self-adjusting in the wheelchair.  
January 4: Session #12  
Things are improving, K (therapist) thought there was substantial progress cognitively over the last two months, Therapist, “We did not expect that.”  
January 11: Session #13 (6 Months Post Stroke, 2 months since beginning therapy)  
Card table is full of research papers; Doc is working on presentations at the computer with reference journals. He is explaining inflammation mechanisms and release of mediators like serotonin from mast cells. Doc is back (cognitively).
January 25: Session #15
Right arm is very mobile. Doc can lift it alone well off the table and move it sideways and backward. Large progress. He is correcting the mispronounced technical names on the medical exam quiz for the therapist said he had scored 90% on the last 2 exams, amazing progress.

February 15: Session #17
Still continues to improve. He wheeled himself into the room, now standing for 10–15 min/day. Arm lift and control good. Minor ability to move right thumb laterally and grasps; so some radial functioning returning. Can (partially) control individual fingers—flexion; weak radial, medial and ulnar functioning.

April 5: Session #23
Now standing 20 min, working on presentations and making plans to leave nursing home. Hand operation continues to improve; independent finger control improving. Also, better control of arm lifting/abduction and adduction. Mentally very clear, no signs of slowed speech or pauses/compensations.

Positive changes in cognitive abilities occurred rapidly. By the third session, using LENS and LASER only, the ability to answer simple cognitive test questions had returned (Figure 5). Before therapy he could not answer even simple test questions with the aid of the staff therapist. Fine motor skills, as illustrated by handwriting with the non-dominant hand, were very poor initially. After the tenth session handwriting quality has improved dramatically.

His first LENS map (figure 6), taken during the first two sessions of therapy, showed significant slow wave activity and suppressed (low) standard deviations (very little blue bar height). The second map (figure 7), four months later, had increased standard deviation, less concentrated activity (bulls-eye patterns) with reduced slow wave amplitudes. The increased standard deviations may indicate more flexibility by the cortex relative to operating frequencies and/or amplitudes. This could be viewed as the lifting of suppression which often accompanies improvements in patient functionality.
SCENAR/Cosmodic—a noninvasive neurostimulation technology.

SCENAR (Self-Controlled Energo-Neuro-Adaptive Regulation) devices and treatment protocols were invented and designed by Alexander Karasjev and A. Revenko. SCENAR works by making the body’s own immune system operate more efficiently and has refined electrical impulses to make the signal similar to that carried in a body’s nerve tissue after distortion caused by the skin, fat, etc. (Karasjev, 1999).

Research indicates that SCENAR serves as a generator of regulatory peptides and works through electrical contact to the skin. The effect of excitement of nervous tissue depends on the type of nerve fibers being targeted. SCENAR generates high-amplitude current pulse. Accordingly, the possibility of excitement of thin fibers, including C-fibers, is considerably higher than at other methods of electro-treatment. (Grinberg, 1998).

The precise biochemical mechanism of the action for the SCENAR is not yet known, although animal experiments have suggested that stimulation releases endogenous opioids. (Sluka, (1999), (Ainsworth, et al., 2006).
In 2007, Dr. Yuri Gorfinkle, MD, analyzed published papers of SCENAR results using SCENAR for certain diseases of the respiratory, urogenital, musculoskeletal, circulatory systems and the ears. Of 17,309 cases that were analyzed, the percent reporting complete symptom remission was 84.91% and the percent improved was 94.75%. The average number of treatments was 12 with a total exposure time of 4.06 hours of SCENAR therapy. (Gorfinkle, 2007).

Minenko’s study of 1,128 patients showed reduced treatment time, increased remission, reduced drug doses, decreased complications, rapid pain relief and other positive changes. (Minenko, Voronkov, 2005).

Researchers at Dallas Presbyterian Hospital evaluated SCENAR as an adjunct in pain control for patients following total knee and total hip arthroplasty (figures 9 and 10). The SCENAR group treated for 20 minutes prior to physical therapy reduced pain medication usage overall and completed rehabilitation therapy earlier than the control group. (Pedadda, Maale, 2010)

SCENAR therapy initiated 24 hours after surgery for broken trochanter repair reduced pain and improved range of motion when used for 20 to 30 minutes prior to physical therapy, compared to the control group treated with a sham device. (Gordonichenko, Gorodetskyi, 2007).

Figures 11 and 12 show results for SCENAR therapy used for post-operative broken trochanter repair. Vertical bars represent the range. The mean value of the score before treatment is represented by a circle and after treatment by a square.
SCENAR has also been shown to reduce pain and inflammation in an edema study. The SCENAR group reported an overall reduction in edema of 54.6% compared to 22.2% for the sham (control) group over the 10 day period. (Gorodetskyi, Gorodnichenko, 2010)

The 65-year-old physician in the previously cited case study attributed his remarkable physical gains and absence of plateau effects in his motor recovery trajectory to the use of the SCENAR/Cosmodic (LET Medical 735AG/Slider), complementing the impressive impact of his LENS therapy on language and cognitive skills. Throughout his therapy the Photonic Stimulator (Ochs Labs) and SCENAR had a very positive effect on inflammation reduction, muscle cramping release, pain reduction, and increases in mobility. Still, the return of motor nerves remained incomplete although improvements continued.

![Figure 13: Pain reduction, ankle fracture](image1)

![Figure 14: Edema reduction, ankle fracture](image2)

**About the Author**

**Larry Michael Beasley, MS, NCTMB, LMT**  
MS Chemistry, Western Kentucky University 1979  
Mike began his career in semiconductor research with General Electric. Over 25 years he developed next generation tools and processes for companies worldwide. In 2001, a stranger’s violent attack on his daughter refocused his efforts to research chronic pain, PTSD, CRPS/RSD and biofilms. Since 2003 he has worked with non-drug, non-invasive modalities and designed protocols to help mitigate the negative symptoms associated to his daughter’s injuries, her 55 surgeries, and those of his clients in Austin, Texas. Mike has developed protocols for the LENS, NeuroField, Photon Stimulator, LASER and SCENAR/Cosmodic. He also instructs in the use of multimodalities.

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*References continued on page 31*
The purpose of this paper is to update people’s understanding about the Low Energy Neurofeedback System (the LENS), how its users are employing it, and how the system is constituted today. This paper starts with a discussion of some general issues and then turns to a technical analysis of some of the data collected from some of the recent LENS sessions.

Discussion of General Issues:
The Low Energy Neurofeedback System is a system that has as its outcome non-deliberate but still active self-regulation to improve neuropsychological functioning and energy. The word “active” is used to contrast the LENS with systems that merely stimulate the brain by exposing the brain to frequencies recordable from that brain that are either too low or too high in amplitude. The LENS is directed at the software of the brain that controls brain’s ability to self-regulate and adapt to new situations. (Ochs, 2006) The activeness of the brain is imagined as twofold: Its initial response to specific proprietary, patented qualities of the feedback in the LENS signal as letting go of its defenses against real or anticipated seizure activity, defenses that may disconnect one part of the brain from another, which cuts its connectivity to prevent kindling, and a process of re-connecting its parts to again allow connectivity and higher functioning. The explication of these two processes is based on 25 years of clinical observation of the wide variety of responses that accompany disruptions of largely unnecessary defensive disabling of the brain’s connectivity within itself, and the observation of the brain’s self-establishment of its connectivity without continued exposure to either the LENS or to operant conditioning. The most interesting aspect of allowing the release of defenses constraining kindling is that both seizure activity remains without the consequence of seizure (even though it is plainly present in the raw EEG), and neuropsychological functioning is plainly and markedly improved (possibly because the person’s energy can be directed toward connectivity and functioning, rather than toward the cutting of connectivity to constrain kindling).

It appears that the LENS is a neurofeedback system, and not a neuromodulation system because the Low Energy Neurofeedback (LENS) software has been implemented on four different standard neurofeedback EEG amplifiers from four different manufacturers without modifying them to produce stimulation, and just by using the OchsLabs, Inc. patented software algorithms for running the LENS on them.
(none of which call for the production of a stimulus). And because the LENS returns a signal to the individual that is based on the EEG that is detected by the amplifier, I again would propose that the LENS is a neurofeedback system. But let’s take a look at the objections of seeing the LENS as a neurofeedback system:

1. The LENS doesn’t involve conscious self-regulation. This criticism couldn’t be truer. Most, however, of my own self-regulation is non-conscious. If I can use the LENS to improve my non-conscious self-regulation then I can use my consciousness to better enjoy my life without having to consciously micromanage my life. So while this criticism appears true, it also appears irrelevant and spurious.

2. The next criticism is the LENS is supposed to involve putting something foreign into the client’s body—maybe something such as “low energy.” Actually, all four of the standard EEG amplifiers on which the LENS has been implemented were able to show a LENS effect, permitting ultra-short exposures to feedback to both modify the EEG and improve functioning. There were no differences between the EEG amplifiers we have used to act as a LENS EEG device and the others used in the field. On this basis it appears untrue that the LENS operates differently from any other neurofeedback EEG amplifier tested, on the issue of producing any kind of penetrating stimulus.

3. And what about the putative lack of self-regulation in the LENS? It seems clear that the LENS leaves people with greater self-regulatory and adaptive capabilities than they had before the LENS. In fact, the ostensive self-regulatory process attributed to conventional neurofeedback appears to be present because of operant conditioning that is largely controlled by the therapist’s adjustments of the learning inhibits and reinforcements, rather than any inherently self-regulatory process of the client. There remains to be studied potentially unrecognized, possibly politically and economically unacceptable, and untested emissions from conventional neurofeedback EEG amplifiers which were discovered by Lawrence Livermore National Laboratories in 2001.

Related to the above objections, is the idea that something must be travelling up the electrode wires, and it must be electrical stimulation. Actually, the LENS has been studied by electrical engineers, biophysicists, and physicists: any stimulation present has also been of the same kind and quality found in other conventional EEG systems.
Effectively, this means that there is no stimulation in the LENS.

Another criticism of the LENS is that exposure to the LENS feedback is too brief to constitute feedback. We have probably not discovered all that there is to know about feedback. The LENS feedback is delivered to the skin of the head, and hypothetically decoded in ways we don’t understand by the brain. The world is replete with experts saying incorrectly, “That can’t happen” (O’Boyle, J. 1999, Wells, J. F. 1988).

On behalf of seeing the LENS as a neurofeedback process:
Those who would specify that the LENS must be a neuromodulation system need to specify some source of alleged stimulation that is different from anything that would emanate from the EEG amplifier of their choice, as well as to account how non-LENS EEG amplifiers, when running the patented LENS software, could also produce a LENS-like effect using very brief connect times and non-linear reasonably rapid response, even after one second of exposure in some instances. The brief exposure can make a difference in an EEG output record; and it certainly can make a difference in a person’s life, even though not all the time, or perfectly.

The LENS approach employs an EEG amplifier that processes data the same way that any other EEG device processes data in the neurofeedback field.

All feedback and other kinds of information travel on some kind of carrier wave. According to Kuttner, F. & Rosenblum, B., when the feedback is visual, the visual feedback travels on packets of energy called photon particles (2011), which are in fact, electrons, and which may have the properties of both particles and waves. In the LENS the electrons traveling in the wires that convey EEG from the head also carry feedback signals back to the head, and generate a small electromagnetic field as they do. Beverly Rubik, PhD, found that the actual voltage traveling in the electrode leads is approximately one million times weaker than the voltage found in the Alpha Stim device (personal communication March, 2008); J. Hoover indicated that the actual current traveling in the electrode leads was measured at .001 micro Amperes, and if made less, the information in the signals would have disappeared in the thermal noise generated by the electrons travelling in the wires (personal communication, 1998). The amount of voltage in the electrode leads is essentially the same as that found in the leads of other EEG systems used in the neurofeedback field. C. Snook has stated that he detected no stimulation pulses in the LENS (personal communication, May 7, 2007).

The duration of exposure to the LENS in any session, also known as “connect time” to the EEG amplifier feedback signals, can range from 1 second to several minutes, and is strongly correlated in different ways with assessments of client sensitivity (in this sense perceptiveness), reactivity (often in our culture seen as sensitivity), and
resilience. Each of these concepts bears a different influence on how long a client is connected to the electrodes of the LENS in any session. A much more in-depth treatment of this topic can be found in Journal of Neurotherapy (Ochs, 2006).

There is a principle in the way the “strength” of the LENS feedback is presented to the client: To achieve something akin to “strength,” the potentially complex treatment settings are matched to the sensitivity, reactivity, and the resilience of the individual. Toward this end the LENS provider has a variety of questionnaires assessing these qualities of the client, and matches the way that the LENS feedback is constructed in its smoothness, brevity, repetition, as well as in other ways, with the physical properties of the information, much like a poet chooses his or her words and captures the attention of reader or listener. The principle might be termed “resonance” that becomes established while matching the feedback to the client, and crafting of the feedback so that minimal feedback may be used for those whose are delicate in consciousness. This apparently weak feedback acts to shape a more effective outcome, because it derives its power from matching client traits that leads to resonance.

In contrast to the way resonance is achieved in the LENS, resonance is also achieved through the use of strong electromagnetic fields as a neurostimulation process. There are stronger neurostimulation processes that achieve their resonance by shaking the molecules of the central nervous system of the client. The LENS provider achieves resonance by using inconceivably “weaker” information to establish and maintain a resonance that tugs on the physiology just enough to interrupt the processes that perpetuate dysfunctions of various kinds, and in a way that supports connectivity of the brain with itself that frees the individual from the disconnection that leads to dysfunction. The same brief process appears to interrupt chronic pain that might otherwise last a lifetime.

The goal of the just-enough-feedback that matches sensitivity is to loosen the individual from chronic dysfunction without leaving the person with yet one more thing from which he or she also must recover. For instance, the typical twenty minutes of other kinds of feedback can leave an individual with a variety of problems. (Hammond, 2008:1)

**Data from LENS Sessions:**

For those clients (n=836) who are 60 days past their last treatment session for treatment started in August of 2013, the duration of the series of sessions averages seven sessions across all diagnoses, with the following standard deviation for the average stated above (Figure 1):

**Figure 1**

For new clients (n=158) within 60 days of starting treatment, the duration of the series of sessions averages five sessions, with the following standard deviation for the average stated above (Figure 2):

**Figure 2**
66.667% the individuals 13 sessions or fewer.
95% of the individuals 19 sessions or fewer.
99.5% of the individuals 25 sessions or fewer.

For those new clients (n=158 who are within 60 days of having started their treatment) using the software within the past several months the duration of the series of sessions averages five sessions across all diagnoses, with the following standard deviations for the 5-session average (Figure 2):

- 66.667% of the individuals received 8 sessions or fewer,
- 95% of the individuals received 11 sessions or fewer,
- 99.5% of the individuals received 14 sessions or fewer.

The changes in the EEG are shown as follows for the connect times and the duration of treatment as stated above.

These changes in measured EEG variables are derived in the following way: First, we have found no consistent relationship between sensor site and increases in functioning. I understand that this observation violates nearly all theory that correlates neuropsychological improvements with feedback to neurological clusters of cells responsible for certain kinds of functioning. Furthermore, we have observed desired changes in neuropsychological functioning when we specifically have placed sensors on the scalp in areas not responsible for those improvements in functioning. We have, however, found consistent changes in the EEG by placing the LENS electrodes (identical to the electrodes used in traditional neurofeedback) using a reproducible organized systematic approach (described in Ochs, 2006) to placing the electrodes during LENS neurofeedback treatment.

It is my impression, without any data at this time, on the basis of having looked at a lot of LENS data, and with some notable exceptions, that LENS treatment times lengthen when the above systematic way of choosing sensor sites is not employed (when sensor sites are chosen on their basis of either statistical deviation from norms or on the basis of the historically relevant cluster of neurons regulating functions). I would expect it to be easy to assess the efficacy of using the OchsLabs, Inc. mapping process because instead of using this process for sensor-site selection, those trained in traditional neurofeedback tend to gravitate toward placing sensors on the basis of EEG values deviating from norms or on the basis of the neurological sites supportive of targeted neuropsychological functions.

To decide upon which sites to place electrodes in this study, we used the Total EEG
amplitude (3–42 Hz) anchored at the site with the highest amplitude (HAS) in the first complete LENS EEG map. We then took the total EEG amplitude at that same site the last time it was treated in a LENS session. I thought it was risky using the HAS-type measurement instead of generally recognized neuropsychological sites, although Larsen did so, as described in his 2006 paper. The risk is that using a HAS-type measurement might lead the reader to invoke the regression-to-the-mean-fallacy to explain the changes. That is, drops in measurements of relatively high amplitude might be thought of as due to regression to a lower amplitude that would have happened anyway and not correlated with the exposure to the LENS. We will not know the answer to this concept until we control for problem intensity in relation to when the LENS was begun, which we will do in the future.

We also measured the site with the lowest amplitude (LAS) in the first complete LENS EEG map, and then took the total EEG amplitude at that same LAS site the last time it was treated in a LENS session.

The HAS and LAS were specific to each client. For one client the HAS might be F3, and for another it might be T6. All the values for the HAS for each client were averaged across all clients. The average HAS value at the start of the LENS treatment is compared with averaged HAS value the last time that site was treated, regardless of which one it is, using a repeated-measures t-test. The same process was applied to the LAS.

Because clinical observation led us to the impression that with the LENS, the amplitudes, dominant frequencies, and coefficients of variability of high amplitude sites decrease, while the low amplitudes, dominant frequencies, and coefficients of variability increase; it is possible to use a specific form of t-test called a one-tailed t-test, and predict that not only the size of the change but the direction of the change, which we did. We predicted that the high values would decrease, and the low values would increase.

One of the severest criticisms that has been leveled at the LENS is that there has never been any demonstration that it could change the EEG. And even if the LENS just triggers a regression to the mean this finding will be significant for the small amount of time in which the LENS can do this (Figure 3, next page):

All changes in EEG activity are significant at beyond the 0.0005 level of probability. The top-most two graphs show total amplitude changes. The left upper two graphs show respectively the HAS and the LAS changes. The upper two graphs on the right show variability changes in the HAS and LAS sites. The bottom two graphs show
dominant frequency changes for HAS and LAS on the left, and similar changes for variability. All graphs show a “certainty” that the EEG changes when used as trained, rather than just a “probability”.

The Wilcoxon signed-ranks test is a non-parametric version of the repeated-measures t-test which allows violation of untested assumptions. The probabilities from the Wilcoxon are even smaller than they were from the t-tests, raising the significance of the changes to an even higher degree. In short, the LENS does change the EEG.

The LENS as a System

The central idea driving the LENS is that it takes less time for feedback information to reset the timing of the neurology that perpetuates dysfunction than it does to operantly condition a different neurology. Both lead to the same place: the reduction in symptoms with the EEG changes. This writer looks forward to testing this hypothesis in future research.

The additional advantages of the LENS are that its non-dependence on cognition makes it a better tool for people who have cognitive impairments; it’s brevity of attached electrodes makes it a better tool for those who cannot sit still. Finally, its rela-
tive artifact immunity makes it ideal for those with severe problems sitting and attending, and allow even for children to receive neurofeedback that is effective while sitting screaming in the lap of a parent.

And nothing says “system” like being able have an experienced clinician remotely review clinical history, both EEG and symptom change data from sessions, and being able to construct a set of treatment parameters remotely and to send them to the LENS provider for immediate use. The LENS allows high degrees of quality control with the remote review of completely anonymized data, and the ability to supply custom settings files to the providers that have worked outstandingly. In this way, decades of experience can be offered in concrete ways to providers who lack experience, adding capability to new LENS providers and added benefit to the client.

We will be testing this new system until we are satisfied that we have improved it so that it works even better than it works now. However, even now, before we have a great deal of data on reductions in symptom ratings that allow us to do qualitative analyses of the symptom ratings we do have, look at them against self-assessments of sensitivity, reactivity, and resilience, and check what happens to the symptom ratings as the LENS sessions accumulate.

**Summary:** The LENS appears to its users as a neurofeedback system that in one form or another has been in use since the year 2001. It appears to those who use it as reasonably effective in unusually short times. The observed changes in the EEG are statistically significant and reproducible.

**References**


ISNR Research Foundation Update
Tato Sokhadze PhD, President, ISNR Research Foundation

This update on our Foundation research supporting activity focuses on an ISNR conference held October 14–19, 2014 at Wyndham San Diego Bayside Hotel. It was a great pleasure to meet in person many of our supporters and donors at our exhibition booth and at other events. We would like to let you know that we really appreciate your continuous support of the Foundation and we value it, as without this we’ll not be able to move forward.

Among several events arranged by the Research Foundation, our annual silent auction was one of the most important, as it helped us to raise over $7,600. We are grateful to our auction item donors (Applied Neuroscience, CNS Vital Signs, Thought Technology, BrainMaster, SoundHealth Products, ElectroCap, Photosonix, AAPB, Peak Achievement Trainer, Paul Swingle, Ed Jacobs, David Trudeau, and many others) and auction bidders for this successful fundraising campaign participation.

Another important event that we had held in San Diego was our Second Annual “Friendraising” Dinner at spectacular “The Start of the Sea” restaurant. Our speakers at the dinner were Roger DeBeus (University of North Carolina, Ashville) who described the stages of the preparation to the NIMH-funded study on neurofeedback application in ADHD treatment, and the role of the CNP group, supported by the ISNR Research Foundation. He did the presentation at the ISNR conference plenary too, but this talk focused mostly on the story of months of the CNP group efforts that finally resulted in federal funding. Another speaker at our dinner was Samuel Turcotte, President of Zukor Interactive, our long-standing friend and supporter, who told us the fascinating history of how he got into development of his neurofeedback games and other media for neurotherapy. Tom Collura announced that the winner of BrainMaster’s annual award for “First Person Science” was Patricia Norris and introduced her achievements in this area. David Kaiser told us the history of his collaboration with Barry Sterman (who could not attend, due to family circumstances) and also the story about the Claude Bernard Club dinner that used to be held during AAPB meetings. We were very eager to revive the tradition of this club in the future and to have it during ISNR meetings. We enjoyed the company of
our past mini-grant winner students, Sommer Christie from the University of Calgary (Canada) and Matthew Goodman from Alliant International University (San Diego), and their brief progress reports. Matthew was the winner of the joint FERB-ISNR Research Foundation grant that incorporated both biofeedback and neurofeedback approaches, and he also gave his presentation at the conference session.

During the banquet ceremony we announced our current mini-grant program winner. The Jay Gunkelman award, sponsored by BSI for qEEG-based studies, went to a group headed by Markus Rogan (Antioch University, Los Angeles, Masters student in clinical psychology, mentored by Drs. Leslie Sherlin and Michelle Craske) for the project titled “Exploring the effects of neurofeedback and mindfulness training on cognitive and vestibular functioning in elite athletes.” Max Sutton-Smolin, one of his key collaborators, accepted the award on behalf of Markus, who could not attend the meeting.

Our small group discussion during the conference mostly centered around describing the main direction of the Foundation and answering the question of what should be done to promote more research activity in our field. On this cycle of mini-grants we have RFA soliciting proposals for the joint FERB-ISNR-RF award, and the winner will be announced in early March, 2015 at the AAPB meeting in Austin, TX. This award was supported by the NEWMind Neurofeedback Center.

Our regular cycle of mini-grant awards will be announced in February 2015, with a deadline for grant submission in June. Award winners for four mini-grants (BSI Jay Gunkelman award for qEEG & neurofeedback, Zukor’s Peak Performance mini-grant, BrainTrain’s ADHD mini-grant for PhD students, and an ISNR RF mini-grant) will be announced by the ISNR conference in Denver, CO, October 15-18, 2015. We strongly encourage your students and young investigators to apply for these grants. One more time, we value your support and really appreciate your contributions to the ISNR Research Foundation. Please continue to support our efforts.
What Has BCIA Been Up To?
Judy Crawford, Executive Director, BCIA

This has been a very busy year for BCIA. Since April 1, 2014, the BCIA Board of Directors has taken charge of all of their own business affairs with Judy Crawford as the Executive Director in charge of the day-to-day business. They are no longer using an association management company as they had since 1981. The new office address is at 5310 Ward Rd, #201, Arvada CO 80002. The website is still the same, at www.bcia.org and we hope that you will check there to find the answers to most of your certification questions, and as well to stay abreast of current news items from our industry, including continuing educational offerings.

BCIA has been offering affordable high-quality webinars since 2012. These started with 90-minute online courses in our Clinical Update Series. In answer to many queries about how to find access to a wider and more affordable group of mentors, BCIA introduced a new type of webinar that is only 60 minutes long and provides the attendee with one contact hour to cover two case conference presentations. Mentoring webinars are appropriate for certification and recertification alike. All webinars are recorded and made available to those who couldn’t attend the original live telecast. The response has been very positive, and as always, it was a good sign to see attendees from countries including Spain, South Africa, Australia, England, and New Zealand, to name a few. If you have topics or speakers to suggest, please let us know. Stay tuned for the 2015 schedule.

Internationally, the word is spreading, as evidenced by the following developments. The Japan Clinical Neurofeedback Association was registered in Tokyo on September 25. The main aims of the organization are to support and promote the efficacy of neurofeedback in Japan, provide information to the public, hold seminars and case conferences for therapists, and to set guidelines of practice based on standards that would be equivalent to BCIA.

A few BCIA certificants in Canada petitioned the Canadian Psychological Association (CPA) to form their own section entitled “Quantitative Electrophysiology.” Thank you to Drs. Atholl Malcolm and John Davis, both BCIA certified in neurofeedback, for their hard work. This new group has already been approached by others within the CPA to cooperate with the Sports and Exercise Section with joint future presentations, since neurofeedback is already being used by many prominent Canadian athletes.

In the near future, BCIA hopes to be able to report progress working with a new group south of the border. We expect 2015 to be a very exciting year for the field and BCIA is proud to be a part of it.