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Dear Readers,

Welcome to the winter edition of NeuroConnections.

This edition has several articles that could help to answer questions of the value of Neurofeedback and biofeedback. If you are similar to me, eventually the two labels get thrown into being either one or the other. Those puzzled looks on client’s faces certainly can cause one to quickly find a common ground for what to call what we are doing. As the neurofeedback field continues to bring in more and more biofeedback artillery the need to just simplify and use the term biofeedback becomes more attractive. I remember when in the 1980s I first started with biofeedback the explanation was so simple. Barbara Brown in her book said bio = biological and feedback = to the client. So the client was receiving feedback from their system which helped them to self-regulate the behavior of the system. Still makes sense as a way to explain what we do.

Tom Collura does a masterful job of discussing the integration of neurofeedback and biofeedback. He looks at the ways in which clients and clinicians can become more aware of their systems and what the instruments are that can be most helpful. Do read, especially, if you have not had a lot of work with biofeedback and are deep in the neurofeedback world. You may find that you will be adding more instruments to your tools and find that they help the clients in many ways.

You will find an excellent article by Arns, van der Bergh and Gunkelman re-

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Letter from AAPB Co-Editor

Welcome to the Winter 2009 edition of NeuroConnections. While outcome studies have typically addressed the efficacy of neurotherapy as a stand-alone intervention, skillful neurotherapy clinicians appreciate the value of a thoughtfully selected companion interventions matched to specific patient needs. In the present issue, our contributors discuss both classical and emerging approaches that can serve as useful adjuncts or alternatives to stand-alone neurotherapy.

Reminding us of our roots in the peripheral biofeedback tradition, pediatric clinician Liz Stroebel returns to present Part 2 of Carly’s Story, highlighting the contribution of classical self regulation skills training in giving children a toolkit to extend mastery over their symptoms outside the clinic, and into their daily lives.

Mark Jensen contrasts neurotherapy with cognitive behavioral and hypnotis-based pain management strategies. Noting the unique contributions of each approach, he argues for a clinical decision tree matching these diverse approaches to individual patient needs.

Cory Hammond presents a compelling case history of successful resolution of intractable pain utilizing an innovative alternative to traditional neurofeedback training, while Nick Dogris, in a companion feature, provides background on the development of this emerging approach.

We enjoyed working with each of our contributors, without whom this issue would not have been possible.

Roger Riss, PhD
AAPB Co-Editor

Letter from ISNR ED

Tom pretty much filled you in with his letter from the president this time. So, there’s not much more to report. We’re still working to develop the Journal of Neurotherapy, having made some editorial changes and are very interested in publishing your research or clinical data. ISNR is also working on a book series in which the first two books will be theoretical and clinical surveys (respectively). Tom Collura is authoring the first book and Randy Lyle will be editor for the second, which will include chapters that are clinical and theoretical in nature and will introduce the full length books that will complete the series. Many of you responded to my email introducing the series and we are now formulating it. We’ll keep you posted.

ISNR is also working on a book series in which the first two books will be theoretical and clinical surveys (respectively).

The Research Foundation is now working on two possible collaborations, the first with the Methodist Rehabilitation Hospital in Jackson, Mississippi for their in- and out-patient populations with traumatic brain injury. The second may be with CHADD in studying neurofeedback with AD/HD and may utilize the consortium to track our progress. Both of these collaborations are in the very early stages. We hope they lead to important contributions to our field and in the areas of brain injury rehabilitation and AD/HD treatment.

This issue comes to you during the festive season. I wish you and your friends and family a healthy and productive 2010. Let’s stay connected.

Cynthia Kerson, PhD, BCIA-EEG
Executive Director, ISNR
It’s not too early to start planning to attend AAPB’s next Annual Conference scheduled for March 24-27, 2010. The planning committee has a number of surprises in store that you won’t want to miss! New this year will be an “Update Series” providing an opportunity for attendees to receive a brief update on trends and material presented last year. In addition, the planning committee has reached out to each of the membership Divisions and Sections who will be sponsoring presentations throughout the conference.

As we travel to San Diego, California for AAPB’s 41st Annual Meeting, we are excited with these new initiatives being taken by the committee under the direction of Chair, Gabriel Tan, PhD. Be sure to make plans to join this gathering of experts in an era of rapid workplace change. Work is particularly timely for clinicians assisting clients to optimize health in an era of rapid workplace change.

V.S. Ramachandran MD, PhD is Director of the Center for Brain and Cognition and Professor with the Psychology Department and Neurosciences Program at the University of California, San Diego, and Adjunct Professor of Biology at the Salk Institute. He is author of over 180 scientific papers. His acclaimed bestselling book “Phantoms in the Brain” formed the basis for a PBS television special. He is best known for his experiments in behavioral neurology which, despite their apparent simplicity, have had a profound impact on the way we think about the brain, and therapeutic techniques to promote cortical reorganization after brain injury.

Norman Shealy, MD, PhD is a world renowned pioneer in chronic pain management, complementary and energy medicine. Dr. Shealy’s work as a neurosurgeon led to the invention of the Dorsal Column Stimulation (DCS) and TENS devices now used worldwide for pain. In 1971, he founded The Shealy Institute, the country’s first comprehensive facility focusing on complementary and alternative methods for pain and stress management. He is founder of the American Holistic Medical Association, and past president of the International Society for the Study of Subtle Energies and Energy Medicine. His work with Caroline Myss led them to found the first doctoral program in Energy Medicine. In 20 years of research into anti-aging, he was the first to demonstrate regrowth of human DNA telomeres, a major key to extending human longevity. Dr. Shealy envisions a day when a lifespan of 140 years of age will be in reach.

Töres Theorell, MD, is Professor Emeritus at the Karolinska Institute in Sweden, as well the Director of the Swedish National Institute for Psychosocial Factors and Health. He has explored the relationship between workplace environment and health risk in more than 400 scientific papers. His work is particularly timely for clini­cians assisting clients to optimize health in an era of rapid workplace change.

We look forward to seeing you in San Diego!

Mark your calendar! Begin making your plans today! Over the years, AAPB’s conferences have been very highly rated for outstanding educational content, presentation of breaking scientific data, and the best networking available in the field. As this group of speakers illustrates, the committee for the 2010 meeting is taking a creative approach in making the 2010 meeting an event that you cannot afford to miss.

We look forward to seeing you in San Diego!

David L. Stumph, IOM, CAE, AAPB Executive Director

Tom Collura, PhD
ISNR President
functional ones (psychology) like the ones my friend revealed with his analysis. In fact when Hans Berger developed the first amplifiers sensitive enough to detect neurol electromagnetism in humans in 1924, he studied his son’s thought processes and behavior first, and only later did he find other uses for this technology such as identifying seizure activity and brain disease. I also explained that since 1965, the year when the fast fourier transform (FFT) was invented (or rediscovered) by Cooley and Tukey, about three times more EEG research has been published under the aegis of Psychology

attention, sleep, unconsciousness, animal behavior

than under the patronage of Neurology. In other words, identifying organic disorders has been a minority application of EEG technology for my entire lifetime.

EEG is a tool and like any tool it may be used in multiple ways by different people. Consider a hammer. Judges use hammers to maintain order and carpenters use them to build houses but we wouldn’t require all hammers to be round at both ends or add a metal claw to a gavel. Same tool, different uses. During most of my examination and cross, I showed a Vend diagram of two overlapping circles, one labeled Psychology, the other Neurology, with their intersection labeled “EEG.” That alone should convey the distinction. Psychology is a science based on inferential statistics and repetition: a phenomenon is best understood when it occurs similarly and repeatedly across individuals and groups and we use EEG technology in a likewise inferential fashion, relying on statistical tests such as M/ANOVAs, t-tests, Bonferroni, and Huynh-Feldt corrections for nonsphericity, to name a few. Neurology is primarily a descriptive science, focused on qualitative techniques of identification such as “eye-balling” a signal to a mental template in one’s head. If a science depends on cross-individual repeat-
There is a growing interest in combining different biofeedback modalities, in particular when EEG is involved. EEG biofeedback (“neurofeedback”) has had a tendency to develop as a separate branch of biofeedback, both rediscovering principles known in traditional biofeedback, while creating its own approaches and paradigms. When combining EEG with traditional (or “peripheral”) biofeedback, attention should be given to the unique capabilities and characteristics of each modality, in an effort to integrate them in a simple, yet effective manner.

Gevirtz (2003) provides an overview and references on HRV, which is both a powerful predictor of cardiac and other health outcomes, as well as a promising mode of biofeedback. He specifically refers to HRV resonance, which occurs when the deviations between high and low heart rate are maximized. This typically occurs at an individual breath rate of about 6 breaths per minute, which the trainee is able to find and maintain as part of the training process.

Other authors refer to HRV coherence, which occurs when the variations in heart rate are maximally sinusoidal, which means that they contain primarily one main frequency, and appear like a smooth wave. Any of a range of strategies are available for training optimal HRV. Fortunately, they are all effective, largely because any approach that restores autonomic homeostatic reflexes must ultimately restore all of the interacting processes that work together towards maximizing the rhythmic fluctuations in a healthy system that is exploring its operational boundaries.

While pursuing the combination of EEG and HRV, there is merit in returning to basics, and providing feedback that is both informative, timely, and aesthetic. When implementing a combined protocol therefore, it made sense to explore simple alpha training, in conjunction with a simple form of HRV training.

The basic design of the alpha enhancement protocol is shown below. In the interest of simplicity, a manual threshold is placed on the magnitude of the alpha wave, as measured with a third order digital filter. The waxing and waning of the alpha is clearly evident. When alpha magnitude exceeds the preset threshold, there is a sound produced. A gentle, midrange flute note is used to indicate the presence of alpha waves.

Alpha waves typically wax and wane continuously, are largest when the eyes are closed, and have their own peculiar properties during feedback training. Alpha waves are maximized when the trainee relaxes, clears the mind, and essentially “gets out of the way.” Alpha waves are reduced when the trainee is anxious, thinking, or attending too intently to the sound feedback. Only by allowing the sounds to come” does alpha training generally allow the trainee to achieve optimal increases, and the associated relaxation and reduction of stress.

In “typical” alpha training, the individual typically has eyes closed, and is passively waiting for alpha waves to occur. Strategies can include adjusting one’s internal state and attitude, letting go of ruminating thoughts, and generally looking to enter a relaxed yet attentive state. When an alpha state is achieved, the individual is generally well tuned into both internal and external events, yet has a sense of emotional well-being, and a nonjudgmental attitude.

The basic design of the Heart Rate training protocol is shown below. The goal of the training is to reveal and allow enhancement of the phenomenon of Respiratory Sinus Arrhythmia (RSA). An individual wears a simple finger sensor that contains a photoelectric pulse oximeter of a standard type and appropriate electronics. The sensor measures the blood flow through the finger, and can be used to find both heart rate (beats per minute) and oxygen saturation (percent). The heart rate and oxygen-
saturation information are converted by the hardware directly into signals that can be used for biofeedback training, simultaneously with up to 4 channels of EEG.

Rather than using metrics such as coherence or resonance, we can simply reward the heart rate variation, when it is in a sustained downward deflection, which is to occur during the exhaling phase. The trainee hears a deeper, flute sound when this occurs. The experience is similar to that from other breathing oriented HRV devices (i.e. Wild Divine). When the trainee is able to perform a relaxing, sustained exhale that “plays” the deep tone, then the heart rate is achieving the modulation that is sought by HRV training generally, including coherence or resonance training. The heart rate trace below shows the rhythmic, sinusoidal changes that are typical of the coherence, resonant heart rate response.

The HRV training protocol produces a repeating, sustained, deep tone during each successful exhale that produces a sustained drop in heart rate, as revealed by the falling phases of the RSA curve. A sustained reward criterion (500 milliseconds) is used to ensure that the trainee receives the tone only for sustained periods of falling Heart Rate. As a learning strategy, the trainee soon realizes that, in order to achieve, the long, sustained exhalations, it is necessary to first take in a large, sustained inhaled breath, so that the lungs are optimally full. Thus, even though the training is essentially only rewarded for exhaling, the trainee must produce a fully robust breathing pattern, in order to achieve sustained rewards.

The combined Alpha and Heart Rate protocol is implemented by combining the above elements, into a single design: Figure 3. Combined alpha enhancement and HRV protocol. Traces, from top to bottom: Raw EEG, Filtered alpha waves, Alpha magnitude, Marker for alpha above threshold (high tone is produced when marker is high), Heart Rate, Marker for Heart Rate Falling (low tone is produced when marker is high), Marker for both conditions true (bell is produced when marker peaks).

It is when both conditions are true that the trainee gets the added feedback of a bell sound, indicating that both conditions have been met for a criterion time. Bells are thus heard only during an exhale, and only when both the deep tone and the high tone are present. It is basically a special reward achieved for performing a particularly good exhale, and then relaxing into it, allowing

Figure 2. Design of simple Heart Rate training protocol. Top trace, Heart Rate (white) and its time average (green). Bottom trace: Marker for Heart Rate falling (HR is below its time average. Both coherence and resonance are visibly evident, as the rising and falling of the heart rate is both maximized, and takes on a clean, sinusoidal shape.

Figure 3
alpha to be produced. It is also possible to easily add visual feedback such as games, videos, or other displays, based upon either or both feedback signals. Figures 4 and 5 show a form of “proportional” feedback, in which a flower cyclically opens and closes, in cadence with the achievement of the HRV changes being reinforced. The combination of a visual display with the feedback sounds is sufficient to introduce the individual to healthy, comprehensive habits of breathing and the associated internal mental states.

The trainee is able to learn a pattern of deep, rhythmic breathing, combined with the internal state associated with an alpha pattern. The subjective experience is rich and complex, despite the simplicity of the design. The breathing pattern is experienced to produce deep, sustained, soothing tones when producing an optimal exhale that is coupled to the RSA. In addition, there are the overtones reminding the trainee that alpha waves are being produced. When the trainee produces a strong, prolonged exhale, and further relaxes the brain, then one or more bell tones are produced. It is the combination of states that produces the bell that makes it a relatively special event, among the ongoing tones that indicate the momentary changes in the state of the brain and body.

So what is produced is, in summary, a simple tone indicating optimal HRV changes, another tone indicating the presence of alpha waves, and a bell that is produced only when both the HRV and the alpha activity are present. The presence of the “total” reward sound therefore becomes a significant event, one that the trainee can achieve through relaxed, yet diligent application of simple and beneficial processes.

It is difficult to say whether this approach adds EEG to HRV, or that it adds HRV to EEG. Each modality expresses itself in its typical fashion, and each component could be effectively trained independently. But this approach seems to fill in some gaps that are left to either method when left on its own. In neurofeedback, it is not uncommon for practitioners to put emphasis on the trainee’s breathing, posture, etc., in order to ensure that they are ready for the learning processes of neurofeedback to occur. When clients are unrelaxed, fidgety, anxious, or physically agitated, it is difficult if not impossible at times, for them to “attend to the screen,” or “allow the sounds...
to come,” no matter how much they try. By providing a task such as HRV, the individual now has additional, positive things to do to move them in the desired direction. Some practitioners precede neurofeedback with time spent on an HRV system, simply to wind them down to be ready for the feedback training.

Similarly, when doing HRV training alone, it is often important to attend to the client’s inner space, coach them with appropriate suggestions, visualizations, or other mental exercises, to further create a comprehensive, relaxed, positive state. By adding alpha feedback, the trainee is further instructed on a beneficial, neutral internal neuronal state, that is complementary to the systemic relaxation and flexibility inherent in HRV training.

By combining the signals, the trainee is provided with more information, in a form that can be integrated, into a more complex and comprehensive feedback task. The complexity now begins to approach more that of a comprehensive, relaxed, positive state. By adding alpha feedback, the trainee is further conditioned to simultaneously clear the mind and achieve an inner balance that complements the body related benefits of the HRV training.

This type of combined training is particularly relevant to the kind of concentration/relaxation cycle described by Sterman and his colleagues (1994, 1995, and 1996). They found that the most effective pilots exhibited an innate control of a cycle of EEG rhythms that alternated between high frequency, low amplitude (“beta” state) and a low frequency, high amplitude (“alpha” state). He further found a bursting alpha phenomenon he called Post Reinforcement Synchronization (PRS) that followed tasks.

In our combined EEG / HRV task, the individual is encouraged to cycle between states of inhalation/concentration, and states of exhalation/relaxation. The protocol is designed to encourage, although not require, alpha to appear preferentially during the relaxation phases of the breathing cycle. Thus, the natural benefits found by exercising the concentration/relaxation cycle are now coupled with the breathing pattern, leading to a more comprehensive body and brain integration of this natural cycling.

References:
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QEEG has been used for the last 30 years and has become more widely available with the increases in processing power of computers which enabled “digital EEG.” Many clinicians nowadays are using QEEG in their practice, mainly for guiding neurofeedback protocols and for some to guide medication prescription.

In our field often findings from published group studies are used to speculate about the best treatment recommendation, such as the theta/beta ratio in ADHD and frontal (alpha) asymmetry in depression. However, this is mere speculation, since there is no theory nor adequate research backing this up for use in individual clients. Furthermore, current use of individualized or personalized QEEG is more technology-driven rather than theory-driven. These two issues will be further discussed below, along with a new theory-driven model which might change the way we interpret QEEG and generates testable hypothesis.

**Averaged group data vs. individual client data**

An often reported finding in ADHD is the theta/beta ratio. Indeed most group-averaged studies comparing ADHD with healthy control groups will show ADHD kids have more “theta” and less “beta” EEG power (also see figure 1 below). However, when looking at this exact same data on an individual level a completely different picture emerges where only 25% of children with ADHD showed excess frontal slowing. Furthermore, an additional 25% of children showed a slowed Alpha Peak Frequency (APF) which showed up in frontal sites as “theta.” Also see Arns et al., 2008 for the full background on these data. Most importantly these two groups responded differentially to stimulant medication as well. So indeed the theta/beta ratio might often deviate, however what is the cause of that? Excess frontal slow or a slowed APF?

In depression research often the frontal alpha-asymmetry is mentioned and investigated based on Davidson’s work. rTMS or magnetic brain stimulation is also partly based on this work with the assumption that depression is characterized by left frontal hypoperfusion. After inspecting over 200 individual depressed clients from both our clients and data from a clinical trial, I can assure that a specific frontal asymmetry is not reliably found in individual depressed patients. In some previous studies which employed group-averaged data we did find the expected frontal alpha asymmetry, demonstrating that our used methodology is fine. So how can this be, that findings are often found in group-averaged data but cannot be found reliably in individual data?

Take the hypothetical example of 100 people sitting in a room and we average their eye-color. The average will be black! How many people do you know with black eyes?

**Time for a theory-driven approach to QEEG**

*Martijn Arns (primary author), Werner van den Bergh and Jay Gunkelman*
This demonstrates that by averaging group data we might end up with a construct which does not exist in reality. Since we are not treating an average group but an individual client this is very important to keep in mind!

**Technology-driven QEEG**

Currently, many new analysis tools and databases have become available to perform more sophisticated QEEG analysis. Given the large number of permutations some packages allow being done on EEG data will make some statisticians frown their eye-brows (think about alpha-correction for multiple statistical tests). Many of these new techniques have been hardly investigated or validated for clinical use and are often adapted from ECoG or depth recordings from multiple cells and directly applied to EEG. Take the simple example of coherence. There are many different ways to calculate coherence and two QEEG studies in dyslexia have shown completely opposite effects for EEG coherence data (Arns et al., 2007 study and Coben et al. Study presented at ISNR). This could even suggest that what we are concluding based on database X (coherence needs to be up-trained), but by performing the neurofeedback with software Y we might be feeding back something completely different (i.e. downtraining). Therefore the technology driven QEEG approach - although very interesting and exciting – should be complemented more by theory driven QEEG and be better validated and standardized. Ultimately it should be the interaction between theory-driven and technology-driven QEEG which will lead to real new discoveries in our field!

**Theory-driven QEEG**

Currently no real theory for interpreting individual QEEGs exists. One of the first published papers on interpreting QEEGs was the pioneering paper of Jack Johnstone, Jay Gunkelman and Joy Lunt on EEG Phenotypes (2005). Although this paper did not represent a unifying theory, it at least summarized the different EEG patterns or proposed EEG Phenotypes and their proposed treatment recommendation for neurofeedback and medication. Most importantly, it provided a testable model!

Over the last couple of years Jay Gunkelman and myself started testing this model based on data generously provided by the Brain Resource Company. We “EEG Phenotyped” 49 children with ADHD, 113 patients with depression and a group of 170 healthy controls. The “EEG Phenotype” construct proposed the EEG Phenotypical patterns to be stable—trait-like—patterns, hence the term “Phenotype.”

Along these developments Werner van den Bergh exposed us via a newsgroup to the Vigilance model of Bente (1964) and at the ECNS conference last year I was also exposed to a presentation from Ulrich Hegerl who presented data on this same model. This “EEG Vigilance” model is originally based on the work by Bente, and nicely fits into the well investigated sleep-wake states also described by Dement & Kleitman (1957), Loomis et al (1937) and Roth (1961), also see figure 2 below. Vigilance in this sense refers to Henry Head's concept of vigilance: “the organization and efficiency of the adaptive capabilities of the individual.”

When reading the description of the A and B states one sees a lot of similarities between the EEG Phenotypes and these Vigilance Stages, however they are now seen as dynamic variants of the EEG which can occur within the same subject as a function of time! Briefly, they observed that when people close their eyes for 10 minutes, their EEG will in most cases cycle through stages of parietal alpha (A1), frontal alpha (A3), an intermediate “low-voltage
increasing doses an increase in alpha is seen, eventually slowing down with the highest dose. Alcohol and T=3 is after 0.5 liter of vodka. The EEG at T=0 shows a low-voltage fast EEG, and with increasing doses of alcohol (T=0 is pre-alcohol and T=3 is after 0.5 liter of vodka). The EEG at T=0 shows a low-voltage fast EEG, and with increasing doses an increase in alpha is seen, eventually slowing down with the highest dose.

Thus, the challenge of the view of stable EEG Phenotypes emerged. This along with some inconsistencies we found from the original Johnstone et al. paper with respect to treatment outcomes, led us to organize a workshop last November with Jay Gunkelman, Werner van den Bergh and myself. This finally resulted in a new theoretical-model which incorporated both the EEG Phenotypes and the Vigilance model from Bente. Most importantly, the data from our EEG Phenotype experiments fit very well into the model. Details on this new model are currently being prepared for publication.

In this new model we view EEG phenotypes as the “predominant vigilance state.” Werner’s important addition of “counter-regulating mechanisms” is very important for neurofeedback. It is well known for instance that sleep spindles serve to keep people asleep, so prevent the brain from going from stage C back to B. Therefore sleep spindles can be considered a counter-regulating mechanism for stage C. In the same way fast beta is a counter regulating mechanism for stage C. In a small N=1 study we replicated findings Yuri Kropotov has reported before, where alcohol (indirectly increasing GABAergic activity) made alpha re-appear again with increasing doses of alcohol and eventually slowed down the alpha peak frequency as well. Also see figure 4 below. Hence this scale can also be regarded a continuum which is partly regulated through GABA.

More neurofeedback research is needed to demonstrate the effects on this scale, by up or down-training the APF. Given some studies and some preliminary results from our practice, we suspect rTMS or magnetic brain stimulation acts more efficiently on this scale. This might explain the differential efficacy of neurofeedback on disorders such as ADHD (= evidence based) and rTMS on depression (= evidence based).

Looking forward to your feedback and input!

Jay Gunkelman started in the field in 1972, starting the first State Hospital applied psychophysiology lab. In the mid-70s he manufactured hardware with FDA registration, though he then moved into the world’s busiest classical EEG laboratory as head technologist. In the 1980s he began to work with the qEEG, and 1994 he entered commercial service as soon as the qEEG was approved for clinical application. Jay has served on Boards for both AAPB and ISNR, initiating the template for efficacy guidelines project during his ISNR presidency. Currently he is a principal in Q-Pro Worldwide, and is a popular lecturer throughout the world.

Werner Van den Bergh is a neuropsychiatrist after having studied at the University of Leuven (Belgium), with postgraduate fellowship at the University of Nijmegen (the Netherlands) and the Behavioral Neurology unit at the University of London. He presents postgraduate education of psychophysiology in the Flemish Association of Psychiatrists, has published several articles and books about ADHD, dyslexia and neurofeedback, and started recently “Centrum Vigilant,” a multidisciplinary institute for diagnosing and treating neurodevelopmental disorders and for postgraduate education.

In summary, the 2-dimensional Vigilance-Brainrate model is grounded in theory and also fits data. We will be writing up this model and refine it further through more literature and data, which should eventually result in a publication. Although this model is still very preliminary, it generates a lot of testable hypothesis and we welcome any further input from you on how to further improve, test and refine this model. We feel that it is time for a more theoretical underpinning of what we are doing!

Figure 4: This figure shows EEG power in the alpha band with increasing doses of alcohol (T=0 is pre-alcohol and T=3 is after 0.5 liter of vodka). The EEG at T=0 shows a low-voltage fast EEG, and with increasing doses an increase in alpha is seen, eventually slowing down with the highest dose.

Martijn Arns studied Biological psychology at the Radboud University Nijmegen. After several projects in the Westmead Hospital in Sydney, the Max Planck Institute in Munich and Organon Research in Newhouse, he started his own company Brainclinics Diagnostics in 2001. He is specialized in applied neuroscience: bringing neuroscience out of the laboratory with the goal to improve diagnostics and treatments in mental health care. He is specialized in personalized medicine, diagnostic services and treatment of brain related disorders (such as neurofeedback for ADHD and rTMS for depression) using techniques such as QEEG, neuropsychological assessments and rTMS.
Jonathan E. Walker, M.D.

- Board Certified Neurologist
- Board Certified Electroencephalographer
- President of the Neurofeedback Division of AAPB
- President of the American Board of QEEG Technology
- Pioneer in the field of neurotherapy research and treatment, he has used neurofeedback in his medical practice for over 20 years

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Carly’s Healing Journey – Part II

Elizabeth Stroebel, Ph.D.

At age three, Carly was diagnosed with Mesenchymal Chondrosarcoma, a very rare aggressive cancer affecting her spinal column. Despite favorable response to cancer treatment, Carly continued, for the next eight years, to experience the challenges of daily pain and the prospect of life-threatening illness. Part 1 of her story appeared in our summer, 2009 issue. Part 2 continues below.

Comment: The case history follows the incremental contributions of peripheral biofeedback in integrative pain therapy. Peripheral training techniques offer a number of specific advantages which serve to complement cognitive behavioral and neurofeedback-based interventions. These advantages include portability of equipment which facilitates early introduction in the hospital setting, and emphasis on skill generalization training to support between-session symptom management needs. While it was the expectation that Carly would receive neurofeedback training in addition to the biofeedback training we reported previously, it has not happened for a variety of reasons. Nonetheless, the second installment of Carly’s story will be of value to readers interested in adding multi-modal biofeedback treatments to their work with children experiencing pain or life-threatening illness. Carly is being encouraged to engage in neurofeedback to address other developmental issues now that her cancer-related pain is under control. Additionally, many of Dr. Stroebel’s approaches (such as earning trust and using the Quieting Response) can easily be adjunct procedures to neurofeedback for pain management or other presenting complaints. Eds.

Earning Trust

A critical point that sometimes can be overlooked in working with children experiencing life-threatening illness is that the child couldn’t trust the disease to be truthful or fair, so I as the ‘therapist friend’ must earn the child’s trust. The ‘therapist friend’ provides a presence of safety rather than authority, and yes, enjoyment in joining the “feedback loop” from the get-go, as the youngsters reveals their story.

As Carly began her biofeedback work, my task was to empower her by listening, giving her control over therapeutic tasks, and keeping her engaged in moving forward. Often, formal assessment protocols and interrupting questions miss what I call the child’s spontaneous “sticky bits in between”; whereby kids reveal the matters of the heart and the physical hurt essential to the therapeutic process. For example, Carly asked, “Why do therapists interrupt when you are getting up courage to talk about the real tough stuff? When that happened to me, it was just easier to say what they wanted to hear or not say anything.”

Carly has read Antoine De Saint-Exupery’s The Little Prince and feels that the quotation, “It is such a secret place, the land of tears” is pictured in this sketch from his book. “I felt just like this little girl inside and out. Look at the how she’s holding her whole self—so full of hurt and as if she’s gonna fall apart into pieces or disappear. Yes, that was me.” One frequently used formal pain assessment tool clearly did not make a connection for Carly. “Those stupid smiley faces don’t have anything to do with my pain. With the pain scales in the hospital I would pick anything. I didn’t know what they were really asking me.” By contrast, she intuitively responded to a simple tactile pain scale, composed of seashells with surfaces varying in touch from smooth to rough and jagged.

Successful biofeedback therapy, like any therapy, is intrinsically woven into the child’s milieu. The initial therapeutic task with Carly began with finding a non-invasive passage into this precious private personal territory of this youngster. This privilege required a delicate balance in the therapeutic process between embracing her suffering and grief, and with time spent gently and wisely setting safe passage for another journey to emerge. This journey hopefully would reshape and dignify her experience by never taking it away, but rather, softening the sorrow and pain, as a platform to move into another phase of her life.

Therapeutic Tasks

Carly actively monitored her emotional responses on feedback from Heart Rate Variability, thermal, galvanic skin response, electromyography and breathing feedback devices. “I like to see what is going on because I feel in control and love to play with the skills I learn. When you have serious illness and you have pain, you feel like you don’t have any control about what’s happening and that makes you afraid.” CDs containing guided visualization exercises and cue cards reinforcing sequential QR (quieting response) techniques support transfer of training by providing the child with a toolkit to interrupt the faulty bracing (Whatmore, 1979) and to lessen the hurt. “I liked it when Liz and I would hook-up separately and do our stuff. Lots of times I would get to my ‘quieting’ quicker than Liz and I know she’s a wiz at it. I told her she was trying too hard to let me get there first and that caused her stress.” Carly transferred the learning techniques from the get-go. She built confidence that she could

Figure 1: “It is such a secret place, the land of tears” — Antoine De Saint-Exupery

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Carly’s Healing Journey continued from page 17

use QR techniques to extend control over her response to pain anytime, any place and with eyes open. “I didn’t realize I was so super powerful.”

Doing my QR

Figure 3: Quieting Response cue card. “Lately, I’ve been feeling great, and sometimes I just forget to practice except when I get into bed. If I do feel some funny twitch then I just zip into my QR. What helps big time are my cue cards.” Carly

“Even though I am 11 years old, I like these funny Q & R drawings (figure 3). They are great pals who watch out for each other. I know how they feel inside, and how much pain they might be having, just like what happens to me. I might not have felt so lonely if my ‘biofeedback buddies’ were by my bed. Older people can use them too because we all like cartoons. This is just one of the many visuals I used. Telling a person to just relax doesn’t get it. Here Q is helping R to let go and stop pushing against the pain. They are doing quiet easy deep breathing and heaviness and warmth exercises to stop the pain. It’s contagious. The cue cards are great for reminding me what to do. Makes me laugh. Just listening to instructions isn’t the same as seeing them. Doing biofeedback lets me jump outside myself to spy on what’s going on in my head and body.”

Through her own experiences in biofeedback therapy she began to internalize the active ingredients of training, i.e., therapeutic relevance, emergency response, discriminating emotional and physiologic arousal states, eustress, adaptive homeostasis, shaping, empowerment, placebo effect, passive volition, transfer of training skills and compliance. The weekly sessions that followed allowed Carly “to say a thing and say a thing” until the need to say it at each session was less relevant.

Evolving Journey ...

I am careful how I use the word change. For some kids, whose trauma dominated their early childhood and beyond, they fear letting go, as if it were some kind of betrayal of the injustice to what they experienced. Letting go of this trauma was ‘a tricky bit’ for Carly. She and I worked on softening the memory with a very helpful GSR training program from Israel called ProRelax. As the screen opens, the array of nine ascending colors appear with the moving ball at the bottom (see figure 4). Hooked up to GSR, Carly uses her self-regulation skills to move the ball from bottom to the top. She feels empowered as the ball slowly softens the intense color on the screen. The memories of her daily battle with illness and pain now soften and recede far in the background of her daily life.

Now, the word change is not a word of abandonment but instead, like the song says, Carly is “Moving on up…” to a life no longer dominated by the threat of illness. During some sessions, she uses this softening exercise to deal with current adolescent growing pains and other life demands. Using this softening technique validates what was and is now possible for Carly.

Over the following year, Carly’s understanding of the emotional meaning of her healing journey continued to evolve. One recent afternoon, I invited her to examine my collection of stones and seashells, selecting those most relevant to her personal healing journey. This process is important for kids to conceptualize the physical and emotional trauma and to see/feel the progression and then the empowerment from their healing journey. As I listened to Carly retell her tale in sea shells (figure 5), I realized that she had successfully integrated much of what I had tried to impart to her about her capacity to take charge over her relationship with her pain, and to find the personal resources to make safe passage through her suffering, pain and grief, without need to deny or ignore it.

The Shift

Upon turning eleven this past May, Carly announced, “You know, Liz, I’ve decided I don’t want that sweatshirt that says “I had cancer” anymore. And I don’t need to tell people I had cancer. They can read my stories but I’m over that. I want to plan my future like continuing my dance classes and gymnastics; and going to college, getting married and having kids.”

On a recent follow-up appointment, I invited her to revisit her collection of stones and seashells. Carly thoughtfully rearranged the shells. “I am placing the shell with the crystal in the beginning and the others in the background. My life is moving beyond cancer. I have new plans for my life. I won’t throw the other shells away, they were a part of my life, but I am living a great life now. I don’t feel under threat anymore, so what is past is past.”

In the past year, Carly spoke at Paul Newman’s Hole in the Wall Gang Camp in Ashford, CT, the week prior to Newman’s
death (figure 6). She danced with Bette Midler. Afterwards, Alec Baldwin patted her on the back and said, “Wonderful job there, kid.” In June 2009, Carly was invited to be part of the gala fund raiser at Lincoln Center NYC for the Hole in the Wall Gang with Julia Roberts and others. In July 2009, Carly and her grandmother, Mimi Olsson, who is on the Executive Board of the Liddy Shriver Sarcoma Initiative, were invited to Washington, DC to meet with physicians, US government representatives and ambassadors from other countries, as part of the global Team Sarcoma Initiative. Carly is not phased by all this recognition.

Carly has been pain-free for the past three months. She has been able to return to gymnastics and recently participated in prize-winning regional events with the North East Dance Academy. As her own pain has abated, her focus has begun to shift to other children who continue to be in pain. Carly is now volunteering as a youth advocate for the much neglected area of aftercare programs for young people with chronic pain and life threatening illness, and is a contributor to the soon-to-be-released “Sailing Away the Pain with QR” aftercare program with this therapist (figure 7).

References


Elizabeth Stroebel, Ph.D is known internationally for her work in the field of applied psychophysiology with children and adolescents. Dr. Stroebel is a member of ISNR and serves as co-chair of AAPB’s Education Section. For 12 years she worked in the UK with professor Linford Rees, MD, past president of the British Medical Association. Dr Stroebel was instrumental in introducing biofeedback-based pediatric pain management services to major teaching hospitals in France, UK and the United States. A graduate of University of London, she has published internationally, most recently contributing to “Biofeedback in der Praxis: Band 1: Kinder” Springer publications, Vienna, 2007 and to the NeuroConnections Newsletter 2007, 2008 and 2009. Her current project in progress is “Sailing Away the Pain: Managing Distress with QR” for acute and chronic pain and illness in children, a multimedia resource set for children, their families and therapists. Previously affiliated with neurotherapy practice groups in Florida and Rhode Island, she is currently in independent practice. She can be reached at elizabethstroebel@hotmail.com.
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This is the story about how NeuroField was developed. I began my journey in the field of Neurofeedback after working in the mental health field for over 10 years. My doctoral training is in health psychology in which I was trained in medicine and psychology in an effort to be one of the new prescribing psychologists. Over time I became discouraged with the western medicine approach to psychiatric illness and was intrigued to learn that biofeedback techniques could be utilized to help this client population. I learned traditional neurofeedback techniques and my private practice quickly morphed into what I now call an energy psychology practice. I practiced traditional Neurofeedback methods and was initially trained by Margaret Ayers, but learned from many others who were kind enough to share their wisdom and knowledge with me.

I had been practicing traditional neurofeedback up until the birth of my son who had been born anoxic and premature. When I met him in the NICU I realized that he suffered significant trauma to his brain and that he would need my help.

Naturally I made the decision to find a way to help him as soon as possible and I knew it would not be easy. Everything I had learned about neurofeedback was geared towards children who were old enough to engage in the training procedure. At that point in time I had not treated infants using traditional neurofeedback methods and had not read any studies suggesting that it was possible to do so successfully. When I attached EEG electrodes to his scalp for the first time my heart sunk as I observed exceedingly high amplitude, low frequency activity. I was concerned for his well being and wondered how he would ever be able to function in the world.

I engaged in a two-year search looking at as many energy devices that I could lay my hands on. Some had merit while others were misleading and did nothing short of relieve the user of his/her hard earned money. Furthermore, many of the companies that made these devices would sell them to anyone, anywhere. All you needed was the cash. I felt my hopes begin to drop until I came across the LowEnergy Neurofeedback System (LENS). When my son was 18 months old he was barely walking, spoke in one and two word sentences, had hypotonia, a blood disorder, and had extreme visual and auditory sensory integration problems. The LENS made sense to me and was grounded in neurofeedback principals that I could apply to my son sooner than later. Upon obtaining this system I began treating my son and he responded very well to the treatments. The LENS had a significant impact on his life and improved his overall functionality. Len Ochs, Ph.D. helped me through this time by mentoring me and encouraging me to stretch the limits of my thinking. Over time I would develop protocols for the

Introduction

NeuroField is an innovative neurotherapy device. It provides extremely low levels of variable DC stimulation through various programs that provide the stimulation at certain pre-set frequencies that are believed to influence the energy field generated by the brain and the body, facilitating certain physiological effects. The low level stimulation is provided through a 19-channel electrode cap and is generated from 6 AA batteries. No formal research exists utilizing the device, and thus it must be acknowledged to patients as an experimental treatment at this time.

Approximately 15 months prior to writing this article I purchased NeuroField. Since that time I have used it following informed consent about its investigational nature with approximately 100 patients. In a large proportion of cases the results have been gratifying and thus far the only side effects I have observed were of someone feeling tired during the remainder of the day, or having insomnia for an evening if too much stimulation was provided. I have utilized NeuroField as part of treatment with attentional problems, anxiety, insomnia, head injuries, chronic fatigue, and both acute and chronic pain.

The Inflammation Reduction program has proven especially effective. I was initially quite skeptical that this protocol could impact inflammation. The first individual that I used this protocol with had a chronic inflammatory condition that results in her clearing her throat of phlegm numerous times throughout each day. When she must clear her throat it sounds very loud and disgusting, perhaps best described as sounding as if she is coughing up a fur ball. After using the Inflammation Reduction protocol with her three times in one session (which required about 3 minutes), this individual reported that for the next 3 days she had almost no need to cough and that her arthritic pain was significantly less. Subsequently I have used the Inflammation Reduction protocol with patients who suffered with neck and shoulder pain, rotator cuff pain, carpal tunnel pain, knee and back pain, headaches, and post-surgical pain. In most of these cases the electrode cap is simply placed over the area of the pain, rather than on the head. In this paper I will report on the most severe case of chronic pain on which I have used NeuroField.

Background History

Dotty was a 71 year old, married woman. In the past she had been a very upbeat, sociable person who enjoyed artistic pursuits, church activities, and had a fun loving sense of humor. These qualities had been overshadowed for more than a decade due to harsh chronic pain. She developed a prolapsed bladder and right ovarian cyst and underwent surgery in 1995. Th initial surgery, a full abdominal entry, resulted in a series of additional surgeries through 2004, mostly to repair recurrent incisional hernias.

Beginning in about 1996 she began experiencing serious pain in the area of the incisions. This led to a neuroma surgery in her lower right abdomen in January 2004, which led to an additional incisional hernia. In July 2004 a hernia specialist excised a football shaped section of her abdominal wall along the centerline, approximately 8 inches long and 3 inches wide. During this surgery they found hernia mesh from a

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LENS that would enhance the system and lead to even better treatment effects. In a matter of two years AJ had improved greatly, but I continued to observe significant suppression in his EEG characterized by low frequency, high amplitude waveforms that had little to no variability. It has been my observation that when the brain is damaged cortical suppression is a byproduct of the injury. As the person heals the amount of suppression reduces and normal variability is once again observed in the EEG. However, the brain does not function in a linear, predictable fashion and variables that cause suppression are many making resolving this issue very difficult. I discovered that one of the ways to resolve cortical suppression is to replenish the system with energy so that it could repair itself. The LENS is a discretainment neurofeedback device that disrupts the brain, causing it to re-organize itself. This process pulls a great deal of energy from the body. I have observed this on multiple occasions while treating my son and many other patients which lead me to the conclusion that better results were obtained in LENS treatment when the body had the energy to repair itself.

The research in this area is clear. The two best methods for achieving good neurological health are diet and exercise. This is something that every one of my patients hears when they come to my office. I insist upon it as neurofeedback results are greatly improved, especially in children. It’s a simple concept really, when the body has good fuel and plenty of oxygen it functions better. In my initial EEG evaluation I make recommendations for supplementation for the patient (or parents) to consider. By the time I would work up the data and schedule the first treatment session I would observe improvements in the EEG if the parents had implemented the recommendations. I would also hear that the child had improved and was doing better. The EEG that was once suppressed with little to no variability was now showing variability and the entire EEG would appear to be improved. These observations lead to the question, “How could energy be introduced to the body so that it could repair itself?” It was then that I came across “The Field” by Lynn McTaggart (2003) who wrote the following:

“At our most elemental, we are not a chemical reaction, but an energetic charge. Human beings and all living things are a coalescence of energy in a field of energy connected to every other thing in the world. This pulsating energy field is the central engine of our being and our consciousness, the alpha and the omega of our existence.”

When I first read this paragraph it had a profound impact on me. We are made out of the energy and the foundation of biology rests upon the foundation of energy. Fritz Albert Popp (2002) demonstrated that light is emitted from organic substances and coined the term ‘biophotons.’ The light that is emitted from organic sources surrounds the source that it is emitted from and creates a standing waveform. Popp also demonstrated that energy ‘looks’ for compartments in which to store itself. It is a natural phenomenon that suggests that energy can store itself in biological and non-biological places. If energy can store itself in compartments then it is feasible to theorize that energy can store itself in the human body. After all we have three major compartments in the body, the gut, the heart and the head. In Chinese Traditional Medicine the ‘triple burner’ or the gut, heart and head are regions in which chi is stored. The power plant of the system so to speak. When this system is depleted the organism is prone to illness. When the system is energized it can repair itself and defend against disease.

The notion that we are made out of energy and have regions in our bodies that can store energy made sense to me, but when I thought about cells in the human body on a molecular level the theory...
behind the energetic system simply did not hold up. I had never been taught that there were compartments in the cells that could store energy. I kept on getting stumped by this because many books that explain cellular biology use a version of the human cell that is outdated and reflective of the physiological thinking of the 1960’s. The cell is drawn in most books as a bag that holds various parts of the cell with the majority being composed of solution. Eventually my search would lead me to a book entitled, “Energy Medicine” (Oschman, 2000), where Dr. James Oschman discusses what is known as the “living matrix.” He describes the living matrix as follows:

“The living matrix is a continuous and dynamic supramolecular webwork, extending into every nook and cranny of the body: a nuclear matrix within a cellular matrix within a connective tissue matrix. In essence, when you touch a human body, you are touching a continuously interconnected system, composed of virtually all of the molecules in the body linked together in an intricate webwork. The living matrix has no fundamental unit or central aspect, no part that is primary or most basic. The properties of the whole net depend upon the integrated activities of all the components. Effects on one part of the system can, and do spread to others.”

I would learn that the cell is not a “bag” of solution, but rather filled with filaments, tubes, fibers, and trabeculae. If each cell in the body contains compartments then the assumption could be made that energy stores itself in every cell of the body (Grass et al., 2003). Furthermore, if the living matrix stored energy then the assumption could be made that the “field” or “biofield” could be emitted by systems of cells if not individual cells themselves. Therefore the overall biofield is a summation of the energy emitted from every cell in the human body and the NeuroField is a subdivision of the overall biofield.

After I had convinced myself that the NeuroField did indeed exist, the question as to whether the field was capable of being manipulated was my next question. Could energy be introduced to the field for the purposes of healing? Physics studies have suggested that a molecular structure loses electrons and photons when it is damaged. Laser light healing therapies suggest that if an energy pulse is introduced to the biofield that resonates at the frequency of the damaged molecules then free floating electrons and photons could be introduced back into that molecular system (Lytle, 2004). Damaged molecules will recruit free floating electrons and photons returning the electron ring to a fully populated state. This allows the system to come back on line, so to speak, in a reorganized, balanced fashion. Once this occurs the body is able to use its own restorative functions to repair itself. I had developed the theory that the biofield is an interactive, intelligent, multi dimension-al phenomena that could absorb energy and disseminate it to the areas of the body that match the resonant frequency of the energy being introduced.

With these thoughts in mind I decided to seek out an engineer to help me make the idea of NeuroField into a reality. It takes an enormous amount of engineering know how to build something like NeuroField and I knew that my knowledge of computers was not adequate to complete the task. Enter Brad Wiitala. Brad is the engineering genius behind the development of NeuroField. Our initial meetings were exciting as Brad developed ideas about how to build the circuitry within the specifications that I was requesting. Within a very short period of time he had drawn up a circuit diagram, was ordering parts, and assembled the prototype. After a couple of prototypes Brad had built a functional system that I could work with.

Brad and I developed a system that could emit frequencies at different amplitudes and durations. Through a proprietary method, protocols were developed that could deliver energy back to the body and re-charge virtually any system in the body. We went through several different prototypes and tried different ideas over a period of six months. During this time I knew we were onto something special and my excitement increased by the day. Brad had developed a solid platform for generating frequencies, but the problem was delivering energy into the biofield. We talked about different ideas, but nothing seemed to work. I had examined many different types of invasive methods, but they were too overwhelming and did not lead to what I considered good treatment effects. It’s hard to break out of traditional methods when you are traditionally trained. In traditional EEG we attached sensors to the head and I had yet to let go of the idea of doing so. Then it occurred to me that if the biofield really exists outside of the body then all I would have to do is simply give energy within the projected biofield. I didn’t have to put any energy into the body. I decided to test the idea and asked Brad to spin up a prototype for me.

On one late evening Brad had just finished changing the circuitry so that I could connect my QEEG cap to the NeuroField device. The cap was not connected to me in any fashion with no grounding or electro-paste. The time had come and I decided to try NeuroField on myself and observe the results. Brad asked me what he should do if something ‘bad’ happened to me. I said, “Call 911.” To which he said, “And what am I supposed to tell them!?” I reached over and activated the X1000 stimulation unit. What happened next changed everything. I had developed a 30 second protocol called Brain Fog Reduction which is designed to wake you up and help you to think clearly. After the protocol had finished I felt instantly awake. The lights in the room appeared to be brighter, but I did not generate insight into these changes because I immediately had some ideas that I wanted Brad to add into the software. This occurred around 10 PM after I had seen patients all day and was tired. The Brain Fog Reduction protocol did its job and by the time I recognized it, it was midnight. I was wide awake, focused and calm. I had fed energy into the biofield and my brain responded to it. NeuroField was born and it was time to get other professionals involved to verify this finding.

I introduced NeuroField to a group of colleagues and we began the process of beta testing the device. After a couple of months multiple reports came in suggesting that NeuroField was having a significant impact on the beta tester’s client populations. This group of professionals interacted online and began sharing information that surprised me. Some reported using the NeuroField cap on different parts of the body to reduce inflammation and pain while others reported changes in thyroid blood levels. The ideas poured in and it was a truly exciting time as we began to discover that NeuroField had multiple applications. I observed an almost immediate response in my son as his EEG suppression lifted he became more functional. He also became trainable with conventional EEG methods which I employed (and continue to employ) with good results. As of the writing of this paper he has improved greatly in almost every way in his life and I am no longer gravely concerned for his welfare as I was since his birth. My intention has always been to help him which was my primary motivation for creating NeuroField. However, I realized that I was ethically obligated to share this technology with others so this technology can continue to be researched and developed over time.

Continued on page 24
As a result I released NeuroField publically in August 2008 only to licensed health care professionals.

After the public release of NeuroField, Brad and I decided to research ways to measure the NeuroField effect. Many of the NeuroField practitioners would select protocols through the process of muscle testing the client. They reported good treatment effects which encouraged me to start the process of muscle testing clients along with taking pulses. It was then that I observed variability in the pulse rate when a person was given specific frequencies. When I observed increased variability, people would report that they liked the “feeling” of the frequency and would rate it higher than those frequencies where there was no noticeable variability in pulse rate. It then occurred to me that the heart could be muscle tested by measuring heart rate variability (HRV) (see figure 1). After testing this theory on my client population for three months I developed the theory that increases in HRV are an affirmative or “yes” response to a specific frequency. I also used the Gas Discharge Visualization (GDV) system to measure pre and post changes in the biofield pre and post HRV sessions. The GDV was created by Dr. Konstantin Korotkov in Saint Petersburg, Russia. After reading Dr. Korotkov’s book “Human Energy Field” I purchased one of his machines. Basically the GDV is a camera that takes pictures of the light that emits from a person’s fingertips. These pictures are then analyzed via a computer program and a ‘map’ of the biofield is rendered based on acupuncture points (see figure 2). After conducting over 250 scans the same result was observed. HRV would increase in response to specific frequencies and the GDV post test scan would show changes in the biofield towards normalization when compared to pretest scans. However, since I was measuring HRV with a third party software it was time to test out the idea with my own HRV hardware.

Brad built a prototype HRV module and we began testing this theory. The HRV measurement module was developed over the course of a nine month period. Measurement protocols have been developed to examine real time HRV changes in response to specific frequencies through the NeuroField stimulation device. The HRV measurement device is capable of measuring HRV in a very short period of time. The NeuroField user sets a threshold or acceptance criterion for increases in HRV. The patient is given specific frequencies one at a time and HRV is calculated after each frequency. If HRV increases above the acceptance criterion, then the NeuroField program will isolate that frequency so the user can give it again at a later time. The idea is to tap into the natural healing wisdom of the body so that it can select the frequency energy it needs in order to repair itself. Initial patient observations have been positive with patients reporting improved/stable mood, reduced anxiety, reduced inflammation responses, and increased attentional ability. A new kind of energy biofeedback had been developed in the form of the NeuroField HRV unit.

The HRV module was formally released at the 2009 ISNR conference in Indianapolis. At this time I am collecting QEEG, NeuroField HRV, GDV and patient subjective report data and intend to report my findings during the 2010 ISNR conference in Denver, Colorado. We continue to conduct further research into this area and plan on releasing other physiological add-on modules in the near future. For more information please visit www.NeuroField.com.

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NeuroField Treatment of Pain continued from page 23

1995 surgery that had not been properly attached, was floating free, and had calcified to the consistency of a tortoise shell. It was determined that this and the accompanying inflammation were the likely source of the pain she had been experiencing since 1996 rather than a neurona.

Back in 1996 the following pain interventions occurred: 1) Lidocaine injections in the abdominal wall at the site of the pain, resulting in 6 hours of complete relief; 2) BOTOX injections in the abdominal wall, with no discernable effect; 3) intercostal lidocaine injections at T9/T10, resulting in 26 hours of complete pain relief; 4) intercostal phenol injections at T9/T10 that resulted in extreme neuritis lasting several months. In early 1997 they traveled to John Hopkins Pain Treatment Center where she had pulsed RF intercostal intervention at T9/T10/T11. This resulted in 2 weeks of complete pain relief and 5 weeks of substantial relief followed by a relapse of the intense pain. The pulsed RF procedure was repeated in May 2007 which only brought 2 days of complete pain relief and a relapse of the intense pain within a week.

Narcotic treatment was then recommended, beginning with a Fentanyl patch in June 1997, which was increased from 0 to 100 mcg/hour alternate days over a period of 6 weeks. She did not develop tolerance to the Fentanyl, experienced nausea and dizziness, severe cognitive effects, and loss of balance (with 5 falls where she hit her head) while only obtaining slight relief from the pain. She was tapered off Fentanyl by October 2007, experiencing moderate restoration of cognitive function and mobility, but with a full resurgence of pain. Her last fall had resulted in a depressed fracture in the right posterior area of her skull. Since that time she continued to experience some balance problems, despite being off Fentanyl.

After years of suffering, in early 2008 Dotty had a subcutaneous stimulator implanted at the site of the pain. It reduces her pain, “but not enough to make the pain tolerable.” Frequent program changes in the stimulator proved necessary to maintain effectiveness and she was never free of pain. She was also placed on Neurontin, Soma, and Lorazepam. Prior to treatment with me husband wrote, “Because of the number of interventions that have been tried with limited or no success, Dotty is skeptical of the likely outcome, but is willing to give it a good-faith try and hope for a good outcome.” In our initial interview she rated her pain during the day (when the implanted stimulator was on) as varying from 2-8 in intensity (on a 0-10 scale). The pain was experienced to the right of her naval with referred pain into the rectum and vagina.

Treatment

The patient traveled from out of state and remained in the Salt Lake City area with her husband for approximately 3 weeks, residing with relatives. She arrived for the sessions with her husband pushing her in a wheelchair, and then holding someone’s hand she would walk unsteadily from the door of my office to a chair. In the 3 weeks prior to our intake interview she had tapered off Soma and Lorazepam. After the intake history session she was instructed in the use of a Photonic Stimulator (an infrared light device for pain management produced by OchsLab, Inc). They took the photonic stimulator home and used it as directed daily over a 3 day weekend without positive result. It was believed that the source of the pain was simply too deep in her body to respond to the photonic stimulator. Therefore, following informed consent in our first treatment session, we began using NeuroField. With the electrode cap held by her directly over the site of the pain we ran the Inflammation Reduction program 3 times, and then another program called CNS Repair. The next day she indicated that she had run out of Neurontin. We used the Inflammation Reduction program 4 times over the wound site, followed by the CNS Repair program once. On the third treatment day Inflammation Reduction was run 6 times, CNS Repair twice, and a swelling reduction program was run twice.

Our next session was 4 days later. Despite having a cold and coughing, which would usually make her pain much worse, she rated her pain level as a 1. Her husband reported that she had been walking with a more upright posture. The Inflammation protocol was repeated 6 times, CNS Repair 3 times, and Swelling Reduction 3 times. By the next day (treatment session 5) it was reported that she was doing much more for herself and was being more mobile. We continued this treatment regime for a total of 14 sessions. After 6 sessions she began walking (a long walk) from the front of the hospital to my office, and then afterwards back out to the parking terrace, without the wheelchair. Her pain level continued to improve. At the end of treatment she was usually experiencing no belly pain unless she “overdid it” by being extremely active, in which case there was mild pain. In her last week of treatment, she and her husband went out to a movie and dinner, which was the first time this had occurred in two years. It should also be noted that in 6 of her sessions we used the Low Energy Neurofeedback System (LENS) for two seconds of feedback each at sites directly underneath O1 and O2, barely above the inion ridge. This was done because of the author’s previous work (Hammond, 2005) at these locations with traditional neurofeedback which has been found to improve physical balance. The patient and her husband also reported improvements in her physical balance and they returned to their home in another state.

Follow-Up

In a one month follow-up her husband indicated that her pain relief had been maintained. Further, they had gone on the first vacation that she had been able to go on in four years.

Seven months after the last treatment session the patient’s husband reported that her only medications are Neurontin and Ibuprophen, and that when her implant is fully charged and she is inactive she does not experience any pain. He said, “It is clear that Dotty’s pain is very much better and doesn’t appear to have lapsed back since the earlier treatments.” She is also significantly more active than previous to treatment. Her husband indicated: “She has been doing more and more housekeeping, cooking, etc. And she has appetite and eats normal food. I have a motorized treadmill and she’s got her distance up to a half-mile at 2 mph.” He said that they both considered NeuroField to be a success.

Conclusion

The author’s clinical experience with NeuroField has been very positive, particularly in applications with attentional problems, head injuries, anxiety, insomnia, and both chronic and acute pain. Both the immediate and the enduring results that were seen in this case of severe chronic pain are encouraging that NeuroField may offer an additional treatment modality with pain cases. Based on my clinical experiences I believe that research with NeuroField is clearly warranted. One of the great advantages of the device is its ease of application and the fact that it can be so easily added to treatment since its use only takes a few minutes.

Reference

The Diagnosis

I come from a family of reading experts. My father is a past president of the International Reading Association and a retired professor who taught hundreds of teachers to teach reading. My mother was a school librarian. By age sixteen, I was teaching kids to read at a VISTA program with one of Dad’s bestsellers, The Emergency Teacher’s Manual, in hand.

Naturally I thought my children would learn to read as easily as ducklings learn to waddle after a mother duck. But the preparation of a lifetime had not equipped me for the lessons I would need to learn and to teach. From the middle of my older daughter’s second-grade year to her entry into high school, I worked on the puzzle of her learning issues. My daughter was determined and valiant. Yet at many points along the way, we were both weary, and I did not know when or whether the answer would come.

I encountered the problem on the day after Christmas the year my daughter was in second grade. She was straining to write thank-you notes. Reading had always been harder than writing for her, and yet, when I read the notes, I saw that her writing skills had deteriorated compared to her first-grade skills. The day school reopened I stood before my daughter’s teacher, not believing a word she was saying about children stopping temporarily at a plateau. I worked on the puzzle of her learning issues. My daughter was determined and valiant. Yet at many points along the way, we were both weary, and I did not know when or whether the answer would come.

I staggered home and woke in the night from the tears sliding down my face.

A return visit to the neuropsych tester revealed the severity of the condition. A difference that is greater than 11 percentile points between the IQ and the reading achievement tests is the definition of a learning disability. For example, a child whose IQ is in the seventy-fifth percentile and whose reading achievement is in the sixty-fourth percentile has a reading disability. The difference in my daughter’s case was 73 percentile points.

I also received a diagnosis: dyslexia. Through the next six years, I dug through research on dyslexia and various treatments. Starting in third grade, my daughter had the benefit of one of the best dyslexia remediation programs, designed by Tufts University’s Dr. Maryanne Wolf, a colleague of my father’s.

Progress came slowly and with great effort. My daughter became a good speller. Timed tests eventually recorded a match between her IQ and her reading achievement percentiles. She got an A minus in French.

But the price was still too high. “I hate reading. Mom, and nothing you can do will make me like it,” my daughter would hurl at me, her discouragement overflowing. She could do short reading passages for a timed test, but sustained reading was so tiring that I worried about the demands of high school.

Along the way I heard two things repeated: “She is lucky to have you as her mom” and “She will improve, but she will never be cured.” But she didn’t feel lucky. She felt like she was always climbing an endless peak to please me while other students coasted downhill.

Enter Neurofeedback and tDCS

A break came early in 2006, when my daughter was in the sixth grade. I read “Emerging Interventions,” the January 2005 issue of Child and Adolescent Psychiatric Clinics of North America. Dr. Laurence Hirshberg, the editor of that issue, also directs the Neurodevelopment Center in Providence, Rhode Island. I set out with my daughter to meet Dr. Hirshberg and learn about the center’s home-based neurofeedback program.

We began treatment designed and supervised by the Neurodevelopment Center in June of 2006. The program improved my daughter’s fluency in spoken language and her concentration, but added no perceptible gain to her reading fluency or comprehension that was not already conferred by her schoolwork and specialized tutoring.

Despite improvements, a perplexing problem had dogged my daughter’s efforts to read from the start: she inserted words and skipped words while reading aloud, at a rate of 5 to 15 errors per 100 words. Her reading voice was mechanical, as if the effort of sounding each word was so great that she could not express the meaning of the text by using an appropriate sequence of tones.

The level of difficulty of the text did not affect my daughter’s performance when she read aloud, nor did her familiarity with the subject. I hypothesized that she had a visual processing problem that prevented her from seeing a line of print the way most people see it. As the sophistication of the reading material progressed, it was likely that her comprehension was likely to drop—for example, when meaning turned on a comma.

As my daughter continued with neurofeedback, I had the opportunity to learn more about research in the field. The website of the International Society for Neurofeedback and Research (ISNR) was particularly helpful, and I decided to order several workshop DVDs from ISNR’s 2006 annual conference. Kyle Fredrick, who provides support for the Neurodevelopment Center’s program of home training, recommended two DVDs by Dr. Juri Kropotov: A New QEEG, ERP Database & Its Applications for Neurofeed-
back and Transcranial Direct Current Stimulation and Application of the Human Brain Institute (HBI) Normative Database for Assessment of Brain Dysfunctions.

Dr. Kropotov directs the Laboratory for Neurobiology of Action Programming at the Institute of the Human Brain at the Russian Academy of Sciences in St. Petersburg, Russia and is a professor at Trondheim University in Norway. His work, I learned from his writings and his DVDs, differs from the neurofeedback with which I was more familiar in three ways.

1. Dr. Kropotov does a QEEG in a way that includes measuring the brain’s response to stimuli or cognitive challenges such as a brief exposure to a word or a sound — cognitive ERPs. This method gives a more comprehensive look at cognitive processing, and therefore it can show where, in the time elapsed after the presentation of the stimulus, the problem arises.

2. The features of Dr. Kropotov’s QEEG database, which comes from a large number of adults and children in three countries, enable it to highlight subtle but significant differences for people whose problems might go undiagnosed when their QEEG data is compared to the same type of information in other databases.

3. Transcranial direct current stimulation (tDCS) directs a very small, carefully controlled amount of electricity into the brain, as opposed to simply providing visual or auditory feedback based on the brain’s own patterns.

Initially I was unable to see how Dr. Kropotov’s DVDs, which arrived near the end of 2006, would be particularly relevant to dyslexia. Then, at minute 28 of the second and longer of the two DVDs, the revelation emerged. “In dyslexic patients [the] P1 component is impaired. For visual dyslexics [the] P1 component for visual stimuli is impaired. For auditory dyslexics [the] P1 component for auditory stimulation is impaired … Coherence is a very good parameter when you talk about dyslexics because in their case the parietal area doesn’t communicate with other areas.” I had come upon a promising lead. My daughter clearly had visual impairment, and, because she disliked talking on the phone, I thought she might have some auditory impairment as well.

I called Dr. Hirshberg, “Where is P1? I can’t find it on any of the site charts,” I said. Dr. Hirshberg, to his credit, did not burst out laughing, but patiently explained that P1 relates to time elapsed after an ERP, in this case, one hundred milliseconds after stimulation.

The timing issue dovetailed with other descriptions of how dyslexia functions after a reader sees a word. I understood that the process of comprehension slows down too much for people with dyslexia at the point when a word is decoded, about three hundred milliseconds after exposure to the word also known as P3. Perhaps Jurij Kropotov was seeing the same phenomenon of dysfunction at around P1 to P3 but from a different perspective than academic reading experts. But I didn’t see how neurofeedback could fix a timing dysfunction.

On the next trip to Providence, in the winter of 2007, I showed Dr. Kropotov’s mention of dyslexia to Dr. Hirshberg, who thought that either tDCS or traditional neurofeedback might be able to improve a timing problem.

I went home with a thousand thoughts running through my mind. Could my daughter withstand another evaluation and another treatment without intolerable resentment? How safe was the tDCS treatment? And, above all, could it possibly work?

We decided to move forward with a new QEEG. I got a workout on the Internet as I downloaded papers by Dr. Kropotov and continued to discuss tDCS with Kyle Fredrick and Dr. Hirshberg. The effect of conventional neurotherapy for my daughter had leveled off, and her reading aloud fluency was stuck. Dr. Hirshberg asked Dr. Kropotov via email if we could meet with him at the September 2007 ISNR conference in San Diego. Dr. Kropotov emailed back, “I would be glad to help the mother.”

In San Diego, Dr. Hirshberg showed Dr. Kropotov the EEG data from my daughter’s latest QEEG, but it was clear that she would have to do another QEEG on a system compatible with Dr. Kropotov’s ERPs and database. Fortunately Dave Myer, whose office in Burlington, Massachusetts, is near my home, was also in San Diego. He had the specialized equipment and could do the QEEG and email the results to Dr. Kropotov, who had returned to St. Petersburg. It took three visits, tears, and the promise of the latest jeans for my daughter. Finally Dave Myer captured the data. The nearly unbearable QEEG proved crucial. One previous QEEG analysis had contained language that claimed my daughter was a “good reader.” Dr. Kropotov found otherwise.

When Dr. Kropotov emailed back the report, the finding most relevant to my daughter’s reading pattern was as follows: “ERPs components. The most significant deviations are found in visual related components generated in occipital and occipito-temporal areas.” Further on, the report stated, “The raw ERPs show strong deviations from normality in occipito-temporal-parietal areas. The strongest deviation is found under the P4 electrode. Taking this observation into account the tDCS protocol might be directed for activation of this part of the cortex.”

Another email added some intriguing information: “She clearly has deviations from normality in visual processing … I’ve seen it [for] the first time in my life [in your daughter’s data]. Quite unusual. You can try an experimental procedure with tDCS. If you agree I can send you a protocol.”

I did agree, and I wanted to do the treatment quickly. Timed tests were coming up at school, and they could be important for my daughter’s future. Dr. Hirshberg and I worked quickly on getting the protocols from the busy Dr. Kropotov, on arranging supervision from an M.D. and sign-off on treatment, and on getting the equipment: the edlith dc-stimulator, from neuroConnGmbH, a company in Ilmenau, Germany. Early one Sunday morning, I flew to Philadelphia to pick up the equipment from a sales representative, who had traveled from Germany to attend the annual meeting of the American Epilepsy Society. On the return trip to Boston, I held my breath going through security at the airport, hoping the guards wouldn’t ask about the dc-stimulator. Handing over the equipment to Dr. Hirshberg in Providence, I said, “It feels like I’m carrying Kryptonite.”

A few days later, near the end of 2007, my daughter had her first tDCS session. Per Dr. Kropotov’s instructions, she did a total of ten sessions: four sessions at T5 for thirty minutes each and six sessions at fifteen minutes apiece at P3 and P4. The anode pad went on the sites, and the cathode pad was on the opposite-side mastoid. Dr. Kropotov feels that ten sessions of tDCS are sufficient and specifically recommends that sessions be limited to ten sessions. If symptoms don’t improve he recommends another QEEG in six months, followed by a revised protocol and ten more sessions.

The sessions occurred at three-day intervals. If we did a session on Monday, the next one was on Thursday. Because we
could do the sessions at home, we finished in four weeks.

Though Dr. Kropotov recommended that my daughter do the FastForward program (a computer-based program for people with dyslexia or auditory processing problems, or both) simultaneously with the sessions, I had to substitute a teen soap opera co-starring a horse to elicit participation from my then fourteen-year-old daughter.

Ace bandages didn’t keep the electrode pads in place at P3 and P4, so I held the active electrode pad myself. This meant I got to watch the teen soap opera, which turned out to be quite entertaining.

The safety measures that were built into the equipment kept me from worrying about the possibility of too much electric current being applied.

“Larry, it looks like you are going to be the first in the neurofeedback field to try tDCS,” Dr. Kropotov had told Dr. Hirshberg when we began the new treatment. One year had elapsed from the day I initially mentioned tDCS to Dr. Hirshberg.

**The Results**

The tDCS treatment results were mixed. My daughter’s spoken fluency improved more than it had when she was doing neurofeedback sessions. Her eighth-grade teacher remarked on her willingness to speak up in class. Only three years before, my daughter had not been able to arrange the words in a sentence in conventional order. I had to unscramble the order before I could understand what she was saying. Her fifth-grade teacher had told me that my daughter’s reading aloud was incomprehensible to the other children.

Even more positive was my daughter’s performance on timed tests. She took two tests before treatment, in November and December of 2007, and one after treatment, in January of 2008. Her reading performance was identical on the first two tests. Her score jumped 22 points, for a 37% improvement, on the third test, the only test following treatment. Her quantitative reasoning score improved even more. She had not had any test-specific tutoring between the second and third tests.

Yet she still hated to read and wouldn’t read a book for pleasure. When she read aloud, the same pattern persisted: the dyslexic-type mistakes of inserting and omitting words, with an error rate between 5 and 15 words per 100 words read aloud. I had asked her to read aloud before and after each tDCS session, carefully noting the number and types of errors on a separate copy of the reading material. The tDCS sessions had produced no sustained improvement in her error rate.

Dr. Kropotov emailed us that we might not see any improvements for six months. In any case, my daughter wouldn’t have done another treatment for all the jeans in Abercrombie and Fitch. Off she went to high school in the fall of 2008.

And there we rested.

Sixteen months after the tDCS treatment ended, my daughter picked up a book for pleasure reading for the first time. She read two thousand pages in one month, in April of 2009. She read on the way to school, she read on the way home, and she read throughout dinner. She asked to go to the bookstore so she could buy more books that featured vampires. She read past her bedtime and had shadows under her eyes in the morning.

Her tutor tested her at length several times. She read aloud with a normal error rate of 1 to 2 words per 100, and those mistakes were the kind a normal reader makes. She read quickly. She read with expression.

I have no way of knowing when her reading ability improved. It could have happened months before she picked up that book; she did do her copious school reading with fewer complaints, starting about six months after the tDCS sessions ended. All I can say with certainty is that her pattern of inserting and deleting words had virtually disappeared when she read aloud in April 2009.

I asked Dr. Kropotov whether, sixteen months later, tDCS could have effected a change of this magnitude. He wrote back, “We see those effects quite often: we start activating the cortex and nothing happens during the sessions (except some mild changes in arousal), then in a while (it depends on the patient) the effect of stimulation accumulates and hits the threshold. After this moment the progress is accelerating.”

“Do not forget that tDCS is a minor intervention with a very small current that provides small changes at the level of cortical activation at the moment. But, to modify the brain, time is needed.”

**Gratitude Beyond Words**

My father’s work in the reading field has helped many thousands of children, but he couldn’t help his granddaughter. She needed a team from the new millennium and a revolution in technology. How fortunate she is to have had the help of Dr. Maryanne Wolf, Anne Knight, Kyle Frederick, Dave Myer, Dr. Hank Mann, Dr. Wolfgang Keezer, Klaus Schelhorn, the developers of the eldith dc-stimulator, and Dr. Juri Kropotov. Most of all, I am grateful to Dr. Laurence Hirshberg, whose ability to answer every question, synthesize a wide range of research, and venture forth to try promising new treatments opened the world of reading to the girl who would never be cured.

I give the last word to my daughter, whom Dr. Kropotov called “a brave fighter.” My daughter’s love of family and exceptional work ethic survived seven years of academic struggle, remediation, and treatment, and I admire her endlessly.

“My Lit and Comp class is easy this year. *Macbeth* is easy. I still don’t like *Wuthering Heights*. What is the big deal? All this stuff is just easy. I have things under control now, Mom.”

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10) Neurorep - W. Hudspeth QEEG Analysis System: Task
    $70.00

11) Supervision and Training Hourly Rate
    $100.00

12) Extra set of Printed Maps sent priority mail
    $35.00

13) Electronic (sent via FTP or E-mail) and Paper Copies of Maps sent priority mail with package purchase
    $20.00
   (Standard package rates only include electronic or paper copies of maps, not both)

14) Overnight Shipping & Handling (Price varies with carrier, destination, & package weight)
    $Varies

total value: $630
Biofeedback and neurofeedback are increasingly popular topics in the mainstream media from optimal performance training of Olympic athletes to the treatment of soldiers with traumatic brain injury. Ian Wickramasekera’s message of “skills not pills” may become more persuasive as drug co-pays increase and public confidence in medication safety is shaken by reports of serious side effects. The military has significantly expanded funding for biofeedback and neurofeedback services and more universities provide this training in their student wellness centers. There has been an explosion in the number of consumer products that teach the concept of self-regulation. The next generation of videogames promises to include biometric controllers that will monitor signals like the EEG to direct game play.

Since consumers frequently have to pay for our services “out of pocket” due to limited insurance coverage, they are rightfully concerned about how to select a qualified provider. Biofeedback and neurofeedback professionals, in turn, seek Biofeedback Certification Institute of America (BCIA) certification to demonstrate their competence in an increasingly competitive marketplace. Our certification numbers are growing both in the United States and internationally. Increased global recognition of BCIA certification is a testament to our high education and training standards and increased recognition of the legitimacy of our field.

We have been impressed by the professionalism of our international certificants. Many have overcome multiple challenges like the limited availability of didactic courses and mentorship opportunities, prohibitive travel expenses, poor compensation for their clinical services, and equipment costs that may be two to three times higher than in the United States. Despite these hurdles, they have chosen the BCIA credential because it is the international “gold standard” for biofeedback and neurofeedback certification.

Many of our best instructors, like Erik Peper, Lynda and Michael Thompson, and John Demos, have taken their didactic programs abroad and have also provided distance mentoring. BCIA now has strong certificants in Austria, Canada, Japan, Mexico, the Netherlands, and South Africa. From what we have heard, Poland and Sweden may be next. We expect these new certificants to produce the first generation of “homegrown” BCIA blueprint teachers and mentors.

The University Initiative, which promotes the creation of biofeedback and neurofeedback courses at universities, has been an important part of our global outreach. We are proud of the first BCIA-accredited neurofeedback curriculum offered entirely in French. This course is offered at the Institut de Neurofeedback du Quebec by Vincent Paquette and Johanne Levesque. We are equally proud that Monika Fuhs at Sigmund Freud University of Vienna now offers biofeedback and neurofeedback courses based on the BCIA blueprints. Both universities have chosen student completion of BCIA certification as a program objective.

The Biofeedback Foundation of Europe (BFE) is one of the few international biofeedback organizations that offer coursework as well as a gathering place to network and exchange ideas at their annual meeting. The Foundation’s advisory board is comprised of leading clinicians and researchers from multiple disciplines who share a common interest in the dissemination of information about our rapidly growing field. Many countries are represented, including Austria, Germany, Greece, Israel, the Netherlands, and Poland. BCIA has staffed a booth at BFE for the past two years. We look forward to maintaining our presence at this meeting and even offering a proctored exam in the future.

The Japanese Society of Biofeedback Research (JSBR) was founded in 1973 with a distinguished history of biofeedback research. The organization represents the Medical, Engineering, and Psychology/Education fields. We are proud to welcome some of their finest clinicians to BCIA, who were originally certified through their own society.

Additionally, there are biofeedback and neurofeedback associations in Austria, the Baltic States, New Zealand, and Switzerland. As our field grows, these organizations should expand, resulting in greater opportunities for collaboration.

BCIA is working hard to eliminate obstacles to both US and international certification. We have made considerable progress in promoting distance learning and distance mentoring, and in providing online access to continuing education and testing.

**Distance Education**

We are proud of the distance education programs that teach didactic coursework based on our Blueprint of Knowledge. There are three distance-based didactic programs for General Biofeedback, two for Neurofeedback, and one for Pelvic Muscle Dysfunction Biofeedback. We encourage more universities and accredited vendors to offer training programs that fulfill our didactic education requirements. This will increase the number of certificants and help professionals to maintain and enhance their skills.

**Distance Mentoring**

Distance mentoring has nearly eliminated the challenge of finding a qualified professional to supervise the learning of personal self-regulation and clinical skills. Many of our mentors successfully use internet-based technologies like Skype® and Go To Meeting® to demonstrate and directly observe...
skills like electrode placement and identification of artifact. Current technology allows our professionals to view a candidate’s screen, observe clinical technique using a webcam, and demonstrate biofeedback program features on the candidate’s own computer. Expected technological advances, like increased bandwidth and faster graphics processing speeds, should expand the availability and richness of distance mentoring.

**Online Continuing Education and Teleseminars**

Previously, several national and international professionals did not pursue BCIA certification or maintain their certification due to the limited availability and cost of continuing education coursework. In September 2009, the BCIA Board launched affordable online continuing education in collaboration with the Association for Applied Psychophysiology and Biofeedback (AAPB) and the International Society for Neurofeedback and Research (ISNR). We selected seminal articles from the Biofeedback Magazine, Applied Psychophysiology and Biofeedback, and the Journal of Neurotherapy that are available online and then developed brief online tests that cover their main learning objectives. To reduce your expenses, we selected articles that are free to the public as well as those that are only free to subscribers, and will only charge a minimal fee for online testing. Please visit the recertification area under the Certificants Only section of the BCIA website at www.bcia.org to read more about this option.

AAPB’s Teleseminar series provides another affordable continuing education option. These innovative 90-120 minute educational programs allow certificants to obtain 1.5 to 2 hours of accredited, Category A continuing education from the comfort of their home or office. These two online options allow our certificants to fulfill their continuing education requirement toward recertification without the added travel expense and inconvenience of closing one’s practice.

**Online Testing**

We are pleased to announce the first generation of BCIA online exams. This process started slowly with the introduction of the human anatomy/physiology exam as an alternative to completing a semester anatomy/physiology course. Based on our experience with this first online exam, we launched online exams for all three certification programs. As of August 2009, we had successfully offered online exams in Mexico and South Africa, with many more to come. Secure online testing has benefited both our American and international colleagues by eliminating their travel costs, making it easier to arrange for exam proctoring, and significantly reducing their special exam fee.

**The Next Challenge**

What is the next major challenge for international certification? In the future, we would like to translate our exams into our applicants’ native languages. This step will not be practical until our core reading lists are available in these languages and there is sufficient demand for these tests. We are hopeful that new translation products will become available in the next decade so that our certification will truly become a “credential without walls.”

We are excited by the potential for international growth in General Biofeedback, Neurofeedback, and Pelvic Muscle Dysfunction Biofeedback. BCIA certification can help promote this development by providing an international standard for didactic education and training in these areas. We are encouraged by the number of international universities that have developed courses based on our Blueprints and that expect their graduates to demonstrate their competence by achieving BCIA certification, so that they will be “More than qualified – BCIA Certified!”

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**Small Group Discussions**

**ISNR 17th Annual Conference in Indianapolis, Indiana**

The small group discussions are held at lunch time each day at the annual conference. They are led by experts on the specific topics and are scribed by volunteers. These discussions have become quite popular, providing a forum for open dialog and informal discussion on specific topics. New treatment protocols are introduced by the attendees as well as the leaders and specific information related to the topic is disseminated. These notes are transcribed from handwritten notes taken during the discussions and therefore may not be written in perfect syntax. For further clarification, please contact the leader. You can find the leader’s contact information on the member’s list at www.isnr.org.

**Neurofeedback for Anxiety**

**Friday, Sept 4, 2009**

**Leader:** Tom Budzynski, PhD  
**Note taker:** Anna Tur

In this small group discussion there were 47 attendees. Initial discussion regarded the history of the research of anxiety and the statistics of prevalence. Bob Gurnee cited six subtypes as determined by the EEG. OCD appears with specific frequencies and localizations. Precautions were discussed with regard to using neurofeedback for anxious clients. The concept of the default mode network was discussed and the following protocols were recommended: frontal asymmetry training, rewarding increases in theta and inhibiting power in beta, inhibiting 19-34Hz and z-score training. Monitoring the anxiety level of the client was also considered and included being aware of the level of anxiety of the client, using the Premack Principle and using brief guided relaxation tapes and the ‘revitalizer’ tape.

**Neurofeedback for ADHD**

**Saturday, Sept 5, 2009**

**Leader:** Lynda Thompson, PhD  
**Note taker:** John Nash

Several topics of discussion were engaged:

1. Discussion of dealing with decreasing medications
with children receiving neurofeedback: It was noted that interaction with the prescribing physician is important; educating parents to be able to articulate their goals with the physician; the idea that as training progresses, symptoms (side effects of medication) can occur and may disappear as medication is stepped down.

2. Drs. Eugene Arnold and Nick Loft- house attended and were introduced as the investigators running a pilot project on neurofeedback with ADHD at Ohio State University. Dr. Arnold commented that we know stimulant medications are effective for about two years. Then the results of the NIMH Multi-Modal Treatment of ADHD Study indicate loss of effectiveness. He also recognizes that adolescents may need lower doses, although they may discontinue altogether because of not likely how they feel on the medication, i.e., overly focused and less gregarious and social. He noted that current pediatric practice guidelines recommend trying to reduce medications if the child has been doing well and then gets worse on a given dose. It was noted that worsening symptoms often bring an automatic dose increase. Dr. Arnold then discussed the OSU study design. This involves single channel feedback and a standardized theta/beta protocol. He noted that while blind to the group membership, he “is seeing variance” in the measures of the first cohort of 19 children. He noted that, seeing variance may indicate something active is happening to some of the children, although the blind protocol prevents anyone from knowing which group – experimental or control – they are in, until the end of the study. The next group of participants will be given pretesting and post-testing with QEEG, although the protocol will remain the same. After the study, differences in response to the treatment will be studied in light of initial QEEGs. A future study may use QEEG data to select a particular endophenotype for treatment. Dr. Arnold then discussed the reluctance of the medical establishment to accept neurofeedback based on the “RUDE” model—new treatments that are Risky, Unlikely, Difficult or Expensive tend not to be adopted easily without very clear proof of efficacy (neurofeedback being Difficult and Expensive).

3. There was a discussion on artifact control during neurofeedback.

4. The use of “not too interesting” displays was discussed, with the idea that children who become bored easily have to learn to stay focused during low level, relatively unstimulating tasks.

5. The use of multiple cognitive tasks during neurofeedback was discussed. Several providers indicated they routinely use listening, reading or other tasks as part of the neurofeedback session. This went into a discussion of generalization of the skills learned during neurofeedback.

6. There was discussion of conscious acquisition of strategies for sustaining interest and focus, as well as “cuing” of the focused state with pre-arranged prompts that can be shared with child, parents and teachers.

7. Ideas about using points during neurofeedback sessions, earned rewards and prizes for points.

8. The idea was discussed that it is useful to be able to demonstrate learning curves in EEG data resulting from training. Also the idea that it is useful to track EEG and behavioral data across sessions and at the beginning, middle and end of therapy.

Neurofeedback for Alcohol and Addictions Saturday Sept 5, 2009

Leaders: Richard E. Davis, MS and Genie Bodenhammer-Davis, PhD
reddavis@charter.net and genie@unt.edu
Note taker: Deb Stokes, PhD

Genie Bodenhammer-Davis encouraged us to review the article in the 2008 volume 12, issue 1 (p. 5-43) of the Journal of Neurotherapy by Estate Sokhadze, Cannon and Trudeau which described how EEGs look in response to the different addictive substances. Article is entitled “EEG Biofeedback as a Treatment for Substance Use Disorders: Review, Rating of Efficacy and Recommendations for Further Research.”

The Menninger Clinic was the first to use biofeedback with addictions. Eugene Peniston transferred to the VA in Texas with the Davis’ and trained them on the Alpha Theta protocol which he said was “given” to him during his own alpha theta session and that it was shown to him during his alpha theta state. He had a behavioral rehearsal or script that he used as an induction before each alpha theta session with clients. He typically did five to 10 sessions of temperature training, allowing the hands to reach approximately 95°, which he said was necessary to begin producing alpha and theta. Then he would perform progressive relaxation techniques with a script or rehearsal. 30 sessions of alpha theta would then be performed, where increases in alpha were primary goal and theta increases were secondary. During the course of treatment alpha would increase first and then theta would cross over the alpha. Clients would often report autobiographical memories and would process this with the therapist after their alpha theta session. The Peniston study was replicated several times.

Scott and Kaiser published a study on poly substance abusers. Alpha theta does not work with everyone. Sometimes, individuals can get more depressed. Stimulant abusers have more theta and tend to have an ADHD profile. Frontal theta was treated first then they did posterior alpha theta work and got good results.

Richard Davis, and Genie Bodenhammer-Davis are achieving an 80% success rate at the University of Texas. Of the 20% who fail it was speculated that they may not be ready to quit. And if not, they can actually go through 30 sessions of alpha theta with no effect. Another reason for failure might be if they have high beta over the vertex and if this is not calmed, they don’t do as well.

Re: number of sessions Peniston did five times a week once a day. Richard Davis does twice a week or more in outpatient setting. At the clinic he tries to see people at least three times a week there. He doesn’t do alpha theta but instead normalizes the qEEG map. These clients often have 2 to 7 Hz slowing or increased beta at Cz. Sex addicts also have elevated amplitudes in these areas.

A three-year study at the Open Door Mission on crack addicts had impressive results using a modified Peniston protocol at O1 site for 40 sessions. Success was defined as no drinking or drugging for six months, no unemployment or homelessness. Success rate was 65% without any

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alcohol and 85% with no more than three drinks.

Jay Gunkelman identified phenotypes associated with addictions. Those with slowed alpha in the parietal area (7-9 HZ vs. 9-11) may not as well with alpha theta. (Look for this article on ISNR website and type in “phenotypes” to locate it.)

Those who have crossover without increases in beta may not remember their alpha theta experience. But remembering is not necessary in order to recover and may in fact be more effective.

Dr. Daniel Amen’s Brain Place online has SPECT images on the ravages of addiction and substance abuse which are very useful to show clients and have displayed in your office.

Abuse and trauma is very common with the addicted population. These individuals can have a bradycardia with Alpha Theta.

Neurofeedback for Depression
Thursday Sept 3, 2009

Leader: Joy Lunt, RN eegjoy@aol.com
Note taker: Deb Stokes

Joy related a case study on a client with severe anxiety where she obtained a qEEG that showed problems in the temporal lobe, which is more indicative of depression than anxiety. Frontal work with this patient was not as effective as temporal neurofeedback. The lesson here was to collect as much objective data (qEEG) as possible before determining best protocols. There is a device called a Biz monitor which monitors the depths of anesthesia by analyzing EEG to determine depth of consciousness. Similarly, the EEG can predict if medications will work for depression. Within a week of dosing, the EEG reflects changes which will predict whether meds will work by measuring frontal theta. There is no need for the client to wait six to eight weeks to see if medications will be effective. If the theta is lowered after meds, the medications will be effective.

Elsa Baer books act left right alpha asymmetry. Look for slow waves such as alpha in the left frontal and temporal areas. Joy works on the central strip first and ascertains the best reward frequency before moving on to frontal areas.

Deb Stokes mentioned that she has had good success with the LENS and Z score for inattention and mild cognitive impairment. No other attendees at this talk were using Z Score or LENS.

Bob Gurnee mentioned that he usually six different databases and has identified 11 subtypes of depression. He cautioned against overtraining the cingulate type of depression. He will not work without a qEEG. Joy encourage us to get as much data as possible before attempting to work with severely depressed clients and cautioned us against working outside of our scope of practice. She recommended that attendees not try to learn qEEG on their own. Get help from others in the qEEG field and start slowly.

Obtain an informed consent to speak with other treatment providers when working with severely depressed individuals. And include the family if possible regarding changes and side effects of the neurotherapy. Educate clients to also talk with their doctor is regarding neurofeedback experiences. Have them at obtain lists of side effects of all the meds they are on from their pharmacist and give this to you so that you can be on the lookout for changes and then send them back to their prescribing professional if they and you feel an adjustment is in order. Don’t make recommendations regarding medications, but refer them back to their doctor. Also keep an eye out for the potentiation effects of medications in addition to neurofeedback and work with the physician to educate them regarding the power of neurofeedback.

Metrics Beyond Power and Coherence
Thursday, Sept 3, 2009

Leader: Robert Thatcher, PhD rwthatcher@yahoo.com
Note Taker: Lexi Meinhold

- There are 6 basic modules in the brain – clusters of very dense connections and there is decreased connectivity amongst them.
- Power is the amount of synchrony of neurons in the cluster.
- Coherence is coupling between clusters/hubs. The scale is 0-1 and measures phase stability between clusters in the same time period.
- Phase shift is the sudden phase in phase between clusters. Ts measured by straightening phase out and then measuring on a 1-360 scale.
- First derivation – if there is no change then the measure is 0. This is known as phase lock.
- Phase reset is the shift in phase followed by phase locking. Longer phas lock and shorter locking generally indicates increased intelligence.
- Bursting activity if inhibiting cells leads to phase shift.
- The purpose is the recruitment of neurons to lock together to mediate activities in the brain.
- Phase lock and coherence have approximately .85 correlation.
- Joint time frequency analysis is the 1/f distribution of all frequencies nested in delta.
- The brain processes information on the falling side of phase.
- Phase shift puts cells at the top of the phase and they activate on the falling side.
- Basic drives will cause the brain to reorganize to meet these needs. Delta controls organization.
- The thalamus links theta to beta, delta to gamma, etc, cross frequency phase shift.
- Phase shift and phase lock occur in milliseconds.
- When expectations and perception agree there is an increase in excitation which converges on inhibitory cells, which in turn inhibit further excitation.
- There is less inhibition of inhibitory cells during novel experiences to aid in stimulation evaluation.
- Phase shift occurs almost always in the thalamus.
- Burst metrics measures how much activation there is in a given area of the brain.
- The insular cortex establishes homeostasis in the brain/body.

Neuromodulation for rTMS
Thursday, Sept 3, 2009

Leader: Martijn Arns martijn@brainclinics.com
Note taker: Randy Lyle

What is rTMS, how does it work, what is the potential?

A brief explanation of how it works was provided by Martijn. Coils, intensity of frequency, systemic in nature. Intervene in one area stimulate related networks. Safety was discussed esp. seizure. They rule out treatment if suspected. Several studies on Parkinson, mixed results. No effect on tremors so far. Motor activity has demonstrated some improvement. Good clinically significant results with combination rTMS/
psychotherapy in depression. Subgroup appears to require “booster” treatments to maintain the gains.

rTMS is an interventive strategy. It makes the brain do whatever you tell it to. Offered an explanation of treatment protocols and what has thus far been effective and with what, primarily depression at this point.

What is available in the US re: rTMS now that FDA has approved it for the treatment of depression? First, do not use the rTMS without qEEG to rule out paroxysmal activity. Several manufacturers. Compared tDSC. Briefly one is activating and makes the brain do what you want. tDSC is changing the resting state and still requires the brain to figure out what to do. Likely they will help different conditions. No research so far with Alzheimer’s. Some reports of odd responses with temporal lobe stimulation. E.g. out of body experiences, alien abductions, etc. Some report (around 15%) headache after treatment. Sleep improvement often occurs.

Harvard has only course for certification for rTMS and tDSC in US. Cost for rTMS machine ranges from $50,000 to $70,000. There is a cheaper model that will only inhibit that will cost around $10,000. Still only one company allowed to sell in the US.

Low Resolution Brain Electromagnetic Tomography (LORETA)

Saturday, Sept 5, 2009

Leader: Leslie Sherlin, PhD
lesliesherlin@mac.com

Note taker: Randy Lyle

How does LORETA change NFB? Not greatly when used in conjunction with Q etc. Still the same when doing LORETA guided feedback. How related to ICON? ICON reduces to eight regions/components. SO perhaps ICON will facilitate more rapid treatment. Advantage for use, assessment as to source and therefore related more specifically to symptom. Discussion of the how and what of ICON and LORETA and how they interact with each other and aid NFB.

Discussion of how LORETA is designed and how it is superior/inferior to MEG, MRI, etc. How is it using gamma? Would LORETA allow for the averaging of groups related to personality disorders?

Conversation about how one might design a variety of studies integrating LORETA and/or ICON or an integration. Is LORETA helpful or useful for diagnosis? Some anecdotal reporting of correlations and particular patterns seen through LORETA. Differences between LORETA, sLORETA and eLORETA?

Hypnotherapy and Neurofeedback-based Pain Management: Efficacy and Future Research

A conversation with Mark Jensen, Ph.D.

Roger H Riss, Psy.D.

Mark Jensen, PhD is a Professor, and Co-Director of the University of Washington Rehabilitation and Research Training Center on Aging with Disabilities. Established in 2008, the Center addresses challenges associated with physical disabilities due to spinal cord injury (SCI), multiple sclerosis (MS), Post-Polio Syndrome (PPS), and muscular dystrophy (MD), and is funded by the National Institute on Disability and Rehabilitation Research (NIDRR). Dr. Jensen is currently in the early stages of a federally funded research program to investigate efficacy of neurofeedback in management of chronic disability-related pain.

RHR: Dr. Jensen, I guess I’d like to start out by finding out a little bit about your work at University of Washington.

MJ: Sure. I’m a professor in the Department of Rehabilitation Medicine and my research focus is on pain management. My program of research is in symptom management. That’s the whole thing…and I’ve looked at, used, and evaluated classic psychological interventions like cognitive-behavioral therapy and; in the last 10 years; self-hypnosis training, both of which are very effective—or very effective for some people, less effective for others. About 5 or so years ago I began to look at neurofeedback for pain management.

RHR: Could you describe the setting in which you see and treat patients?

MJ: I’m primarily an academic… In the past I have seen patients in the clinic, but over the years I’ve moved more and more into research. So, my role is to develop interventions as a way to relieve suffering in groups of people rather than treating individual patients in the clinic. Both are very important, but it’s just I’m a researcher. So these days, I’m not in the clinic at all.

RHR: Are your research subjects recruited from a particular pain clinic or program at the Medical School?

MJ: Actually, our primary resource for research subjects are large federally funded research projects studying individuals with disabilities. So, we have a federally funded model system of spinal cord injury. We have a rehabilitation research and training center funded by the National Institute for Disability & Rehabilitation Research (NIDRR) in multiple sclerosis. We have a rehabilitation research and training center in ageing with disabilities funded by NIDRR. These studies have pools of individuals, hundreds of individuals, who participate in various research projects. If they have significant pain, for example, and have expressed a willingness and interest in participating in research, we would contact them and say, “Would you be interested in participating in a clinical trial to look at,” say, “cognitive-behavioral therapy” or “self-hypnosis training” or now “neurofeedback?”

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RHR: You said that approximately 5 years ago you became curious about the potential role that neurofeedback might play in pain management. Could you tell us a little bit about that journey and what led you to that question?

MJ: Sure. We were getting significant success with self-hypnosis training. And some patients say this changed their lives; they now had control over their pain, and it was fascinating. Yet there were subsets of patients who would say “this isn’t doing a thing for me.” So, there were quite variable responses. I heard, at a hypnosis meeting, John Gruzelier give a talk on using neurofeedback to improve creativity and performance in opera singers. And he described the neurofeedback and it was consistent with research, with which I was already familiar, that looked at the effects of hypnosis on EEG activity. I got the idea, while I was listening to him, that maybe we could use neurofeedback as a tool to train people who don’t respond well to hypnosis, to teach them how to get their mind into that hypnotic state so they could then be ready respond to hypnosis. So, I got some funding to look at that and to explore that. And I found that neurofeedback itself was providing relief, so I am now thinking, well, maybe some of these interventions like self-hypnosis and neurofeedback, even biofeedback, maybe even cognitive therapy have a similar underlying mechanism that is changing EEG activity. So, I just now got funding to look at that question, and that’s what we’re doing right now.

RHR: So, Dr. Gruzelier’s presentation led to the question “can neurofeedback be a bridge towards enhanced hypnotic susceptibility?” How did you begin to explore that question?

MJ: Well, I got the equipment, I got some training with EEG Institute, and then I saw 3 or 4 patients and they had different types of pain— they had variable response but I observed enough response to continue to pique my interest. In fact, interestingly, one patient who received combined hypnosis and neurofeedback got the best response. I was very intrigued by that. That’s also consistent with studies in the 1970s by Melzak and Perry. And so, I used that as pilot data—to get the funding for the current research project we are just beginning. Actually, we have one just starting this week—yesterday we started—and a second one we anticipate funding because we got a very good score.

RHR: Oh, excellent. So, does your 2007 “Journal of Neurotherapy” paper reflect the results of those first pilot study patients?

MJ: Pilot patients? No, actually that data was collected Carolyn Grierson, RN, in a series of reflex sympathetic dystrophy (RSD), or complex regional pain syndrome (CRPS) patients treated in her clinic. As part of my own learning process I interviewed her to talk about protocols to use, and she said she had this dataset, but she didn’t know how to write it up. I said, “Well, give us the data, and we’ll write an article.” So, no I haven’t published my own pilot study case studies yet. I publish 20 articles a year or so and that one is still on the backburner. I have other ones that have to get out. But also that was, I was just experimenting, so those case studies weren’t systematic, as systematic, but I still have the data, and I may publish those.

RHR: How did the effect sizes for pain relief with neurofeedback in that 2007 “Journal of Neurotherapy” paper compare the effect sizes you have seen for hypnosis and self-hypnosis interventions?

MJ: Well, with respect to pain, there are two types of responses. One is the immediate effect on experience of pain when you do hypnosis. And that’s a rather large effect size for most people, 80% to 90%. When you sit down and focus your attention and it lasts for several hours usually. Sometimes it comes right back, sometimes it lasts for days. But on average it lasts for several hours, so that is self-hypnosis as kind of an aspirin. Very effective as long as it lasts, but then the pain comes back.

RHR: Yes.

MJ: But then there’s a smaller subgroup of people for whom hypnosis seems to shift how their brain processes perception and pain information. For them, there’s a more or less permanent change in the experience of daily pain. I don’t want to call it a cure because the pain never goes away, but it is a substantial drop that is relatively permanently maintained. That happens in less than 50% of people. In our samples, it has happened as infrequently as 22% in some samples of patients with spinal cord injury related pain and as much as, I think it is 42% or 44%—I think 42%—in patients with pain related to cerebral palsy. So there are really two things that are happening; one is that hypnosis can make the brain shift in that subgroup of people and the other is that it can give people a skill that they can use on a daily basis and that’s the largest majority of people. And then you have on top of that people who seem to respond more to CDs—(CDs individualized to the patient, not off the shelf.)

RHR: Yes. I’ve had patients describe to me the CD as a way for them to carry my voice outside of the therapeutic session.

MJ: And there are people that are able to take your voice and forget the CD and just on their own do it. Many patients do both. So that’s a source of variability as well. Beyond pain relief, then, hypnosis will have effects on other variables—improved sleep, improved confidence, overall quality of life, general well-being—and almost everybody finds some benefit from self-hypnosis training outside of the pain issue. So when you ask the question, you know, what are the effect sizes, it really depends on which of these outcomes you are looking at. Similar considerations apply to neurofeedback outcomes.

RHR: Carolyn Grierson’s data suggested that CRPS patients receiving neurofeedback tended to demonstrate that first class of relief that you described—an immediate if transient reduction in pain intensity within the therapy session. Did you gain any impressions about long term persistence of pain relief in those patients?

MJ: Well, we did not formally study that question, but my recollection is that she had variable responses. What we plan to do next is investigate long-term change in pain levels with neurofeedback. In the next two years, we’re going to pilot 10 patients with refractory spinal cord injury pain and see what the long-term effects are in these patients. Now, that said, refractory spinal cord injury may be the most difficult pain problem to address. By contrast, the easiest pain problems to address are probably phantom limb pain and headache. Almost any treatment you use—you can fall off a log and help a patient improve his headaches or phantom limb pain. You know, whether you use EMG biofeedback or thermal biofeedback or self-hypnosis.
training or relaxation training or autogenic training those problems tend to get better.

**RHR:** Could you briefly describe CRPS and perhaps comment on its underlying mechanisms?

**MJ:** Yep. It’s a diagnosis based on a cluster of symptoms, and its onset usually involves onset of severe intractable pain in following a relatively minor injury. Often, pain may set in following a period of disuse due to casting or bracing of the limb, for example, when you sprain an ankle and you put it in a brace and you don’t use it... Symptoms include an increased sensitivity of the skin such that light brushing results in the experience of excruciating pain, out of proportion to the original injury. There are so-called atrophic changes where the skin in the painful area becomes blotchy and swollen, and it has a tendency to spread within the limb. It’s usually a limb, but can be other places. It can even spread to the other limb. A causal factor seems to be inactivity. That is, people who early on don’t move it, it seems to spread faster. In those who exercise and move the painful area, it tends to stop, or, if they get it early enough to make it disappear. Yet, movement is extremely painful. And, in terms of mechanism, nobody has the answer. It may be multiple mechanisms. For some, it may be mechanisms involving dysregulation at the level of the spinal cord, others more central dysregulation.

**RHR:** I’ve heard it described as a sympathetic activation issue, is that over simplistic?

**MJ:** Yes, that’s the old theory. That’s why they changed the name from reflex sympathetic dystrophy (RSD) to complex regional pain syndrome. RSD implied a sympathetic response. If that exists, it indicates a self-medication, a self-numbing so to speak, of the brain in response to pain. We’re really in speculation here, so don’t, you know… These are all things we want to start to examine in my research. How responsive is that problem to neurofeedback if you encourage… All of this is leading towards increasing their relative alpha, right? But if you do that, is the improvement in pain associated with that because theta decreases when you increase alpha? Is it because you got more alpha? Or is it because when you have more alpha you have less beta or some combination? Or different in different people? So, we need to… It’s fascinating! So, it’s fun to be in a field where there are so many unanswered questions.

**RHR:** Absolutely.

**MJ:** Because we want to know—it would be nice to know—what the primary changes because then you can target that. Is it just the decrease in theta? Then you can just decrease theta and forget about the other stuff. But maybe you have to decrease theta and at the same time increase alpha. Maybe increase theta in the motor cortex, but decrease it in the sensory cortex. So, a wave band may be adaptive in some areas and maladaptive in others.

**RHR:** as, for example, when parietal alpha is increased during migraine headache pain?

**MJ:** Yep, yep.

**RHR:** OK. In the 2007 study, neurofeedback interventions were tailored to the individual. This is very consistent with your previous comments that we’re not going to necessarily find a consistent pattern associated with that because theta decreases when you increase alpha? Is it because you got more alpha? Or is it because when you have more alpha you have less beta or some combination? Or different in different people? So, we need to… It’s fascinating! So, it’s fun to be in a field where there are so many unanswered questions.

**RHR:** I’ve sometimes wondered whether that increased slow wave activity reflected a self-medication, a self-numbing, so to speak, of the brain in response to pain.

**MJ:** Yes, that’s a reasonable hypothesis. The hypothesis that’s been put out there, and again it’s controversial, is that the problem is in the thalamus and the thalamus, for example, following a spinal cord injury, might get inadequate input, and so it starts throbbing theta throughout the brain. And that interrupts pain inhibition processes that the cortex engages in. So, it’s like, you know, you need to have the people build a house, but then you gas them with sedation and they can’t build a house. But, you know...
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"Hey, look, I hurt all the time," that can represent a large number of different problems. Two people experiencing the same pain, one may be a thalamic dysregulation, one may be nociception from a peripheral injury that’s inflamed. One may be related to a childhood trauma and a brain that’s overactive and predisposed. I mean, so, I suspect the variability in responding has to do with the heterogeneity of any sample of persons with chronic pain even if they have the same “diagnosis.” Like persons with headache or low back pain or even complex regional pain syndrome—everybody’s pain is way different.

**RHR:** Interesting!

**MJ:** Right. Pain is fruit—one time I’d have an orange and one time I’d have an apple.

**RHR:** Can you discuss the role of emotional distress in maintaining or increasing susceptibility to pain?

**MJ:** Well, there are theories, but no one model in my view. My belief is that emotions and prior trauma can play a role in some people, but it doesn’t necessarily play a role in all people. Pain involves many networks of the brain. So, it goes back to this idea that for some a childhood trauma, which sensitizes that person’s brain maybe because they have a genetic predisposition to respond in a certain way to trauma, sensitizes some people and makes them more vulnerable to develop chronic pain. I believe that’s true, I don’t think that’s been proven, but I believe it’s true. But it’s not—I can’t see a pain diagnosis that has a single explanation. I think it varies, again, from person to person.

**RHR:** When we treat our pain patients using neurofeedback or hypnosis, is it your impression that we are in fact treating pain or are we treating the associated emotional response to pain?

**MJ:** The answer is a resounding yes!

**RHR:** To both!?

**MJ:** And in different people.

**RHR:** OK.

**MJ:** For some people, it is the sensory input which is the driving force in cortical activity. For others, it’s an emotional reaction that is paramount. What you need… You need to do a careful evaluation and develop hypotheses. Is this person’s experience of pain linked mostly to the thoughts they have about the pain? If so, that leads to hypnosis and cognitive restructuring. Is it associated with her emotional response? If so, that leads to hypnosis, but with a different focus, and also to neurofeedback with a focus, perhaps, on the ACC and training calming activities and perhaps as well to cognitive restructuring. Is it mostly a sensory issue, that is, the sensory cortex? If so, maybe then you’d want to focus mostly on self-hypnosis and neurofeedback. Is pain primarily a peripheral issue? Or is it due to problems in the spinal cord? If so, then you might want to teach a person to cope more effectively using an acceptance-based approach. The clinician treating the patient with pain, I think, in order to be effective must be trained in the use of cognitive restructuring, self-hypnosis training, and neurofeedback. Most clinicians—it’s a rare clinician who has all that training, and I strongly encourage any clinician who’s interested in treating patients with pain to get the training they need to really understand the patient and treat the whole patient and not just focus on one component.

**RHR:** Can you tell us more about your current research plan?

**MJ:** Sure. What we’re doing is we’re comparing EEG pattern activity in individuals with refractory neuropathic pain due to spinal cord injury—to people with spinal cord injury without pain, and to healthy controls. We are going to develop a single protocol for neurofeedback and, based on what we find in the pattern, see if we can help patients learn to shift their brain from one that looks like it’s hurting to one that looks like it’s comfortable and see what that does to peoples’ experience. We plan to pilot that in 10 patients over the next 2 years. If the second study is funded, and we are cautiously optimistic because we got an excellent priority score from NIH, we will compare the effects of a single session of neurofeedback, hypnosis, and transcranial direct stimulation and two controlled conditions; one a sham transcranial direct stimulation and second a simple meditation on the activity and reports of pain. The goal here is to see if there’s a single underlying factor that is associated with the three treatments that are thought to modulate each activity and to see if we can identify a pattern of change in EEG associated with decreased pain. The main reason was to see if we can identify the pattern or patterns to target in neurofeedback training.

**RHR:** Yes, If we were specifically exploring the hypothesis that neurofeedback training may assist an individual in increasing hypnotic susceptibility, we might speculate that alpha-theta training would be the place to go. Yet, in your pilot work so far, you’ve seen that other forms of training may be effective.

**MJ:** Yes, you know, I don’t know that there’s a single hypnotic state. My view is that there’s a hypnotic “ability.” That there’s ability to shift brain states, to move into sleep, to become relaxed in the face of a middle of a storm, to become focused or to spread your focus. I understand that this is not necessarily…that there’s extreme controversy in the hypnosis field about what hypnosis is, and I think what it is depends on who it is you’re talking to. It’s like, what is love? It’s hard to say love has to be this. It’s probably different for different people. But I think what neurofeedback does is it helps people learn to shift brain states. That, in my view, that’s hypnosis. It’s not—again—it’s not necessarily what people in the hypnosis field would think. But it’s kind of where I am. I think it’s kind of a skill thing.

**RHR:** I understand that our colleague Leslie Sherlin is playing a role this study.

**MJ:** Yes, he’s a co-investigator in the study. His role is to bring his expertise in EEG data acquisition analysis and neurofeedback to help guide us.

**RHR:** Is it your impression that neurofeedback and hypnosis likely impact pain at the same level? Or are they complementary, and addressing different aspects of pain management?

**MJ:** Yes, I think they work synergistically, that each works with the other one to help the patient “get it.” A person who’s struggling with hypnosis and they get neurofeedback and they find that state and they go, “Oh! This is where I’m heading.” Once you get somewhere, it’s easier to get back there. Or a person who is using neurofeedback may be kind of dependent on that feedback; but if they can learn self-hypnosis strategies for shifting brain states on their own without the machine, they go, “Oh! I can do this on my own at home!”.
RHR: Sure. With regard to self-hypnosis, is there a training resource that you would direct clinicians towards?

MJ: The Society of Clinical Experimental Hypnosis and The American Society of Clinical Hypnosis both put on excellent training workshops. Joseph Barber has a book out on using hypnosis for chronic pain management. My colleague, Dave Patterson, is about to publish a book. I anticipate… it’s on my list of things to do is to write a very easy manual for using hypnosis for chronic pain. You know, when am I going to find the time to do that? But it’s on the list. It’ll happen… things that get on my list eventually happen. But those are the resources. My belief is that hypnosis without neurofeedback and neurofeedback without hypnosis are less effective than the two combined. So, I think a neurofeedback clinician should learn hypnosis and hypnosis clinician should learn neurofeedback. I don’t know if that’s true, but I believe that’s true.

RHR: Well, doctor, thank you so much for a very interesting discussion, and I’m sure our readers are going to be excited to hear about what you have planned. Do you have any other closing thoughts or comments?

MJ: You know, I just think that my view is that our work field, both as clinicians and researchers, is very honorable. We’re working hard to help decrease suffering in the world, and we probably don’t take enough time to pat each other and ourselves on the back about the good that we’re doing.

RHR: Thank you so much.

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CHADD Update

On Friday October 9, President Tom Collura and Executive Director Cynthia Kerson were invited to a lunch with the leaders of CHADD, and their Professional Advisory Board (PAB). We were hosted by CHADD Chief Executive Officer Clarke Ross and PAB president Ann Teeter Ellison. Included in the meeting were 18 CHADD Board and PAB members, and Drs. Eugene Arnold and Nicholas Lofthouse, of Ohio State University. While the meeting was cordial, the intent was to query the ISNR representatives on several critical and pointed issues related to the acceptance of neurofeedback in the treatment of AD/HD. Among the topics given priority were the criteria and status of acceptance standards for neurofeedback as an efficacious treatment, the importance of sham feedback in placebo-controlled blind studies, the status of practitioner qualifications and licensure, and the availability of neurofeedback equipment in the general public. ISNR took the position that neurofeedback has been shown to be an efficacious and specific treatment modality for AD/HD, and that it is a complement to existing methods including medication, behavior therapy, and other forms of therapy. The CHADD group was generally receptive, and curious about the state of QEEG and treatment protocols. They appear to not be well informed regarding the status of QEEG-based treatment, and the fact that neurofeedback is largely a “diagnosis-free” method, that treats underlying brain dysfunction, not the diagnosis.

As you may already know, ISNR has been invited to view the CHADD position statement on neurofeedback, and to propose revisions thereof. And, we have also been invited to submit an article on how to evaluate and select a potential neurofeedback provider to be published in either Attitudes or ADDitudes. CHADD intends to publish the article along with a statement that CHADD does not recommend neurofeedback, but wishes to inform families who may want to consider it as an option. While many of the CHADD Board and PAB members were enthusiastic and supportive, there continues to be a voice that indicates that neurofeedback should not be recommended at all, and that it is largely unsupported by research. ISNR expressed the opinion that in individual as well as collective studies and experience, that neurofeedback is surely effective and should be recommended, even in the absence of a preponderance of matched-group, double-blind, placebo-controlled studies. ISNR expressed the desire to have CHADD help the ISNR to plan and conduct such studies, including possibly providing a channel for experimental subjects to be recruited through CHADD. The Research Foundation’s consortium program is being considered as the mechanism for this collaboration.

Drs. Arnold and Lofthouse also were supportive, and are interested in working with ISNR to both help ISNR design and plan research studies, as well as having ISNR help them to plan the next phase of their research. Their current work is focusing on demonstrating that there is a treatment effect in neurofeedback, and that it is dose-dependent, i.e. affected by frequency of training sessions. They look to ISNR to help move forward with alternative choices of protocols and instruments, for further work. Drs Arnold and Lofthouse have asked to present the results of this pilot study at our conference in Denver.

This is a turning point that is timely and important. Establishing neurofeedback as a CHADD-sanctioned viable modality for AD/HD will benefit both organizations’ members. CHADD members will find an alternative to behavioral and medication treatments and ISNR members will find a welcoming new client base. But, there is much more work to be done and ISNR is committed to doing its part.

Reported 10/14/09 by Thomas F. Collura, Ph.D.
QEEG-T, President, ISNR and Cynthia Kerson, PhD, BCIA-EEG Executive Director
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