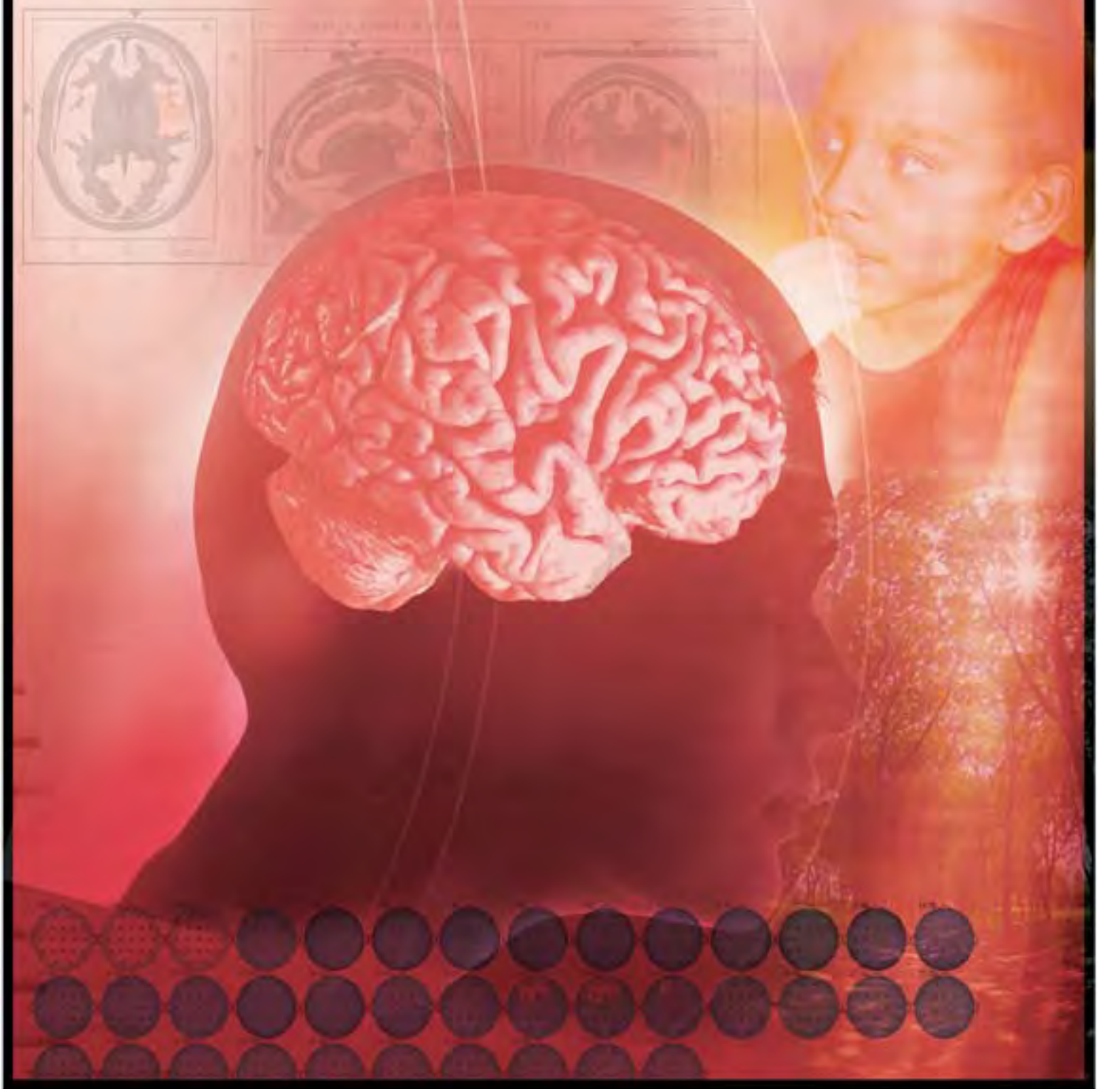
NEURO CONNECTIONS Newsletter

Winter 2011

A joint newsletter from the
& the aapb Neurofeedback Division



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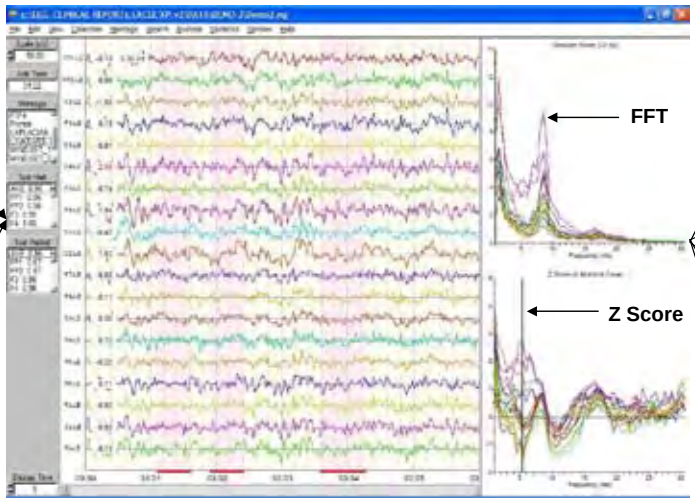

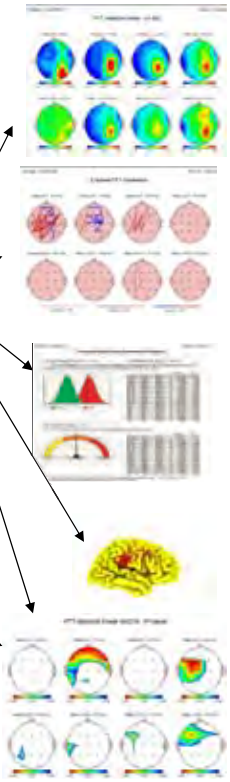

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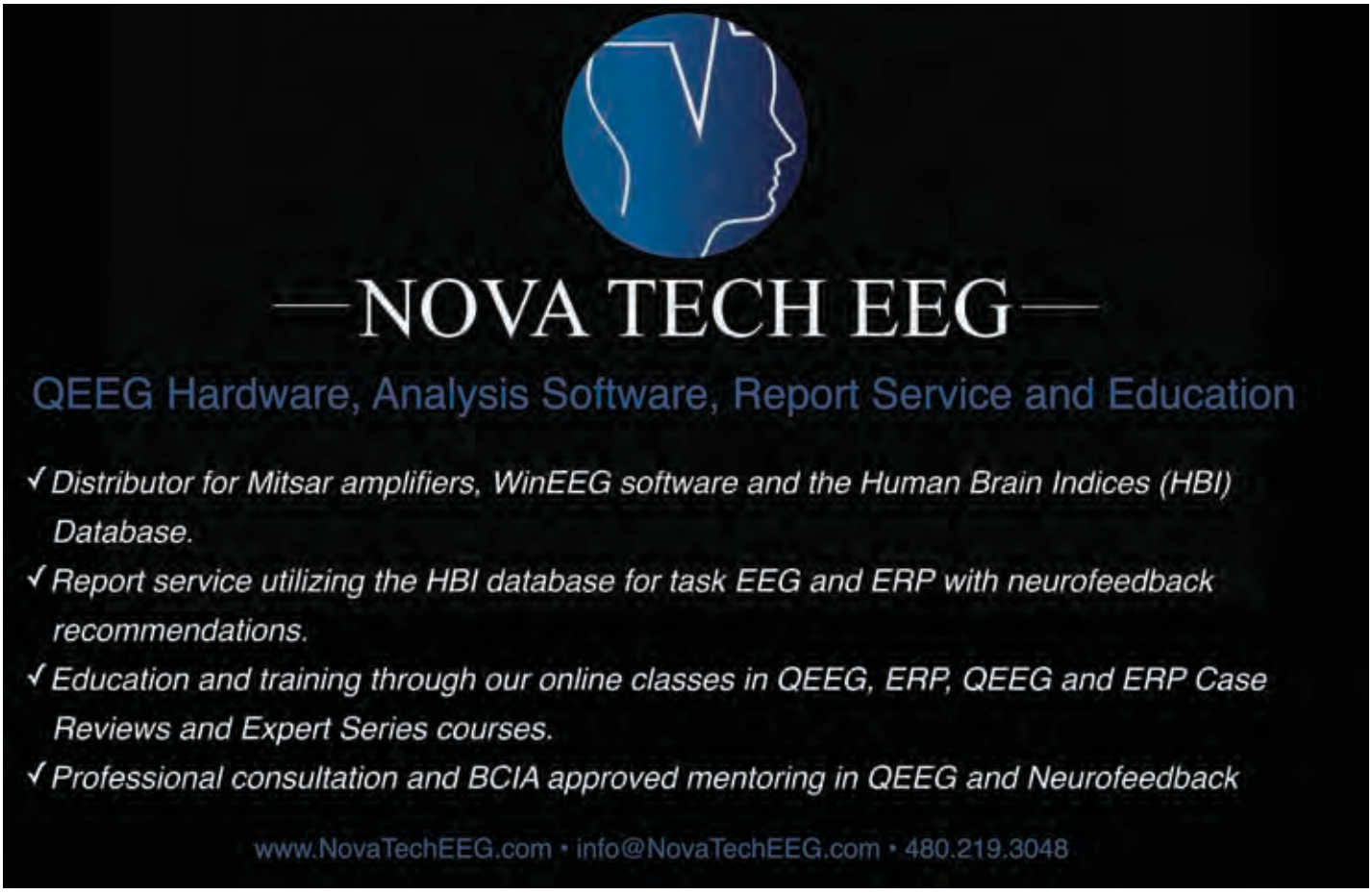

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NeuroConnections is published four times a year and will consider all materials pertaining to the practice and/or promotion of neurofeedback.

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ISSN 2151-6987 (print)
ISSN 2151-6995 (online)

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Letter from ISNR President

Richard E. Davis, MS

Six weeks have passed since our nineteenth annual conference in Carefree, Arizona. This year's conference was the second best attended in ISNR's history, with over 475 attendees. The diversity of presentations and workshops continues to make our conference one of the best in applied neuroscience. I would like to thank the Conference Committee, Conference Committee Chair, Leslie Sherlin, and Conference Coordinator, Ann Marie Horvat, for their commitment and hard work in producing this outstanding event. They created a venue that was educational, entertaining, relaxing, and esthetically pleasing. I hope all of our members are planning to attend next year's conference in Orlando, Florida which will be held on September 20-23, 2012. I am anxious to see how next year's conference tops this year's.

Both the opening and closing presidential remarks at this year's conference addressed the changes that ISNR has been undergoing. Our professional society is experiencing a major transition to accommodate the growth and maturity of the organization. In May of this year, ISNR contracted with the Non-Profit Center of LaSalle University's School of Business to fill our Interim Executive Director position. This Center offers a unique service to non-profit organizations such as ours by providing a combination of experienced personnel to serve in interim executive director roles as well as expert organizational consultation. Our Interim Executive Director, Karen Forbes, has been using her non-profit administration experience to help the organization re-structure its accounting/financial operations, policy and procedures, and administrative/organizational processes. We are presently undergoing a voluntary financial audit and are changing the way that administrative processes are handled. All of this should create a more efficient organization that can better manage the growth we are experiencing as well as position ISNR for future expansion in both membership and member services.

ISNR currently is in the process of recruiting a permanent Executive Director, with a hiring target date of December 1, 2011 and January 2, 2012 start date. We will keep the membership updated on the progress of our hiring efforts. See the ISNR website for the position description and recruitment announcement. We welcome your efforts to help us recruit the best possible candidates for this very important position within our organization.

By the time you receive this newsletter, the new ISNR website should be up and running. Leslie Sherlin previewed the new look during his opening comments at the recent conference. The new website has a crisp, clean look, offers a new shopping cart, and several functions not available on the old site. A special new web service addition, also previewed at the conference, is the streaming videos of various workshops and presentations from past conferences. A subscription is available for accessing these videos that provide a great educational and training opportunity for members.

During the conference, a call was put out for new members, with the goal of exceeding a total membership of 1,000 for the first time. This effort resulted in ISNR reaching 1006 total members by the Saturday night Banquet; quite a milestone for the organization. We now hope to achieve a twenty percent increase in membership over the next year. It would be great to have over 1200 members by conference time this next year. Much of the recent growth has been through Institutional

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Letter from AAPB NFB DIV President

Siegfried Othmer, PhD

This year's ISNR meeting seemed to have more invited speakers who were comfortable talking neurofeedback. In the past, one had the feeling that some presenters were there mainly to collect their speaking fees, and were not really prepared to engage with us on our core assumptions. There is a rising tide in the neurosciences that is lifting all boats, even ours. The conversation is shifting toward a language of networks, of structural and functional connectivity, as the key issue in psychopathology, and toward neuromodulation as a strategy for functional recovery. And there we are, having occupied that space already for some decades.

What has allowed this shift to occur—and it is really just getting underway—is the fact that the discussion is taking place on the rather dry ground of neurophysiology as opposed to our historical battleground of clinical claims. The latter exposes us to a surround of cat's claws that inhibit progress. The contrast between these two frontiers was dramatically on view one day at the conference, when in quick succession we had a presentation by Nick Lofthouse on plans at Ohio State for yet another 'definitive' study of neurofeedback for ADHD, a presentation by Andrew Hill on physiological changes with neurofeedback, and a presentation by Martijn Arns on using physiological measures to judge outcomes for ADHD and depression.

Andrew Hill's study, which tracked Event-Related Potential (ERP) data along with Event-Related Spectral Perturbations (ERSP) through training with some of our favorite old protocols for ADHD, for com-

ISNR Mission Statement

To promote excellence in clinical practice, educational applications, and research in applied neuroscience in order to better understand and enhance brain function. Our objectives are:

- Improve lives through neurofeedback and other brain regulation modalities
- Encourage understanding of brain physiology and its impact on behavior
- Promote scientific research and peer-reviewed publications
- Provide information resources for the public and professionals
- Develop clinical and ethical guidelines for the practice of applied neuroscience

AAPB Neurofeedback Division Mission Statement

To improve human welfare through the pursuit of its goals. The specific goals are:

- The encouragement and improvement of scientific research and clinical applications of EEG technology and neurofeedback.
- The promotion of high standards of professional practice, peer review, ethics, and education in neurofeedback.
- The promotion of neurofeedback and the dissemination of information to the public about neurofeedback.
- The division is organized for the purpose of carrying on educational and scientific objectives and is not to be operated for profit.

parison with sham training, demonstrated that physiological measures can readily distinguish active training from sham. Functional changes with training were tracked with the Lateralized Attention Network Test (LANT) of Michael Posner. The results of functional testing were nothing to write home about, but then the training only involved five sessions and the trainees were normally functioning at the outset. Significantly, the physiological measures were able to distinguish between the different protocols being used, which were our favorite old C3beta and C4SMR, along with C3SMR and sham.

With respect to the lukewarm results of the functional testing, I cannot resist interrupting the narrative to note that the whole point of having several protocols to choose from was to match the protocol to the client. Research designs invariably treat the different cohorts as homogeneous, a wildly inappropriate assumption. As soon as individualization of protocols is allowed, then it does not take long to discover that responses to training are sometimes highly specific in terms of reinforcement frequency as well. One of the key differences between the clinical world and the research world is that the astute clinician does not continue to beat a dead horse, whereas the researcher remains wired to his original research design. If clinical success means abandoning the assumption of static protocols, then the clinician loses no sleep over the matter. But back to the story.

The surprising finding was that changes in ERPs started showing up even in the first session, and were confirmed in the fifth session. Changes in ERPs and ERSPs distinguished training groups from the sham group. Effects on ERSPs were lateralized, being stronger on the training side. Resting eyes-closed band amplitudes in theta, alpha, SMR, and beta all distinguished between the active and the sham groups.

The study implies that physiological measures can readily distinguish active training from sham. This, it seems to me, disposes of the placebo argument wholesale. Lasting effects are seen which differentiate protocols from each other, and collectively differentiate them from sham. Why then do we still offer the greatest deference to those who would still, at this late stage, breathe life into the placebo hypothesis of neurofeedback?

So we come next to the proposal out of Ohio State to perform the definitive sham-controlled study of neurofeedback for ADHD, and many in our field are still in fibrillation about the prospect of having our collective fate tied to the outcome of a single such study. Now Nick Lofthouse certainly appears to be a competent, well-meaning researcher. But what is the best possible outcome of such an enterprise? It is the finding that EEG feedback, done in the traditional manner, is indeed better than doing nothing. This will hardly be enough to move the NIH off its pharmaco-centric posture. A lot of effort is being mounted here for small beer.

What if we took the approach of arguing the case for EEG neurofeedback in a physiological frame? The basic argument that physiological function can be enduringly altered with feedback on physiological variables has by now been very well established by the biofeedback community. Is the EEG among the physiological variables that are suitable for such training? All of the EEG feedback studies collectively make the case that it is. Now most biofeedback modes are aiming at system functioning rather than specific diagnoses, most particularly the auto-regulation of the autonomic nervous system. Are matters different in EEG feedback? They can be, of course, as in the targeting of something so specific as dyslexia or auditory processing disorder. But that is not where we have distinguished ourselves to date, by and large. In most current applications of EEG feedback, the benefit derived with respect to the discrete disorders is best explained in terms of better functioning of core regulatory systems, i.e. at the systems level. Here we are referring to improved regulation of tonic arousal level, to more appropriate regulation of affect, to better autonomic balance, to enhanced cerebral stability, to appropriate interoception, to improved executive function, etc.

And how is the cause of improved functioning at the systems level served by EEG feedback? A good working hypothesis is that we are directly affecting the functional connectivity of our resting state networks. This is readily testable. In support of this hypothesis one could already offer all of the evidence that neurofeedback—by various approaches—dramatically alters, and generally improves, coherence relationships within cortex. This occurs irrespective of whether or not coherence relationships are explicitly targeted in the training. This finding is neither tied to diagnosis nor to disorder. It is not tied to level of initial deficit. Most notably, it is not tied to protocol. Some protocols indeed work better than others, but those that are effective clinically can all be shown to affect coherence relationships favorably.

This means that group studies are not actually needed to prove the essential claims of EEG feedback. It means that all of the case data accumulated by clinicians to date can collectively make the case for the core hypothesis. This is in effect what has happened. Our evolving convictions about what is possible with neurofeedback have been consolidated largely without the benefit of group studies with standard controlled designs. Likewise protocol development has progressed—where it has done so—entirely on the basis of individual case observations.

This is not a flaw. Protocol refinement is really better served by working with individuals over time, with intra-individual variability as the most significant confound. The question of optimizing reward frequencies, for example, has to be examined within individuals rather than via group studies. And protocol refinement is in the best of hands with real clinicians, as opposed to naïve researchers recruited from academia. The research world has not come to terms with this, so in that respect we have been fortunate in not having the NIH regiment the development of this field over the past few decades.

The core claim, then, is that neurofeedback can positively influence any brain function that is governed in any fashion by our resting state networks, provided of course that structural connectivity networks are sufficiently intact to support the change. Again, all of the clinical work to date can be brought to bear in support of this proposition.

With this background we turn to the presentation by Martijn Arns on the value of using physiological variables such as ERPs to document change with neurofeedback for ADHD and depression. No quarrel there from this quarter. Now with regard to the work with depression, Arns referred to results achieved with repetitive Transcranial Magnetic Stimulation (rTMS) rather than neurofeedback. He motivated this by declaring that neurofeedback was ineffective in application to depression. I was startled by this declaration and took the matter up with him after the talk. He pointed to the lack of published group studies in his defense. I counter-argued that he was making an affirmative statement, which required its own affirmative evidence. As we are constantly reminded, absence of evidence is not evidence of absence. The proper statement would have been to declare the matter as still unsettled.

Now on the basis of the 'grand hypothesis' above, one would certainly expect neurofeedback to be effective for depression. If it is not, then that would indeed be fatal to the maximal position. So what is the evidence in this regard? It turns out that the first reports of benefit of standard SMR/beta neurofeedback for depression came out of Sterman's laboratory, where UCLA graduate students recruited for the training routinely reported an easing of depressive symptoms. Sterman never made much of this because that had not been part of the formal hypothesis, but one might also argue that the evidence is more robust for not having been looked for at the outset. Margaret Ayers certainly saw the recovery from depression in connection with her work with TBI. We saw benefit for depression in our early work as well, with a slight modification of the Ayers protocol, and we started saying so openly in the 1990-1 timeframe.

This declaration was met with considerable skepticism and even hostility at the time. This was when Lubar was still making the case that neurofeedback was for ADHD and not for anxiety and depression. Not long

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Letter from AAPB NFB DIV President

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thereafter, Peter Rosenfeld gave the idea of neurofeedback for depression scientific respectability with his hemispheric asymmetry protocol, modeled after Davidson's work on the hemispheric bias with respect to approach and withdrawal. The training didn't really work very well in the general case, so the technique has gradually faded from view. That development might in turn have dampened the expectation that neurofeedback would be helpful for depression. If the 'scientific' protocol didn't work very well, then what hope was there?

Meanwhile, however, the early approaches just kept on working. More recently, Cory Hammond has come out with a depression protocol that looks remarkably like what we were doing twenty years ago—namely beta1 training with a frontal or pre-frontal, left-hemisphere bias. That stands as confirmation of a sort for our early protocols. For our part, we realized that the most intractable cases of depression, namely agitated depression and suicidal depression, required mainly right-hemisphere training, which took us some years to develop. More recently, data on veterans with PTSD shows depression scores being cut in half within ~six sessions in close to 80% of trainees. Suicidality has remitted in nearly all cases to date. Within the clinical community, depression is not being discussed as a thing apart. It is not either more or less intractable than other conditions encountered in EEG training. So depression is not a counterexample to the more inclusive vision of neurofeedback as potentially aiding all conditions grounded in the disregulation of our resting state networks.

In reflection on the ISNR Conference, I was most riveted on the invited talks, but when it comes to progress within our discipline, the critical contributions are being made by the people who have committed themselves to this field. This field has been built, and continues to be built, largely on the contributions of scientist/practitioners and their technical support teams, the developers and manufacturers. The discipline has been built from the bottom up, and it is moving into a 'crowd-sourcing' phase, with the enlargement of the central core of contributors. This is a very healthy development, and there is no need for the NIH to come in for a do-over and a re-write.

That is not to say that there isn't a useful role that the NIH might play. In his review of imaging technologies in *Science* in 2009, Karl Friston lamented that by their very nature as resting state networks, these could not be readily subjected to experimental manipulation without disruption. One is reminded of the dilemma that a quantum mechanical system cannot be probed without affecting

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ISNR President

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Memberships in Europe, Australia, and Mexico. Because of this increase in our international members, the ISNR Board of Directors is working to establish an Advisory Committee made up of international members who will assist John Davis, International Member at Large, to explore and make recommendations to the Board on how to best represent the increased membership interests in these countries.

Changes have also come about within the structure of the *Journal of Neurotherapy*. Martijn Arns, PhD and Randy Lyle, PhD have completed their term as JN Editors and have handed off the duties to Adam Crane, PhD, and Efthymios Angelakis, PhD Please support our new editors by suggesting or writing manuscripts to submit to the journal. A tremendous thank you to Martijn and Randy for the outstanding job they did as immediate past editors, especially for their success in keeping the journal current with full content. Their efforts should allow us to secure *Medline* indexing when we apply in the near future.

The 2012 Board of Directors was installed at the conference. New Board Members include Leslie Sherlin, past president; Randy Lyle, president elect; Anne Stevens, treasurer; Sarah Prinsloo, secretary; Nolan White, sergeant at arms; Rex Cannon, member at large; Rob Coben, member at large and John Davis, international member at large. Sarah, Rob, John, and Randy were voted in as the new members joining the Board. Randy and Rob have served on the Board in previous years, but John and Sarah are serving for the first time. I am looking forward to working with this excellent group of people.

In addition to hiring and training a new Executive Director, major plans for the 2012 Board include an extensive strategic planning process to take place at the Board retreat this spring. During that time, work will be done to strengthen the committees and add committee members. I hope many of you will consider joining an ISNR committee and becoming active in your professional organization.

Contact me with inquiries on how you can contribute a bit of your time and ideas to our association. Also, don't forget to donate to the ISNR Research Foundation. By donating time to ISNR and funds to the Research Foundation, you can help support the field that supports you. We all need to invest in ourselves by investing in ISNR.

Richard E. Davis, MS

Letter from ISNR Co-Editor

Merlyn Hurd, PhD

Dear All,

Welcome to the winter 2011 edition of *NeuroConnections.*

This edition is focusing on mTBI, a condition that is often misdiagnosed or not even diagnosed. One of the surprising findings using Neuroguide database and SKIL database has been the number of ADHD children and adults that were mTBI candidates. Yet such a finding is rarely talked about or even researched. When you have a client with an undiagnosed mTBI and their history keeps pointing to that possibility, the decision as to the approach one will use regarding this subject is onerous and causes many questions. I believe we need to become as aware of the markers, the impact of mTBI on the treatment and the client's life. Recently, two different young clients were trained by me. One had had two frontal facial hits in a team sport that is known for such accidents. Fast moving objects in a team sport can be uncontrolled. The second was a performer who had been hit during a performance. The first young woman had immediately sought neurofeedback and 14 sessions later was back in the team sport with all faculties intact. The second had been to many neurologists, psychiatrists, and internists, with little agreement as to what were causing her headaches, poor sleeping, cognitive dysfunction, fatigue and inability to work. One neurologist had told her to go home and for six weeks stay in a darken room with no stimulation, which she did. Oddly, there wasn't any improvement. These two clients are not unknown to us and it grieves one to see the second type of client who has now, 1 year later, begun neurofeedback, Neurofield and LENS and is doing exceptionally well. Still not back to work, yet headaches are down to .05, fatigue is reduced, sleeping has improved, brain fog is reduced and she is beginning to "be more like I used to be". So, be sure to read Dr. Thatcher's excellent article on what is mTBI, what are the discriminate markers, what are the connectivity deviations and much, much more. This is a must read article when you work with mTBI clients.

Dr. Koberda's article gives us guidance in the detection of mTBI. You will learn even more about what we need to look for. Dr. Ochs has gifted us with a strong article

on the use of Low Energy Neurofeedback system for mTBI. This article will help you to understand the LENS system and how it works, what the impact on the mTBI brain is and how it works so rapidly to bring about the healing necessary for the restoration of faculties. The first client I talked about above was trained only with LENS. The second is being trained with a number of Neurofeedback instruments and software. Presently, to address the headaches HEG has been used. Neurofeedback, Neurofield and LORETA are all part of this client's training. Dr. Carmen has written an article for us on the use of HEG for mTBI and it will be in the Summer edition.

Since depression is often a part of the disorders with mTBI, Dr. Hunt-Harper and Dr. O'Brien have provided us case studies of treating treatment resistant depression clients. This is an article you will find most helpful also, especially, when such clients often make us dive for the books and call other experts to find the way to "get to health for the client." And the Thompson's article on combining biofeedback and neurofeedback for a Parkinson's patient reminds us to consider a more comprehensive treatment plan for any diagnosis or set of symptoms.

Finally, welcome back the Ride the Waves brain teaser. Be sure to take a look at the images and write up your impressions and send them to either myself (merlyn@nyneurofeedback.com) or Cynthia Kerson, Managing Editor. We will publish the explanations in the next edition of NeuroConnections.

Winter is here and it is time to have fun in the snow!

Sincerely,

Merlyn Hurd, PhD, BCN Fellow

Letter from AAPB Co-Editor

Roger Riss, PhD

With the exception of headache, no neurological disorder occurs more frequently than concussion, or mild traumatic brain injury (mTBI). If a 2004 World Health Organization estimate of 600 cases per 100,000 population is extrapolated to reflect the 2011 US population, this would equate to more than 1.8 million new injuries annually in the United States alone (Cassidy et al., 2004). Among returning veterans, The Pentagon has estimated that about 115,000 soldiers have experienced mild TBI, while the RAND Corporation study, Invisible Wounds of War, suggests the much higher number of 400,000 total TBIs, the majority of which are classified as mild.

Most individuals experiencing mTBI enjoy functional recovery within three to 12 months without extensive therapy (Carroll et al., 2004). Nonetheless, there is a general agreement that approximately 15 to 20% of mTBI patients demonstrate persistent functional disability well beyond the three-month acute recovery window. Even non-hospitalized mTBI patients who return to work quickly are not without residual symptoms. Among a group of young men who returned to work within five days of injury, 20% continued to report symptoms at three months, including memory, concentration

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AAPB Co-Editor
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and coping difficulties at work (Wrightson & Gronwall, 1981). By six months post injury, 30% or more have not yet returned to gainful employment (Boake et al., 2005). The vocational implications of delayed recovery can be devastating. According to the American College of Occupational and Environmental Medicine, by the time that an injured worker has been away from work for six months, the odds for them to ever return to full employment drop to 50-50.

Most individuals experiencing mTBI enjoy functional recovery within three to 12 months without extensive therapy.

Medical costs associated with mild brain injury have been estimated at $17 billion dollars per year, yet the value of that care has been brought into question. A recent World Health Organization task force (Carroll, et al., 2004) reported limited evidence that the therapies currently offered mild traumatic brain injury survivors are associated with improved function or vocational outcomes. This pessimistic view expressed by WHO task force members stands in contrast with the life changing outcomes we in the neurotherapy field have come to expect in our own clinics. Lack of recognition for neurotherapy as a first line treatment for mTBI impedes our ability to serve this deserving population, serving as a reminder that clinical success alone will not gain attention from decision makers unless supported by an incremental body of outcomes research. Hats off to the contributors of this issue, for their efforts, as both scientists and practitioners, to meet to this need.

Roger Riss, PhD

Boake, C., McCauley, S. R., Pedroza, C., Levin, H. S., Brown, S. A., & Brundage, S. I. (2005). Lost productive work time after mild to moderate traumatic brain injury with and without hospitalization. *Neurosurgery, 56*(5), 994-1003; discussion 1994-1003.

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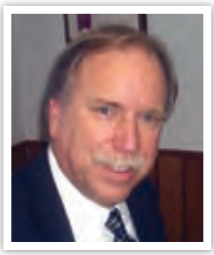
Wrightson, P., & Gronwall, D. (1981). Time off work and symptoms after minor head injury. *Injury, 12*(6), 445-454.

Letter from AAPB ED

Is Society Ready for Health Care Alternatives?



David L. Stumph

It never ceases to amaze me that it is so hard to acquire insurance coverage for treatments that don't have horrifying side effects. Why is this so difficult? I have a theory that can be summed up in one word: laziness.

Today's society is so reliant on drug therapies to address every type of disorder that we (collectively) have become lazy in the way we address our own health care needs. We take the easy way out. Since insurance carriers cover what has become known in society as traditional, high cost treatment modalities and, for the most part, ignore the long-lasting, lower cost benefits that biofeedback and neurofeedback deliver, society simply goes along with the accepted norm. One needs to look no further than to AAPB's publication, *Evidence-Based Practice in Biofeedback and Neurofeedback* for research and data demonstrating the effectiveness of these non-invasive therapies.

It is always interesting to me to see a 60-second pharmaceutical ad on television that includes 30 seconds of warnings about the side effects. And if you listen carefully to the warnings, they can be quite terrifying, in fact possibly worse than the disorder the drug is prescribed to treat. As a result of such ad campaigns, I believe that society is starting to listen more carefully and is finally starting to take notice.

As society becomes more aware of the hazards of drug therapies, thanks in large part to the pharmaceutical advertising campaigns, coupled with the ever increasing cost of insurance premiums, sometimes as much as 20% each year, I believe that society will become disillusioned with the entire health care system in its current context, particularly in the United States which has always been touted as the best in the world. In a recent study, however, published by the World Health Organization, the US health system only ranked 37, well behind France, Italy, Spain, Austria, Japan, and a host of others.

My point is that, while it is likely to take time, change is in the air. Interestingly, that change is being driven by insurance carriers, pharmaceutical companies, Wall Street. While these groups continue to focus on profits, they overlook the ultimate, longer term pulse of society, which is beginning to question more frequently than ever the benefits of the traditional health care system. How many times have you heard stories about individuals who feel so much better after they stop taking all the drugs that they had been prescribed? It happens. It is not uncommon for one prescription to be offered to offsets the effects of another, and another drug is prescribed to offset the effects of the second prescription, and so on, to a point where one treatment begets another and we forget why we needed treatment in the first place. It's no wonder that the drug and insurance companies are flourishing.

Is society ready to embrace alternative health care options such as biofeedback and neurofeedback? Perhaps not yet, at least in mass. But a change is coming. And as the professional societies representing these effective alternative treatment modalities, AAPB, BCIA, and ISNR, must be ready not only to embrace it but to lead the change to a more healthy and more effective forms of treatments.

David L. Stumph, IOM, CAE

Letter from Interim ISNR ED

Karen Forbes

Dear Members and Friends of ISNR,

The end of the year seems to get everyone thinking about the events of the year ending and the promise of the year beginning. My "year" at ISNR was only six months, but what an amazing time it has been! As an interim executive director, I have the opportunity to work with many organizations with different missions and at different levels of organizational development. Each has its own Board members, volunteers and staff with varying backgrounds and familiarity with nonprofit best practices and experience. Those who have chosen to share their time, talents and resources in positive ways to reach out to students, researchers and seasoned practitioners through ISNR are among the most dedicated, thoughtful and professional individuals I have encountered. The annual ISNR conference is the most visible result of their efforts. Countless hours over many months are donated by

committee members to ensure that attendees from near and far, at every level of experience have appropriate opportunities for professional growth through workshops, presentations, discussion groups and informal gatherings. These dedicated volunteers consider everything from the coffee service to the keynote presentations, from continuing education requirements to evening entertainment, and from attendee comfort to affordability. And, they do all of this, not as a fund raiser in support of the organization but as a member benefit. It is a Herculean project conceived and achieved by amazing volunteers to whom I tip my consultant hat.

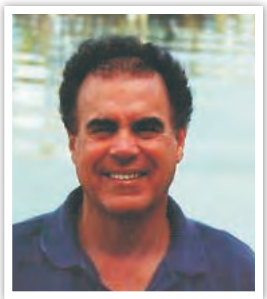
As we all wrap-up 2011, I will wrap-up my time with ISNR and share in the excitement for the future, from a distance. During 2012 the society will continue to reach out to include new members from around the world and to implement meaningful membership benefits for all, to continue to produce printed and on-line materials that inform and instigate thought, and to continue to orchestrate opportunities for professional growth at the annual conference. ISNR will also welcome their permanent Executive Director. His/her skills will likely include previous success managing an association according to sector best practices, membership development and engagement, editing and design of printed materials, financial management with high level—"board-ly"—reporting experience and of course, knowledge of and the belief in the benefits of neurofeedback and related therapies as a treatment for a variety of conditions. 2012 will be a landmark year for ISNR!

To those who provided positive input and support for the interim project—thank you! To those who shared their time, talents and resources that led the society through this period of transition—thank you. And, to those who have a vision for the future for both the ISNR and potential uses of neurofeedback to improve the lives of many—thank you! Now is the time to volunteer to serve on a committee to help sculpt ISNR's future—there is a place for everyone!

Wishing you all a happy, healthy and successful 2012!

Karen Forbes

Loreta Z Score Biofeedback and Traumatic Brain Injury



Robert W. Thatcher, Ph.D.

Quantitative EEG (qEEG) analysis does not provide a definitive stand alone test for traumatic brain injury nor is it a screening test for TBI. Instead, qEEG is used as an adjunct along with other tests such as clinical history, neuropsychological tests, MRIs, etc. to help a clinician to diagnose and treat patients with a history of TBI. In the last 30 years the application of qEEG to evaluate traumatic brain injury has grown dramatically with applications in traumatic brain injury intensive care units (Buzea, 1995; Classen et al, 2000; Haug et al, 2004; Cottencea et al, 2008; Shields et al, 2007), correlation with neuropsychological tests in TBI patients (Thatcher et al, 1998a; 1998b; 2001a, 2001b), correlations with biophysical measures in TBI patients (Thatcher et al, 1998a; 1998b; 2001b), prognostication of recovery (Fabregas et al, 2004; Grindel et al, 2006; Thatcher et al, 1991; Theilen et al, 2000; Nenadovic et al, 2008), reduced connectivity in neural networks (Johnson et al, 2011; Castellanos et al, 2011) and treatment of mild to moderate TBI patients using qEEG operant conditioning (also called neurofeedback) to change the frequency and phase relationships in the brain (Thornton, 2000; Thornton and Carmody, 2005; 2008; 2009; Tinius and Tinius, 2001; Hoffman et al, 1996a; 1996b; Ham and Packard, 1996; Duff, 2004; Ayers, 1987; Byers, 1995; Schoenberger et al, 2001). Thus, there is currently a wide spectrum of clinical applications of qEEG in traumatically brain injured patients by psychiatrists, neuropsychiatrists, family practitioners, internal medicine doctors, neurosurgeons, clinical psychologists and neuropsychologists as evidenced in the vast scientific literature that has accumulated over that last 20 years[1]. For example, a survey of the National Library of Science medical database using the search words "EEG and traumatic brain injury" produced over 2,800 qEEG peer reviewed citations.

In general, the scientific literature presents a consistent and common quantitative EEG pattern correlated with mild TBI (mTBI). Namely, reduced amplitude of alpha, beta and gamma frequency bands (8–12 Hz, 13–25 Hz and 30 - 40 Hz respectively) (Mas et al, 1993; von Bierbrauer et al, 1993; Ruijs et al, 1994; Korn et al, 2005; Hellstrom-Westas, 2005; Thompson et al, 2005; Tebano et al, 1988; Thatcher et al, 1998a; 2001a; Roche et al, 2004; Slewa-Younan, 2002; Slobounov et al, 2002). Changes in EEG coherence and phase delays have also been consistently published for qEEG and fMRI (Thatcher et al, 1989; 1991; 1998b; 2001b; Hoffman et al, 1995; 1996a; Trudeau et al, 1998; Thornton, 1999; 2003; Thornton and Cormody, 2005; Johnson et al, 2011; Castellanos et al, 2011). The reduced amplitude of EEG is believed to be due to a reduced number of synaptic generators and/or reduced integrity of the protein/lipid membranes of neurons (Thatcher et al, 1997; 1998a; 2001b). EEG coherence is a measure of the amount of shared electrical activity at a particular frequency and is analogous to a cross-correlation coefficient. EEG coherence is largely amplitude independent and is correlated to the amount of functional connectivity between distant EEG generators (Nunez, 1981; 1994; Thatcher et al, 1986; Thatcher et al, 1998b). EEG phase delays between distant regions of the cortex are mediated in part by the conduction velocity of the cerebral white matter which is a likely reason that EEG phase delays are often distorted following a traumatic brain injury (Thatcher et al, 1989; 2001a). In general, the more severe the traumatic brain injury then the more deviant the qEEG measures (Thatcher et al, 1998a; 1998b; 2001a; 2001b).

The relatively high consistency (homogeneity) across qEEG analyses of traumatic brain injury is because of a common etiology due to the biomechanics of rapid acceleration/deceleration of the brain inside of the human skull (Ommaya, 1968; 1995; Ommaya and Hirsch, 1971; Davis, 2000). The physics of rapid acceleration/deceleration provide a deductive cross-validation of qEEG studies of traumatic brain injury based on the laws of inertia. For example, the temporal lobes and frontal lobes sit in bony "vaults" with apposition to the frontal and temporal bones and rapid acceleration/deceleration always maximally impacts these brain regions to some extent, independent of the direction of impact of a force on the skull because of the fact that the brain sits on a bony hard surface (Ommaya, 1968; 1995; Davis, 2000; Sano, 1967). In the case of closed head injuries the forces are much greater in the orbital frontal, frontal poles and anterior temporal lobes than anywhere else in dependent of the direction of the impact to the skull. In the case of whip lash, the forces are posterior to the skull and include brain stem stretching and torsion forces. In the case of IED energy forces that arise directly beneath the body the forces are direct upward through the spinal canal and brainstem resulting in sleep and anxiety problems in addition to the common frontal and temporal cortical injury. The consistency of biomechanical forces in closed head injury, in contrast to penetrating head wounds, is due to three common forces: 1- a

1 The American Academy of Neurology position paper (Nuwer, 1997) does not support the use of QEEG to evaluate TBI and thus neurologists typically do not use qEEG for this purpose For example, the author of the AAN position paper recently estimated that less than 100 neurologists use qEEG in TBI evaluations (Dr. Nuwer's April, 6, 2004 deposition in "State of Florida vs. Samuel Harris", pg. 67, lines 1–10.).

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percussion force that travels from the point of impact on the skull to the opposite side of the skull in less than 150 msec often producing a coup contra-coup pattern and disrupting protein-lipid neural membranes, 2- linear forces that are maximal in the frontal and temporal bone to brain interfaces that result in contusions of the frontal and temporal lobes and, 3- shear/rotational forces where different densities of brain tissue move at different rates (e.g., gray matter *vs*. white matter) that result in swelling of axons and diffuse axonal injury. Because of the high sensitivity of qEEG, detection and quantification of coup contra-coup patterns related to the point of impact against the skull is also a common finding. Finally, 3-dimensional electrical source localization and co-registration with MRI help to further identify the brain regions most affected by TBI and to aid in linking patient symptoms and complaints to functional specialization in the brain (Thatcher et al; 1998b; 2001; 2005; Korn et al, 2005).

qEEG Current Source Localization and TBI

In the last 15 years new advances in 3-dimensional source imaging or QEEG neuroimaging have evolved to the point of high sensitivity and high localization accuracy (Pascual-Marqui et al, 1994; Pacual-Marqui, 1999; Thatcher, 2011; Hernandez-Gonzalez et al, 2011). Low resolution electromagnetic tomography (LORETA) is easy to use, has been cross-validated in numerous studies and has high localization accuracy. There are over 750 peer-reviewed publications on the topic of QEEG and LORETA which is too extensive a literature to review here. LORETA is free at: http://www.unizh.ch/keyinst/NewLORETA/Software/Software.htm and it is also helpful in the evaluation of coup contra-coup patterns. The importance of 3-dimensional source imaging as an adjunction to the evaluation of traumatic brain injury is that it provides clinicians with a method to link the patient's symptoms to functional localization in the brain (Thatcher et al, 2005; Korn et al, 2005; Boyd et al, 2007; Leon-Carrion et al, 2008a; 2008b). High sensitivity and specificity arises because one can test hypotheses prior to launching 3-dimensional electrical source imaging. This is done by predicting frontal and temporal lobe and network deviations from normal that are present in patients with a history of TBI and complaints such as short-term memory problem, attention and concentration problems and/or depression (see Johnson et al, 2011).

Figure one shows an example of reduced functional connectivity in the posterior cingulate part of the default mode network in a group of mild TBI patients (mTBI) in comparison to control subjects (NV). Reduced functional connectivity following a traumatic brain injury was reported for other parts of the default mode network (Johnson et al, 2011) as well as in surface qEEG coherence studies (Thatcher et al, 1998b). The application of LORETA coherence and LORETA phase differences between Brodmann areas provides clinicians with important tools to evaluate the affects of trauma on various networks in the brain.

Surface qEEG Biofeedback (Neurofeedback) and the Treatment of TBI

One of the earliest qEEG biofeedback studies was by Ayers (1987) who used alpha EEG training in 250 head injured cases and demonstrated a return to pre-morbid functioning in a significant number of cases. Peniston et al (1993) reported improved symptomology using qEEG biofeedback in Vietnam veterans with combat related post-traumatic disorders. More recently Hoffman et al (1995) in a biofeedback study of fourteen TBI patients reported that approximately 60% of mild TBI patients showed improvement in self reported symptoms and/or in cognitive performance as measured by the MicroCog assessment test after 40 sessions of qEEG biofeedback. Hoffman et al (1995) also found statistically significant normalization of the qEEG in those patients that showed clinical improvement. Subsequent studies by Hoffman et al (1996a; 1996b) confirmed and extended these findings by showing significant improvement within 40 sessions. A similar finding of qEEG normalization following EEG biofeedback was reported by Tinius and Tinius (2001) and Bounias et al (2001; 2002). Ham and Packard (1996) evaluated EEG biofeedback in 40 patients with posttraumatic head ache and reported that 53% showed at least moderate improvement in headaches; 80% reported moderate improvement in the ability to relax and cope with pain and 93% found biofeedback helpful to some degree. Thornton and Carmody (2005) reported success in using qEEG biofeedback for attention deficit disorders in children with a history of TBI. An excellent review of the surface qEEG biofeedback literature for the treatment of TBI is in Duff (2004).

LORETA Z Score Biofeedback

A new method of EEG biofeedback is the use of LORETA Z score biofeedback to directly train deregulated or unstable functional systems of the brain linked to symptoms that arose as a consequence of a traumatic brain injury. This new method involves the use of quantitative EEG to identify unstable or deregulated brain regions and network nodes linked to a patient's symptoms followed by LORETA Z score biofeedback to train toward improved stability and regulation in the affected brain regions. For example, short-term memory with the memory networks (anterior cingulate, prefrontal cortex, hippocampus, temporal lobes) or attention and concentration problems and the attention network (prefrontal cortex, parietal lobes, anterior cingulate, temporal lobes, default mode network) or mood dyscontrol and the mood networks of the brain (insula, medial and lateral frontal lobes, amygdala). Once symptoms and complaints are linked to functional networks in the brain then LORETA Z score biofeedback is used for

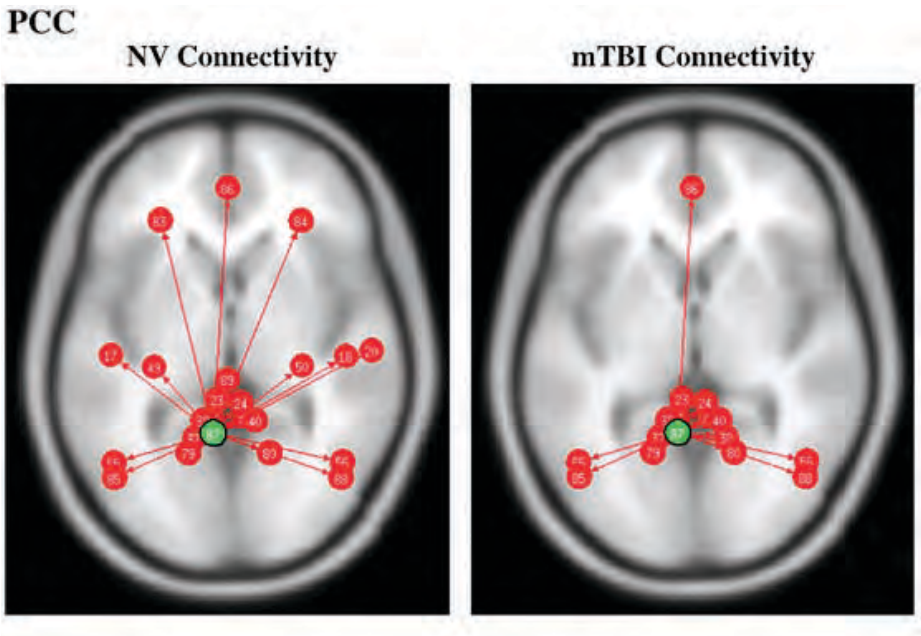

Figure 1: *Example of reduced connectivity in mTBI patients (right) in comparison to control subjects (NV) (left) using fMRI functional connectivity analyses (see Johnson et al, 2011 for details). A general reduced connectivity is reported in the default mode network in mTBI patients in comparison to controls (From Johnson et al, 2011).*

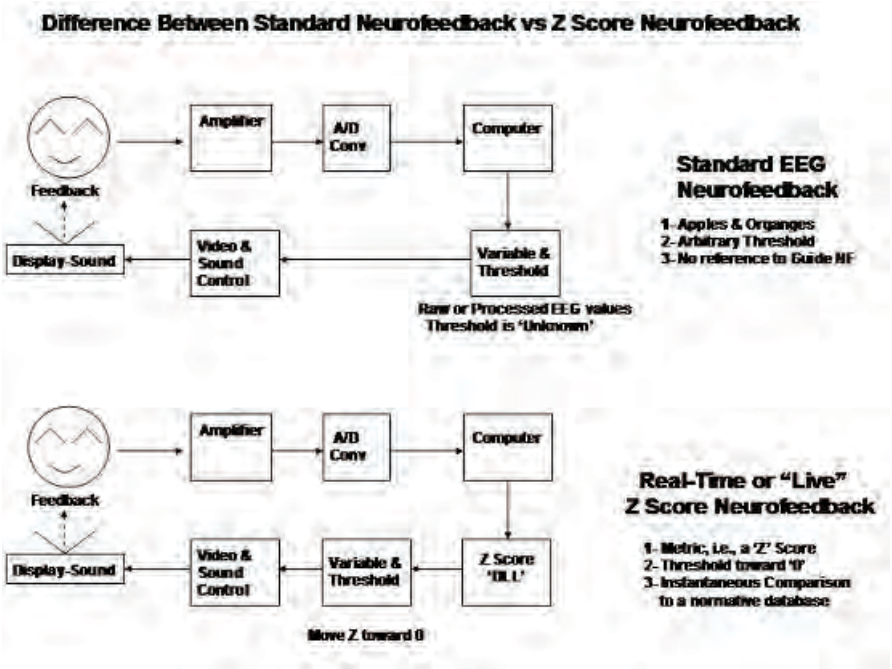

Figure 2: *Illustration of the difference between 'raw score' EEG biofeedback (Top) and 'Z score' EEG biofeedback (Bottom). The methods are the same except that the raw scores are instantaneously transformed to Z scores with respect to the mean and standard deviation of an age matched reference population of healthy individuals. Z score biofeedback simplifies by reducing disparate EEG metrics (abs. power, relative power, coherence, phase, phase shift, etc.) to a single common metric, i.e., the metric of a Z score. Z score biofeedback also takes the guess work out of the process by providing the clinician with a common target toward which the brain is reinforced, i.e., the center of an age matched group of healthy individuals.*

the treatment of mild to moderate TBI. The goal is to achieve consistent improved clinical outcome in fewer sessions than is possible using surface EEG biofeedback or even surface Z score biofeedback.

Z score biofeedback was first developed by myself and colleagues in 2004 and is now commonly used by hundreds of clinicians and is distributed by a variety of EEG biofeedback companies (e.g., BrainMaster, Thought Technology, EEG Spectrum, NeXus, Deymed, Neurofield, Advanced Brain Monitoring and Mitsar). The ability to achieve improved clinical outcome in fewer than 20 sessions using Z score biofeedback has been documented in several publications (Collura et al; 2008; 2011; Hammer et al, 2011). Figure two illustrates how Z score biofeedback differs from raw score biofeedback by using a real-time or "live" comparison to an age matched reference database of healthy normal subjects (see Thatcher & Lubar, 2008). This is similar to the use of a real-time blood test where the patient's cholesterol or liver enzymes are measured instantaneously or in real-time and the clinician treats the patient by modifying the organ systems responsible for the deviant blood constituents. Z scores are a statistical measure of distance from the center of the age matched reference normal population and therefore Z scores provide a simplified 'threshold guide' in which the goal is to reinforce toward Z = 0. This is in contrast to non-Z score biofeedback or 'raw score' biofeedback in which the threshold for reward is arbitrary involving many different measures with different metrics. For example, as illustrated in the top row of Figure 2, with raw score biofeedback the clinician must guess at a threshold, e.g., reinforce if theta rhythms are less than 6 microvolts or maybe 10 microvolts or inhibit if beta is greater than 12 microvolts or should it be 10 microvolts or reinforce coherence when it is greater than 0.3 or maybe 0.5 is the correct guess for the threshold? In contrast, as illustrated in the bottom row of figure two, Z scores simplify EEG biofeedback by using a single common metric no matter what the EEG measure, providing age and location matching and by removing the guess work because now the feedback threshold is not arbitrary but rather is uniform and always involves reinforcing toward Z = 0. This does not mean that a patient will ever attain exactly Z = 0 because none of the normal subjects are exactly at Z = 0 because this is only a statistical value that a large group of normal subjects deviate around. Instead, one is reinforcing increased stability and homeostasis in brain networks that have been damaged or have become deregulated. Z = 0 is a statistical "ideal" just like the center of the "normal reference" blood range when using a blood test.

LORETA Z score biofeedback differs from surface Z score biofeedback by targeting specific Brodmann areas or 3-dimensional locations of hubs and modules of networks linked to the patient's symptoms. With the surface EEG a given scalp electrode is sensing electrical potentials generated in many different parts of the brain that are mixed together at the scalp, e.g., the Cz electrode is detecting a mixture of sources from the occipital, frontal, temporal and parietal lobes. In contrast, LORETA is a mathematical method that unscrambles the mixture of electrical sources and provides a 3-dimensional depth source analysis at resolutions of less than 1 cubic centimeter (see Pascual-Marqui et al, 1994 and Pascual-Marqui, 1999 for the mathematical details and validations). The use of raw LORETA Z scores has the same limitations as the use of surface raw scores in comparison to Z scores. That is, biofeedback of raw LORETA values involves the use of an arbitrary threshold where the clinician must guess at whether or not to reinforce for current densities greater or less than a certain value. Raw LORETA phase differences and raw LORETA coherence suffer from the same complexity and arbitrariness and metric apples and organges. In contrast, LORETA Z score biofeedback removes the guess work and provides an age matched normal reference population as a guide and simplifies absolute power, relative power, coherence, phase, phase shift, phase lock, etc. to a single common metric, i.e., the metric of a Z score where one reinforces toward Z = 0 where zero is the idealized center of a group of healthy individuals.

DoD/VA LORETA Z Score Symptom Check List for TBI

The US Army has implemented LORETA Z score biofeedback as a standard clinical treatment of active duty military personnel involved in an extensive rehabilitation program (Fort Campbell Warrior Resiliency and Recovery Center or WRRC). Drs. Joel Lubar, Marc Zola and David Twilley are involved in the implementation of LORETA Z score biofeedback that follows an extensive clinical evaluation of each soldier including surface qEEG evaluations. A link of the patient's symptoms to deregulated or unstable networks of the brain known to be vulnerable to rapid acceleration/deceleration injuries is made through the use of a symptom check list that follows the 'Co-occuring Conditions Toolkit for Mild Traumatic Brain Injury and Psychological Health' or CONUS. In Neuroguide this is a tab located in the symptom check list panel for Z score biofeedback and labeled 'DoD/VA'. An example of the DoD/VA symptom checklist is shown in figure three and Table I. The upper left panel is a symptom check list based on the Defense Centers of Excellence

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of Psychological Health & Traumatic Brain Injury which was established by the Department of Veterans Affairs in 2009 and incorporated into the DoD/VA clinical practice guidelines. The items in the Dod/VA tab reproduce the 2009 "Co-occurring Conditions Toolkit: Mild Traumatic Brain Injury and Psychological Health" (CONUS) for Concussion, Posttraumatic Stress, Depression, Chronic Pain, Headache and Substance Abuse Disorder. The right panel are Brodmann areas and the lower left panel are hypothesized Brodmann areas known to be related to a given symptom or assessment based on the scientific literature. The lower middle panel are the matches of deviant qEEG Z LORETA Z scores to the hypothesized Brodmann areas linked to the patient's symptoms. The lower right are the mismatches of deviant LORETA qEEG Z scores that are likely related to compensatory processes. The goal of this procedure is to separate the 'weak' systems from the 'compensatory' systems and to target the 'weak' systems for EEG biofeedback training and reinforce movement of the weak system toward Z = 0 which is the center of an age match normal population. Specific Brodmann areas can be trained such as the anterior cingulate gyrus in depression or attention deficit or the parahippocampus in attention deficit or the left angular gyurs in dyslexia, etc.

Z score LORETA biofeedback includes current density and coherence and phase differences between Brodmann areas and network nodes. The goal is to identify the network nodes linked to the patient's symptoms and then to reinforce toward Z = 0 which is the center of a group of age matched and healthy individuals with no history of trauma, no history of neurological disorders and no history of psychological/neuropsychological problems. The project at Fort Campbell involves careful monitoring of all soldiers and extensive behavioral and psychological evaluation prior to implementing LORETA Z score biofeedback and pre *vs.* post treatment assessment at various stages of the rehabilitation program. The LORETA Z score symptom checklist is specially adapted from the Defense Centers of Excellence of Psychological Health & Traumatic Brain Injury manual. The items in the DoD/VA tab reproduce the "Co-occurring Conditions Toolkit: Mild Trauamtic Brain Injury and Psychological Health" (CONUS) for Concussion, Posttraumatic Stress, Depression, Chronic Pain, Headache and Substance Abuse Disorder. The selected network Brodmann areas related to different symptoms and clinical history of soldiers that suffered a TBI are based on a survey of the National Library of Medicine database using search terms such as "fMRI and traumatic brain injury" or "PET and traumatic brain in-

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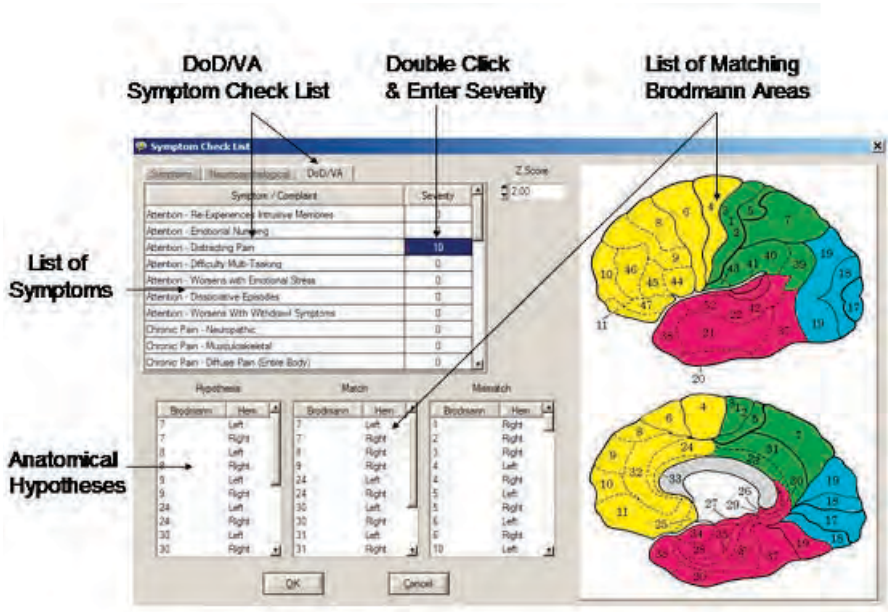

Figure 3: *EEG biofeedback of LORETA Z scores that are linked to the patient's symptoms and complaints. The upper left panel is a symptom check list based on the Defense Centers of Excellence of Psychological Health & Traumatic Brain Injury. The items in the Dod/VA tab reproduce the "Co-occurring Conditions Toolkit: Mild Trauamtic Brain Injury and Psychological Health" (CONUS) for Concussion, Posttraumatic Stress, Depresson, Chronic Pain, Headache and Substance Abuse Disorder. The right panel are Brodmann areas and the lower left panel are hypothesized Brodmann areas known to be related to a given symptom or assessment based on the scientific literature. The lower middle panel are the matches of deviant qEEG Z LORETA Z scores to the hypothesized Brodmann areas linked to the patient's symptoms. The lower right are the mismatches of deviant LORETA qEEG Z scores that are likely related to compensatory processes. The goal of this procedure is to separate the 'weak' systems from the 'compensatory' systems and to target the 'weak' systems for EEG biofeedback training and reinforce movement of the weak system toward Z = 0 which is the center of an age match normal population. Specific Brodmann areas can be trained such as the anterior cingulate gyrus in depression or attention deficit or the parahippocampus in attention deficit or the left angular gyurs in dyslexia, etc. (From NeuroGuide 2.6.9).*

DoD/VA Symptom Check List – Adapted from the US Army CONUS Manual

Attention-Re-Experiences Intrusive Memories	Concussion-Difficulty Multi-Tasking
Attention-Emotional Numbing	Concussion-Short-Term Memory Problems
Attention-Distracting Pain	Concussion-Difficulty Concentrating
Attention-Difficulty Multi-Tasking	Concussion-Sleep Problems
Attention-Worsens with Emotional Stress	Concussion-Balance Problems
Attention-Dissociative Episodes	Concussion-Problems Controlling Anger
Attention-Worsens With Withdrawl Symptoms	Concussion-Depressed Mood
Chronic Pain-Neuropathic	PTSD-Hyperarousal
Chronic Pain-Musculoskeletal	PTSD-Sudden Fear Reactions
Chronic Pain-Diffuse Pain (entire body)	PTSD-Excessive Sleep-Lethargic
Chronic Pain-Pain Triggers Memories of Trauma	PTSD-Difficulty Falling Asleep due to Rumination
Mood-Emotional Numbing	PTSD-Mood Disorders
Mood-Irritability	Sleep-Fear of Sleep Due to Nightmares
Mood-Emotional Fatigue	Sleep-Difficulty Falling Asleep due to Rumination
Mood-Physical Fatigue	Sleep-Difficulty with Sleep Due to Withdrawl Symptoms
Mood-Lack of Enjoyment of Daily Activities	Sleep-Early AM/Night Time Awakening (unexplained)
Mood-Impulsivity	
Mood-Activities Driven by Medication Needs	
Mood-Hyperarousal	

Table 1: *Symptom list from the US Army CONUS manual which is used inside of Neuroguide for the purposes of Z score biofeedback where symptoms are linked to networks in the brain.*

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jury" or "EEG and traumatic brain injury." A list of brain networks associated with TBI and that are consistently reported in the scientific literature were used to determine the Brodmann areas to target for LORETA Z score neurofeedback. A qEEG assessment is used to rank order the most deviant nodes and hubs of the networks most commonly associated with TBI and follow-up pre vs post qEEG analyses are used to assess the progress of treatment. The US Army program is just getting started and is scheduled to continue for the next five years during which extensive pre vs post treatment assessment and statistical analyses will be conducted.

Robert W. Thatcher, PhD, *is president and chief executive officer of Applied Neuroscience, Inc., Dr. Thatcher's academic, scientific, business and personal achievements are innumerable and require thirty-three full pages to list. He has authored and co-authored six books, more than forty book chapters, hundreds of journal articles and spoken at dozens of presentations and seminars. He holds a Bachelor of Science degree in chemistry from the University of Oregon, a Ph.D. is biopsychology from the University of Waterloo, attended the Albert Einstein College of Medicine for his post-doctoral training in Neurobiology and the New York Medical College for his post-doctoral Neurophysiology training. He has served as a board member of the American Board of Electroencephalography and Clinical Neurophysiology, a National Institute of Health Scientific advisory board and an executive board member of the EEG and Clinical Neuroscience Society. He has been the recipient of the Hans Berger Award of Merit (Association for Applied Psychophysiology and Biofeedback, Neurofeedback Division, May 16, 2008) and the Life Time Achievement Award for work in the scientific specialty of QEEG (American Board of Certification of Quantitative Electroencephalography, September, 1998.) He has administered public and private grants, directed the Applied Neuroscience Institute at the University of Maryland and the Neurometrics Clinical Service at Shock Trauma, University of Maryland. These accomplishments represent only a small portion of Robert's lifelong achievements. At home in Redington Shores, Florida, Robert enjoys his free time by boating, reading and being with family.*

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Detection of Mild TBI

J. Lucas Koberda, MD, PhD

Mild traumatic brain injury (mTBI) is frequently seen in general neurology practice. Any alteration in mental state at the time of the accident (eg. feeling dazed, disoriented or confused) indicates diagnosis of mTBI. In the United States approximately 1.4 million people sustain TBI each year. Most of these injuries are classified as mild, with 80% of patients treated in the emergency room without hospitalization. This syndrome occurs after even mild head trauma with or without loss of the consciousness and consists of symptoms of daily headaches, frequent dizziness, fatigue, and memory and concentration problems. In addition, depression, anxiety, insomnia and other behavioral problems may be associated with this condition. There is increasing evidence that even whiplash types of injuries may cause mTBI due to rapidly occurring acceleration/deceleration, which contributes to brain dysfunction. Unfortunately, there is no single test to confirm this diagnosis in the clinical setting. Results from imaging techniques including magnetic resonance (MRI) and computed tomography (CT) are completely normal unless major head injury occurs where intracranial bleeding can be found. Another MRI modality called diffusion tensor imaging (DTI) is more sensitive in detection of diffuse axonal injury seen after head trauma; however these findings are also not usually seen after minor brain injuries. The introduction of quantitative EEG, which utilizes direct recording of electrical activity from the scalp is an application also used in mTBI cases. After injury, the distressed neurons can be identified by observing specific TBI patterns of neuronal electrical activity. Therefore the application of quantitative EEG (QEEG) seems to be a very promising tool in evaluation of patients suffering from mTBI.

I would like to present a case of mTBI from my practice illustrating my approach to patients suffering from post-concussion syndrome.

A 20-year-old college student fell off her bike and sustained head injury with possible short lasting loss of the consciousness and subsequent symptoms of frequent daily headaches, short term memory and concentration problems. Also she reported being confused for 2 hours after the accident and complained of subsequent sleeping problems. The patient indicated that her CT of the brain completed at the local ER was unremarkable. Her neurological examination in my office was normal; however computerized neurocognitive testing (NeuroTrax, WHERE IS THIS COMPANY?)) showed impairment of memory function (Fig. 1). Memory score was 60.2 (expected mean score-100 where 1 SD-15) which was below 2 standard deviations (SD). Subsequent QEEG analysis (Neuroguide, Inc. St. Petersberg, FL) showed increased bilateral temporal theta power (Fig 2), specifically in the 6 Hz range,

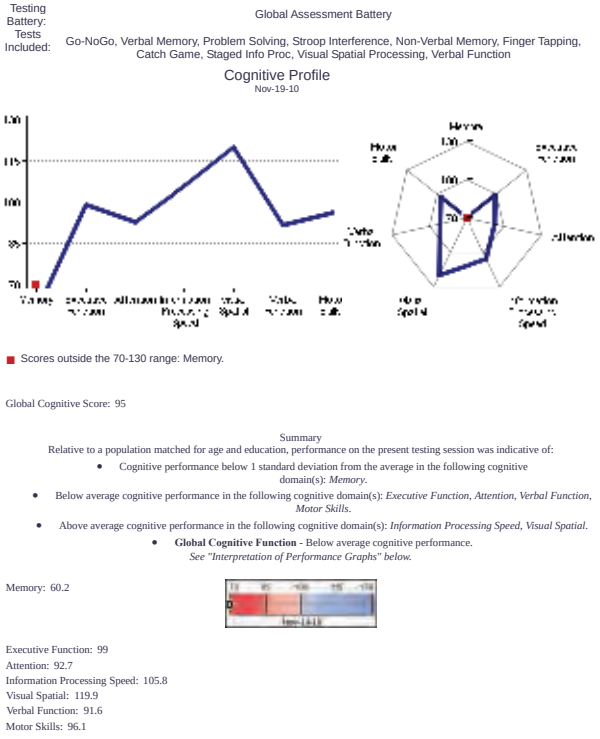

Figure 1: *Patient with mTBI using NeuroTrax (Fresh Meadows, NY) neurocognitive testing*

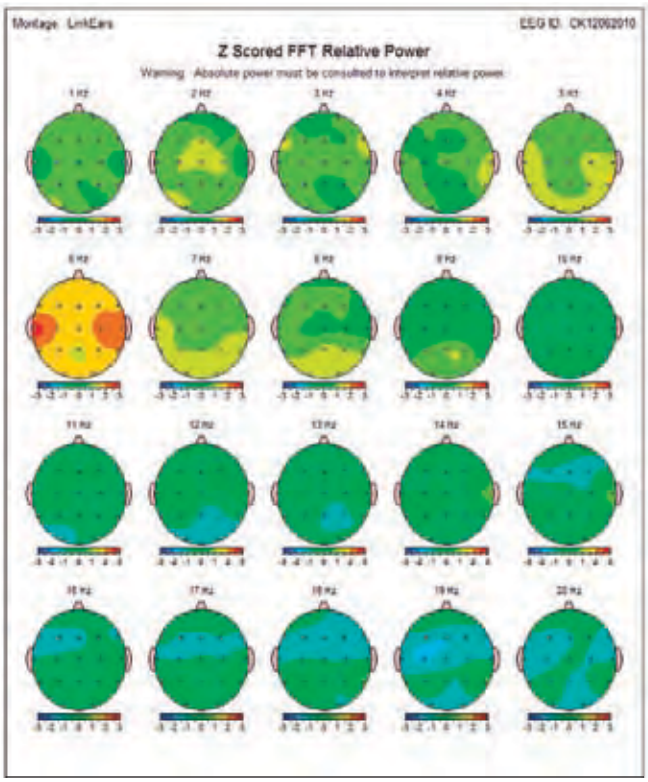

Figure 2: *Patient with mTBI -increased bilateral temporal (6Hz) theta power*

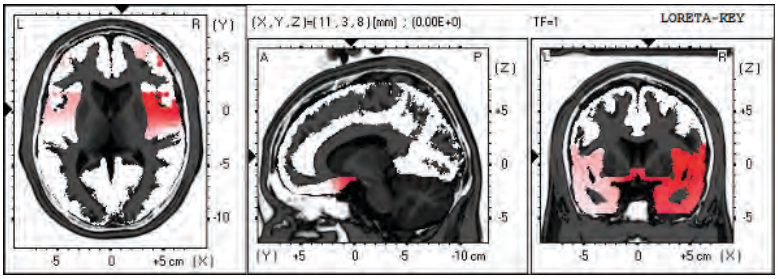

Figure 3: *Patient with mTBI-increased theta activity in bilateral temporal location (LORETA)*

indicative of temporal lobes dysfunction. Low resolution electromagnetic tomography (LORETA) analysis confirmed bilateral temporal localization of theta activity (Fig 3.) Additional traumatic brain injury discriminant analysis (Neuroguide, Inc.) was also consistent with TBI findings (Fig 4.)

Patient was treated with 25 mg of Amityrptyline at night, however shortly thereafter patient's symptoms diminished and after several months since the accident she reported normalization of her cognitive performance and resolution of headaches.

This case illustrates the clinical benefit of QEEG utilization in neurological practice where mTBI patients are frequently evaluated and gives evidence of objective functional neuronal dysfunction related to prior head injury. QEEG complements well neuropsychological testing and is consistent with patient's history of the accident and presenting subjective symptoms.

Dr. Koberda is the director of the Tallahassee Neuro-Balance Center and Clinical Assistant Professor at Florida State University-College of Medicine, Tallahassee FL.

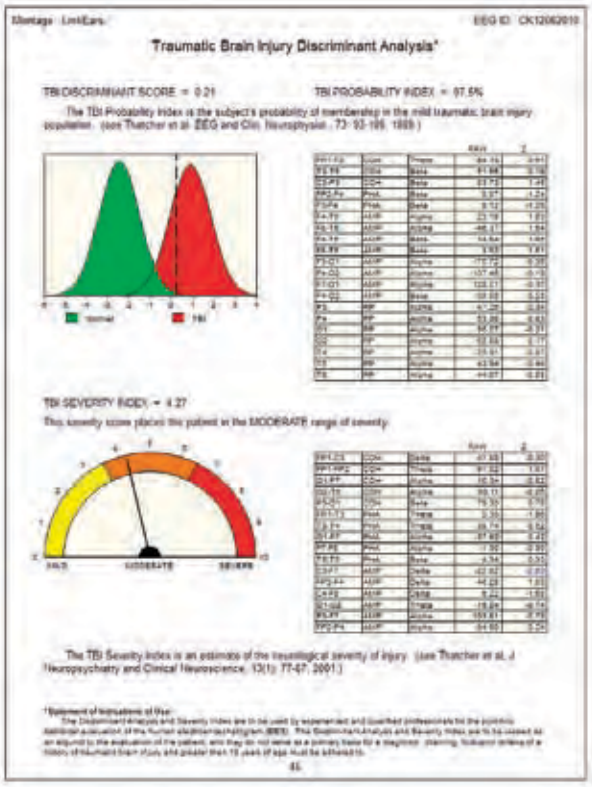

Figure 4: *Patient with mild head injury-positive traumatic brain injury discriminant analysis.*

Improving Quality of Life Using Biofeedback Plus Neurofeedback

Michael Thompson, M.D. and Lynda Thompson, Ph.D.

Introduction

Often our work using neurofeedback (NF) combined with biofeedback (BF) is done to improve the client's quality of life. This is particularly true in cases where complete remission of symptoms is an unrealistic goal, as with neurodegenerative conditions. This article is written to encourage people in our field to work with such clients and to do so in a thoughtful way. It will briefly provide the rationale and describe the procedures we used when we worked with a woman who has Parkinson's disease (PD) and Dystonia over a decade ago (Thompson & Thompson, 2002). The review was inspired by recent research involving EEG training in marmoset monkeys, which used marshmallows as a reward for producing bursts of SMR activity. This research is leading to work involving an animal model for PD (Philippens, 2011). Then we will share some things we have learned since we worked with that case, showing how more recent research in neuroscience and improvements in biofeedback interventions in the last decade, especially regarding networks in the brain, heart rate variability training and brain-body links have expanded our understanding of how our work may assist these patients. The review provides further support for a combined NF plus BF intervention in conditions that range from fibromyalgia to Tourette syndrome, Parkinson's disease and perhaps even some forms of dementia and Alzheimer's disease (Scheltens, 2010).

One important bit of advice before beginning this discourse: be modest in your claims. Follow the adage "Promise less and deliver more." Be sure that you tell the client that this work is experimental. Explain the rationale for why there might be some improvement but be very clear that there is no expectation of reversing a progressive disease. The goals are to improve quality of life and perhaps gain self-regulation skills that might help in the management of symptoms. At best, one might slow the progression of the disorder. Remember that NF only has established efficacy for Attention-Deficit/Hyperactivity Disorder and for epilepsy (and then only for reduction in frequency and perhaps duration and severity of seizures, not cure). All other applications must be described as being experimental. In short, keep basic ethical principles in mind, including practicing within the limits of your knowledge. Do not over-sell the field because then we all lose credibility.

Case Example

Here is the story, in brief, of our client. (More details are available in *Thompson & Thompson, 2002.*) "Mary" approached us in April 2000 after hearing about NF, hoping we could try neurofeedback to treat symptoms associ-

ated with her advanced Parkinson's disease. She had been diagnosed with PD fourteen years before and had tried all the traditional medical approaches, plus some experimental ones, like the transplantation of fetal cells into the caudate in December 1998. Massage, meditation and yoga were used, too, though her problems with focus made meditation difficult. She was taking Sinemet (levodopa and carbidopa), which was the same medication taken since her diagnosis, and was additionally taking Requip (an agonist to supplement the L-dopa medication), Amantadine (a dopamine reuptake inhibitor to lessen dyskinesia), Ativan to relieve rigidity, and Prepulsid to prevent reflux. She had suffered from dystonia for five years (which can be a side-effect of L-dopa) and had sudden, painful muscle contractions, especially on the left side, producing twisting movements of her leg, shoulder, head and jaw. The fetal cell transplant had restored her mobility, though she was unable to complete some of her 1-year post surgery follow-up testing, such as the MRI, due to her uncontrollable movements. She had continuing problems with daytime sleepiness and had 1 to 2 naps per day) plus sleep problems at night characterized by nighttime awakening and inability to fall back to sleep. She had trouble focusing and finishing things and had not been able to read a book for 4 to 5 years. Additionally, there were symptoms of dysphoria that bordered on depression. She still did presentations and fund-raising for the Parkinson's Society. However, the adrenalin rush before a talk increased her symptoms of muscle spasms and "freezing" (an inability to initiate action that is common in advanced Parkinson's), which could leave her standing on stage unable to move or speak. Despite her challenges, she was an incredibly positive person and worked on reframing things in an optimistic way. She was clearly a motivated client who was not going to give up. She was willing to try anything that might help. A year ago she was in touch to say that she appreciated the work done a decade ago and updated us regarding surgery she underwent for deep brain stimulation that eventually, after taking more than 12 months to fine tune the stimulation, had improved her mobility to the point where she could dance again.

Regarding the EEG intervention, we did 30 sessions of NF plus BF training from June–December 2000 and a further 12 sessions on a sporadic basis over the next 12 months mainly using the Procomp+/Biograph (Thought Technology, Montreal, Canada) or, occasionally, the F-1000 (Focused Technology). The Biograph instrument allowed for simultaneous recording and feedback for EEG and also 6 additional channels for EMG and peripheral biofeedback. The F-1000 equipment did EEG plus temperature and electrodermal response (EDR) and had a separate add-on for respiration. Regarding EEG, we first did a single channel EEG profile using a sequential (bipolar) placement at FCZ and CPZ (midline on either side of CZ) and found 13–15 Hz was exceedingly low amplitude and 9 Hz was very high in amplitude. There was also an increase in amplitude at 29–31 Hz, which is in the range we later came to call the "busy-brain" frequencies (Thompson & Thompson, 2006). It thus made sense to do training to increase sensorimotor (SMR) in the 13-15 Hz range because her symptoms of uncontrolled motor movements matched the EEG pattern and might be improved with training. Sterman's early research also suggested that it could improve sleep because sleep spindle density at night increases proportionally to SMR increase daytime (Sterman, 1996). That was a main training goal for her EEG parameters. On the F-1000 she would begin with breathing paced at 6 breaths per minute and then do what was called a Tansey screen that rewarded an increase in 14 Hz activity (Tansey,1986). The low frequency inhibit was set for 6-10 Hz to bracket her high amplitude "thalpha" activity and the high frequency inhibit range was 25–32 Hz in order to reduce her peak in the high beta range and double as an EMG inhibit. We began with sequential placement (FCZ-CPZ) due to her EMG artifact being high but were able to switch to a referential placement (CZ-left ear) as her dystonic movements decreased.

Regarding the biofeedback intervention, we used all the modalities available with the Thought Technology equipment: EMG sensors on her trapezius muscles to help the shoulders relax; temperature sensor on her little finger to help with relaxation; EDR sensors for arousal level (counteract her sleepiness/low arousal); a plethysmograph (blood volume pulse sensor) for heart rate; and a respiration belt around the abdomen for diaphragmatic breathing. The main emphasis was on learning to breathe diaphragmatically. This equipment allowed us to do accurate neurofeedback at the same time as biofeedback. With the range of feedback screens available, we could measure and record all parameters while having any one modality, or any combination of modalities, on the screen and controlling the audio feedback. At the beginning of each session she practiced effortless, diaphragmatic breathing to achieve synchrony between respiration and heart rate changes on an RSA screen (respiratory sinus arrhythmia). (This was a forerunner to the heart rate variability training currently used for vagal afferent feedback to the medulla, which increases parasympathetic tone and has other positive effects on the affect.) Other feedback, such as increasing skin temperature, was also sometimes used to decrease sympathetic drive. Then she continued the effortless breathing while doing the NF (increasing SMR, decreasing 6–10 Hz and 25–32 Hz) with a variety of screens.

Results were that she became able to walk unassisted whereas, at the start of training, she had used a cane and, sometimes, another person to support her. She became able to read novels again after her first dozen sessions. Dystonic movements were markedly reduced and her movements, in general, became smoother. Her two-year post-surgical follow-up went well: she could stay still for the MRI and she recovered from being off medications after a few days, whereas the previous year it had taken her months to get stabilized again. She got back to doing art and was able to write again. The biggest difference in quality of life was, she said, "I can breathe my way out of freezing." Her L-dopa medication had been reduced by about 1/3. Using less L-dopa medication is important, as it may extend the period of time of effectiveness for this L-dopa medication. After training she continued using an agonist (Comtan had replaced Requip) and Amantadine. She had discontinued Prepulsid and the Ativan (lorazepam) was used only occasionally on an as-needed basis, whereas before training she took it once or twice every day.

Theoretical Rationale for the NF plus BF intervention

What was the reasoning behind combining NF with BF? In this patient with Parkinson's, due to the dystonic movements, we had to begin with respiration at about 6 breaths per minute because the quality of the EEG signal was poor because of her uncontrolled movements that were due to both tremor and dystonia. The effect of this slow, diaphragmatic breathing is to increase vagal parasympathetic tone and to give vagal afferent input to the nucleus of the solitary tract in the medulla (Porges, 2007). This links directly to the locus coeruleus (which has efferents to the hypothalamus, the thalamic relay nuclei, the amygdala, and the basal telencephalon), the basal ganglia, and to cortical areas such as the cingulate gyrus including, for example, Brodmann Area 25 in the ventral rostral portion of the anterior cingulate (Gevirtz, 2010). (BA 25 is where implants are done for intractable depression.) Thus there are direct effects on the 'affect network..' One function of these connections is to influence the hypothalamic-pituitary-adrenal axis and modulate responses to stress (Thompson, 2007). Skin temperature and electrodermal training add to this because they tend to decrease sympathetic drive and thus decrease physiological responses to anxiety and stress. We call this the "bottom-up" effect of BF.

The "top-down" effects are accomplished by NF, which will have effects on the same nuclei and the same motor, executive, and affect networks (Seeley, 2007, Thompson, 2009, 2011). In this patient with Parkinson's, the BF resulted in sufficient control of undesirable dystonic movements to allow her to more effectively take part in NF training done

Continued on page 20

Software Design at Thought Technology

Software development is a tough job. I don't mean the actual programming; I mean that conceiving a software program is tough job… Whenever we get a new idea, I ask our programmers: "Can it be done?" and their answer is always the same: "This is software, anything can be done! All you need to do is tell me *exactly what you want*! And that is the problem . . . Sometimes knowing exactly what we want is very difficult . . .

Thought Technology has been developing software products since the mid 1980s. You'd think that, by now, software development would be so easy for us that we could do it with our eyes closed, but . . . Each new project comes with a new set of challenges and difficulties, requiring new and creative approaches. As I am reflecting on the enormous task of moving our 60 employees and main offices to a bigger, better corporate space of 13,000 square feet, I am also remembering the times when we were just a handful of people, working in a Montreal basement. In those days, computers were a lot simpler than they are now but the challenge was to cram all our ideas in less than 64 Kilobytes of RAM!

30 years later, we are getting ready to release version 6.0 of our flagship software platform, BioGraph Infiniti. Each new version is a challenge because we have to meet the needs of increasingly more sophisticated and demanding clinicians, an ever-evolving PC technology and more stringent rules for FDA, CE and various international medical technology regulations.

BioGraph version 6.0 reflects those requirements with a redesigned and slicker user interface, a professional grade sound manipulation engine, powerful new session review capabilities and a slew of new features for physiological biofeedback, z-score neurofeedback as well as slow cortical potential (SCP) and evoked potential (EP) work. Of course, because the "first time" experience should always be a pleasant one, we've made BioGraph's installation process as simple and automatic as possible.

Thought Technology takes software design seriously and strives to produce professional, clinically relevant products that can be used in small practices, research laboratories, hospitals and corporate settings alike, with full confidence. Our software IS intended for clinical use! The challenge of software design is one that we accept with assurance because, after 30 years of experience, we know what we want. We want to deliver the best product we can and meet the needs of all our users with a minimum of stress and a maximum of satisfaction.

Hal Myers, PhD, President, Thought Technology Ltd.

Improving Quality of Life continued from page 20

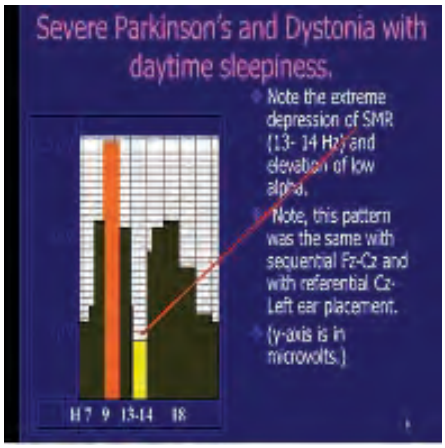

Figure 1: *Single channel assessment profile (from The Neurofeedback Book. 2003)*

over the central FCz–CPz area of the brain, which is, of course, over the cingulate gyrus.

The calming effects may have been due to reduction in sympathetic drive, as we know that the sympathetic system directly innervates the fusiform fibers of the muscle spindles that, in turn, influence muscle tone. Interestingly, gamma motor efferents from the red nucleus also control the fusiform fibers of the muscle spindles. The red nucleus slows down its firing rate when SMR, generated from the thalamus, is increased. Thus our rational was to condition her to raise SMR while she was doing diaphragmatic breathing so that there was a dual effect on muscle spindles. In 2000 it was not known that heart rate variability (HRV) training alone would result in an increase in SMR. This was recently documented at our ADD Centre and presented at the 2011 meeting of the ISNR (Reid & Nihon, 2011). Even with our more limited knowledge and with only single channel EEG assessment to guide training, the combination of NF to raise SMR while also doing RSA training assisted this lady to gain substantial control over her movements and her 'freezing' in a very short period of time. It also resulted in her being able to lower her anxiety, read, return to doing fine crafts, and reduce her medication.

What was the functional neural effect of adding the NF? Our aim had been to influence SMR and to influence affect (anxiety) by training over BA 24, but she achieved not only motor control but also improved mood and executive functioning/attentional improvements. The answer as to why these effects were achieved has to be due to network properties in the brain. "The typical BA is differentially engaged in 40% of behavioral (cognitive, perceptual, emotive) domains" (Lloyd, 2007). What lies behind this observation is the fact that each Brodmann area is just one part of one or more neural networks that involve cortical–subcortical connections and coordinated activity with many other functionally related cortical areas (Thompson, 2007, De Ridder, Dirk, 2009, 2010). This may be one reason why practitioners have obtained good results when only doing NF over a single site such as Cz. Training at the Cz site may influence several neural networks possibly by having an effect on the anterior cingulate (AC) gyrus. Networks that involve the AC that were relevant for our client with PD include the executive (and attention) network for reading, the affect (and distress) network for anxiety, the motor control network for movement and fine craft work, and the salience network so that she could pay attention to the right things (ed's note: you can read more about these networks in the Thompson, Thompson, Thompson & Hagedorn article in the summer 2011 issue of NeuroConnections). The effects on the executive network may have occurred in part due to our addition of metacognitive strategies (part of each training session is done on task, with the task determined by client needs) and the effects on the affect network were almost certainly due to the combination with BF and an emphasis on attaining a calm, relaxed, open awareness mental state.

In order to have such wide ranging effects, the cortical area to which we are directing our NFB, must have a way of connecting to many functionally related but distant cortical areas while, at the same time, inhibiting all other non-relevant functional areas of the cortex. Action, whether motoric or cognitive, must be focused in order to be effective. How is this accomplished? It would appear to be that a particular network is affected and many functionally related cortical areas are synchronized in that network to accomplish the task at hand.

In order to better understand the concept of networks, the reader should remain aware that, although long distance cortical connections are probably involved, these connections are essentially excitatory. What then is a possible mechanism that could be responsible for activating a single network while inhibiting the non-relevant cortical areas in order to focus attention and action on a single function to accomplish the task at hand? Although gamma synchronization of basket cell inhibition of pyramidal cells may be one key

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Improving Quality of Life continued from page 20

for understanding cerebral dynamics, these cortical connections to inhibit pyramidal cells are short distance. There must, therefore, be another inhibitory mechanism and, indeed, this focusing of action while simultaneously inhibiting areas that are not necessary for a particular task seems to involve subcortical structures that are broadly labeled the "basal ganglia." The structures that comprise the basal ganglia include the striatum (putamen and caudate), the nucleus accumbens, the globus pallidus and the substantia nigra (pars reticulata and pars compacta). You will also hear another term 'lentiform nucleus.' The lentiform nucleus comprises the putamen laterally, globus pallidus medially and the innominate substance, which contains the anterior perforated area, inferiorly. The diagram below shows the red nucleus, the basal ganglia and the substantia nigra. It is in the substantia nigra that dopamine is produced and the death of those dopamine-producing cells produces the symptoms called Parkinson's Disease. (Nucleus of Luys is another name for the subthalamus.) The right insula (shown) and the left insula (not shown) are, with their connections to the nucleus accumbens and other structures, part of the affect network. The affect network is shown in Figure 3.

Affect Information Flow

In simplest terms, in figure 3, information related to "affect" traverses from the orbital frontal cortex (OFC), medial frontal cortex, anterior cingulate gyrus (ACG), hippocampus (HC), amygdala, and entorhinal (ERC) / uncus area to the basal ganglia, including the nucleus accumbens and ventral pallidum, and from there to specific functionally related areas of the thalamus, such as the medial-dorsal and anterior nuclei of the thalamus. The thalamus then connects back to areas of the cortex, including the anterior cingulate, which has functions related to control of the affect network. The result is regulation of mood and emotional reactions (after Kropotov, 2009).

When a cortical area is stimulated, such as the anterior cingulate gyrus in our example, it will stimulate a specific area of the striatum (putamen or caudate). That area will then, by a process called 'lateral inhibition,' inhibit all other areas of the striatum so that they do not fire and inhibit the globus pallidus (GP). The GP is like a functional map of the cortex. Its background firing rate is very rapid and it inhibits the thalamus. However, the one area of the GP that is inhibited by, for example, the putamen, will not inhibit its corresponding specific functional area of the thalamus, opening that thalamic gateway to all functionally related areas of the cortex (Kropotov, 2009). This may intensify a selected program of action which involves many, often widely separated, areas of the cortex while allowing inhibition by the GP of all other areas of the thalamus to continue. (It is a little hard to follow but think of it like a double negative making a positive: inhibition of inhibition leads to activation.) This series of steps underlies the production of brainwaves that are influenced by thalamo-cortical loops. Re-setting the rhythms of those thalamo-cortical loops, such as increasing rhythmic activity across the sensorimotor strip in the 12–15 Hz range, is often the goal when doing operant conditioning of brainwaves. As seen in our client with PD, changing the brainwaves through NF is accompanied by changes in behavior.

Conclusion:

NF training over the central midline structures such as the anterior cingulate, especially when combined with heart rate variability training, may have profound effects on complex networks that involve functions related to affect, executive, motor, salience, and default networks. This is likely the reason that our patient with advanced Parkinson's Disease and dystonia increased her quality of life, becoming able to function at a much better level and regaining her ability to control symptoms, including dystonic movements and 'freezing', and to again read novels and do highly skilled fine crafts and art work.

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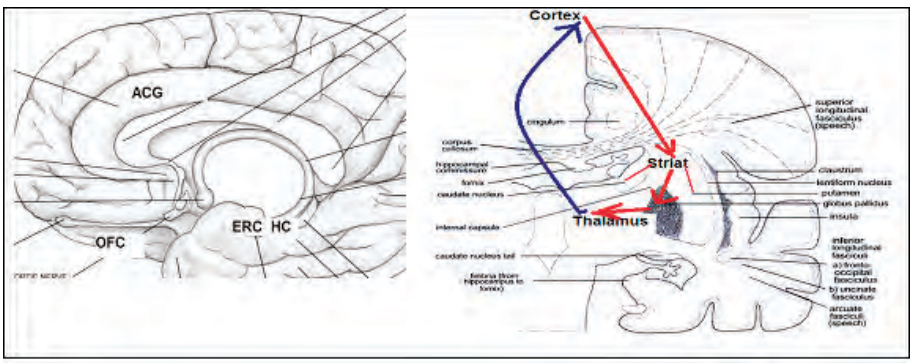

Figure 2: *Schematic Diagram of a Transverse Section showing the Right Cerebral Hemisphere and Midline Structures (from Wikipedia)*

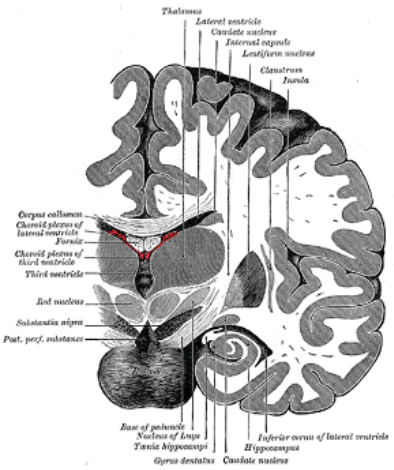

Figure 3: *Overview of main structures in the Affect Network: Mid-sagittal and Coronal Views (Drawings by Amanda Reeves from The Neurofeedback Book, 2003)*

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Working with Traumatic Brain Injury using the Low Energy Neurofeedback System (the LENS)

Len Ochs, PhD

While there have been many uses of the Low Energy Neurofeedback System (the LENS) to increase self regulation, there is probably no problem easier, more rewarding to work with, and more rapid to respond than mild-to-moderate traumatic brain injury (TBI) (Hammond, 2007; 2010). It is important to understand that the significant and rapid effects from the use of the LENS have been seen primarily when the TBI is preceded by high levels of functioning, and without other co-morbidities. This paper presents a brief discussion of the LENS, how it is designed to work, and some details of how the LENS improves self-regulation with TBI. This paper presents my clinical viewpoint, as the developer of the LENS, and not necessarily the viewpoint of OchsLabs, Inc.

The LENS, defined:

As the Low Energy Neurofeedback System the LENS uses the same electrodes, electrode application techniques, EEG amplifiers, and EEG monitor displays as any other neurofeedback application. (Ochs, 2007) (Bland, 2000)

The LENS feedback travels back to the person over the same electrode wires that convey the EEG signals to the ordinary (unmodified) EEG amplifiers, rather than using a visual EEG display or sound for feedback purposes. The LENS often yields surprising results in the rate at which they are observed.

There are several ways in which the LENS differs from other neurofeedback techniques. Each of these will need to be independently verified of the next few years. These are:

1. The LENS feedback strength is below the threshold of perception and is neither visible nor audible.
2. Even though the feedback is not perceptible to the client, the EEG changes in response to these signals. Low EEG amplitudes rise and become more variable; high EEG amplitudes drop, and tend to show reduced variability through the use of the LENS approach.
3. The EEG signals traveling back to the client are profoundly week, a million times weaker than the signals from the Alpha Stim, for example, and can be as brief as .01 seconds long (and up to perhaps 60 seconds in length) at any one sensor site. While feedback of this brevity and weakness are often difficult to think of as feedback, and while feedback information that is not perceptible is usually not thought of as feedback, the feedback information is a function of the client's dominant frequency at any moment at that site, and if removed from the system, the results almost completely stop – showing the importance of the feedback signals for the regularly obtained results. (Bland, 2000)
4. Some people think of the LENS as a stimulation system. While our research shows that neurofeedback amplifiers having digital circuitry can produce some stimulation, the stimulation of these amplifiers is so weak that the signals emanating from them approaches the level of weak electromagnetic fields having the strength of a few photons, and subject to scatter by the molecules of the soft and bony tissues of the head. (Bland, 2000, Mobley & Vo-Dinh, 2003) There is no evidence that the signals from ordinary EEG amplifiers used in neurofeedback are strong enough to penetrate the skull.
5. The sensitivity of the LENS client determines the duration of the feedback at any particular sensor site, with longer episodes of feedback being reserved for less sensitive clients.
6. The EEG feedback, while neither visible or audible, nor penetrating into the tissues of the brain due to their weakness and brevity, are still thought to be detectable by the brain as events on the skin and detectible by the extreme sensitivity of the brain to skin signals resonant with its own activity. One of the procedures used by the LENS approach documents the differential responsiveness of the brain to the LENS feedback in contrast to whatever subliminal stimulation may be present just by using an electronic amplifier.
7. The LENS uses both one and multi-channel feedback, as appropriate. The sites used in treatment are determined from the report using a single-channel topographic mapping procedure that sorts the sites in a number of ways to linearize the non-linear systems and subsystems of the brain.
8. The LENS uses topographic brain maps to indicate where the clinician is to place the electrodes on the head, and in which sequence. As part of the LENS Foundations and Advanced trainings, we define the decision process for choosing which part of the maps to use as the source of guidance on electrode placement.

Case Report:

This is the case of a 22 year-old male who was initially said to have been injured playing sports. He was said to be mildly head injured. He dropped out of high school and was basically supported by a friend, unable to hold any job due to his violent nature. He attributed his inability to graduate from high school to his poor attention, memory, and reading skills. He said friends and family shunned him; they were afraid of him because of his unpredictably violent nature. He occasionally held jobs as a bar bouncer. The only thing he saw that he was good at was hurting people. He was a tall, heavy, young man who smiled readily. He was taking no medication.

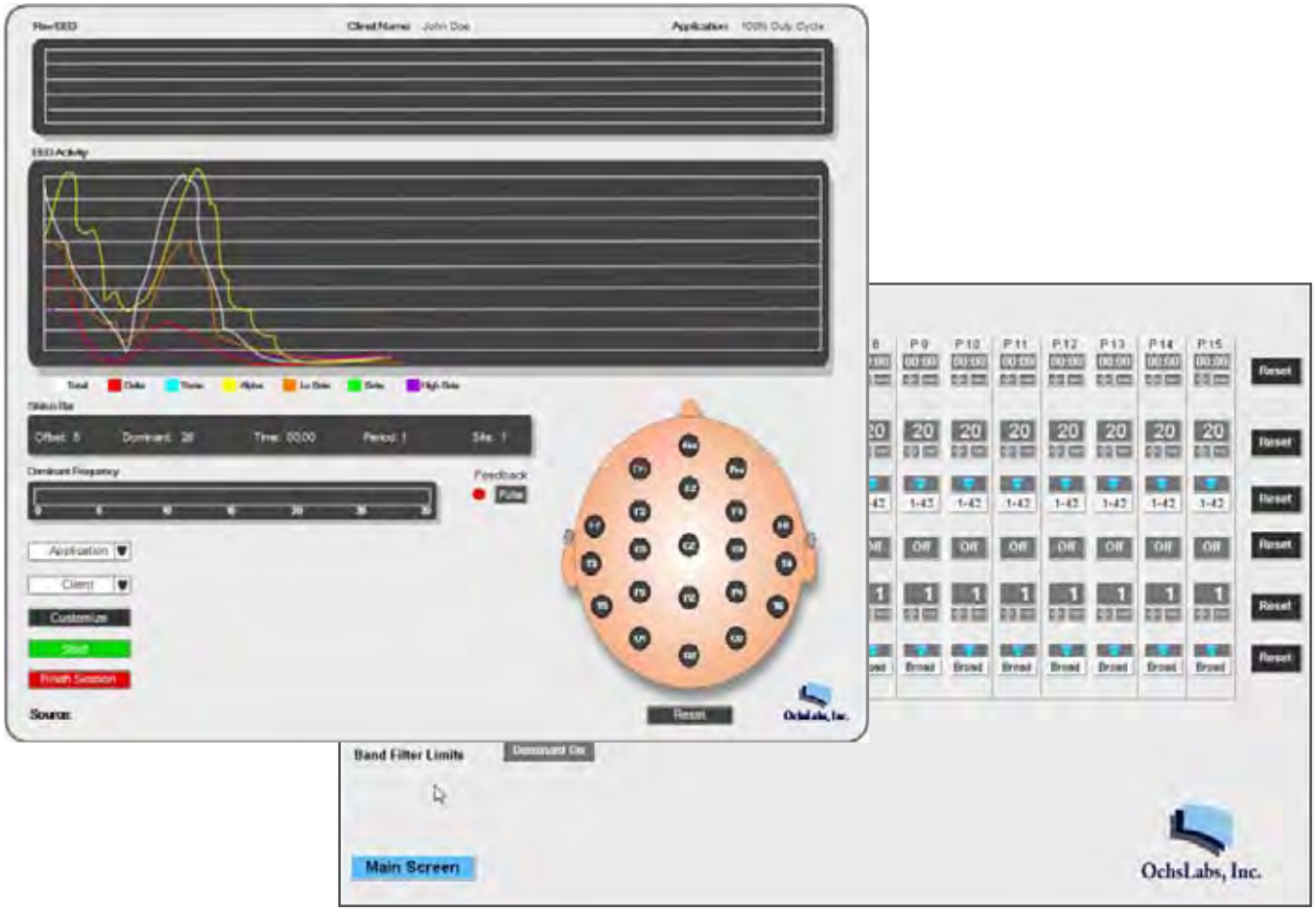
He was difficult to involve in treatment. He made several appointments, but "forgot" them. He finally admitted that he was reluctant to engage in a treatment process because he was "sick of telling the same old story without going anywhere." He admitted that a friend offered him a motorcycle if he would enter treatment and so he did. It was amusing to have him finally show up for treatment, only to watch him launch himself into his "same old story." When I interrupted him to ask to not tell me his story he appeared astonished and asked what else was possible. I said that we might try some feedback sessions, instead.

As he appeared quite concrete in his language and stories about himself, and as he appeared to have trouble taking in, storing, retrieving and using in-context information, I decided that he might benefit from long exposures to the LENS feedback. I then did several evaluations on him, concluding an entire LENS map, in a single session. The below map is not a qEEG map, as it used a single active sensor that sequentially sampled each of the 10-20 sensor sites, and also FPz and Oz. No simultaneous data is recorded, and no information on measured interrelationships among the sensor sites is available. The data is not subjected to discriminative database analysis. The maps are intended only for linearizing the non-linear systems and subsystems of the EEG to inform the LENS provider where to place the electrodes and in which sequence. The mapping data appears in Fig. 1:

The above maps represent the first set of maps from the head-injured client. The second set of maps below represents the second and final set of maps from the same client. To compare the two sets of maps, compare the set of maps on the left in Figure 1 with the set of maps

Continued on page 24

LENSware 2



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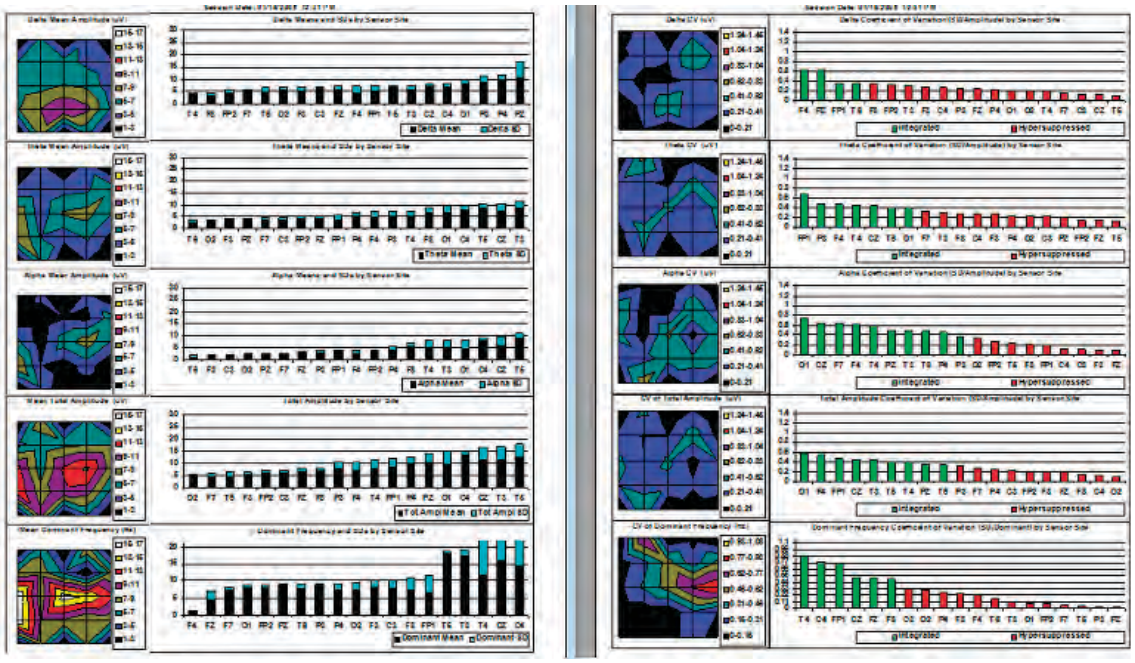

Figure 1

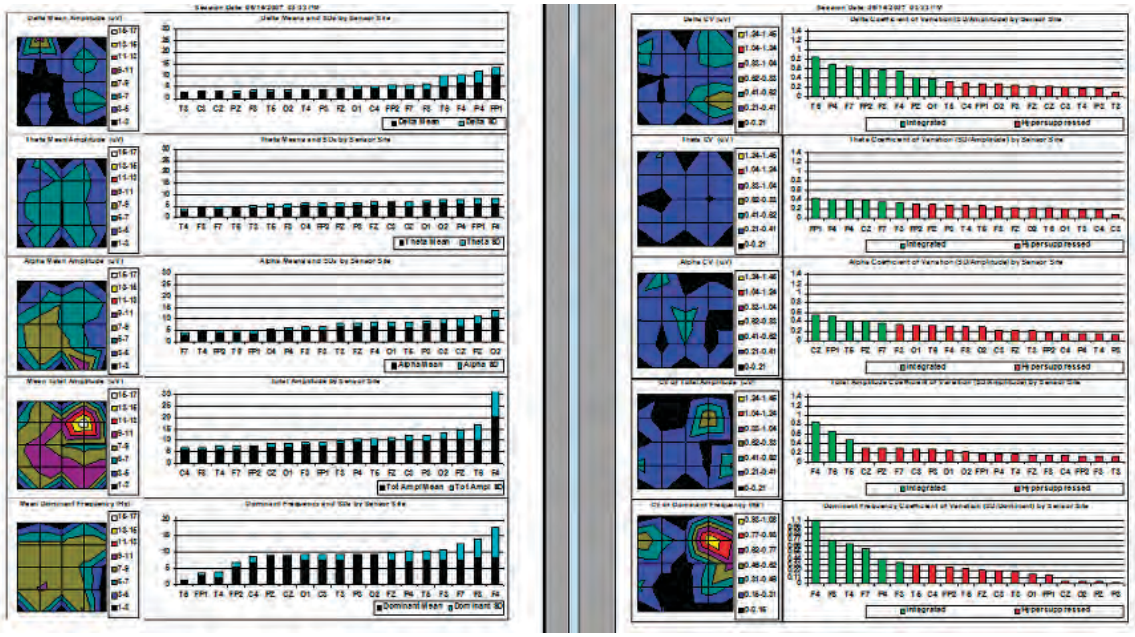

Figure 2

on the left side of Figure 2, and the set of maps on the right in Figure 1 with those on the right in Figure 2. Ordinarily those with mild traumatic brain injury complete their LENS experience in approximately 6 sessions if they were high functioning prior to their accident. However at approximately 10 sessions the client blurted out that while, once in treatment, he could see that his explosiveness was going to remit, he never expected that he would become happy. He also said that he had been suicidal before starting treatment because he could not tolerate the isolation of people fearing him, or seeing him so severely physically injuring others to the extent that he did. He also added that being hit in the head was an accepted way of interacting and growing up in his family; nobody thought anything of it as all the children were beaten equally. It took a total of 19 sessions to finish LENS treatment to his satisfaction, at which time he announced that he had been accepted into underwater welding school, was about to graduate, and had a job in Hawaii; he would be traveling there immediately to work under water on a vessel's cracked hull. Although that was the last I saw him, I heard from him recently—four years after he finished his treatment. He said that he was now a supervisor at the same company he went to work for at the finish of his LENS sessions. It almost goes without saying that at least some of his learning problems were no longer at issue, although maturation may also have played a helpful part in his increased vocational competency.

Some comments are due about the initial and final LENS maps. The square topographic maps on the left in each block are surface EEG maps made with a single channel EEG.

The active lead is moved to measure the EEG activity at each sensor site. A LENS Report Generator assembles the data as pictured. To the right of the topographic maps is a set of bar graphs, the order bar (sensor site) of which dictates which sensor site is to have the active electrode applied to it in treatment, and in which sequence. In the bar graphs, the black part of the bar represents the site's average amplitude. The light blue portion of each bar represents the average variability at each site as the measured average standard deviation from the mean at any site.

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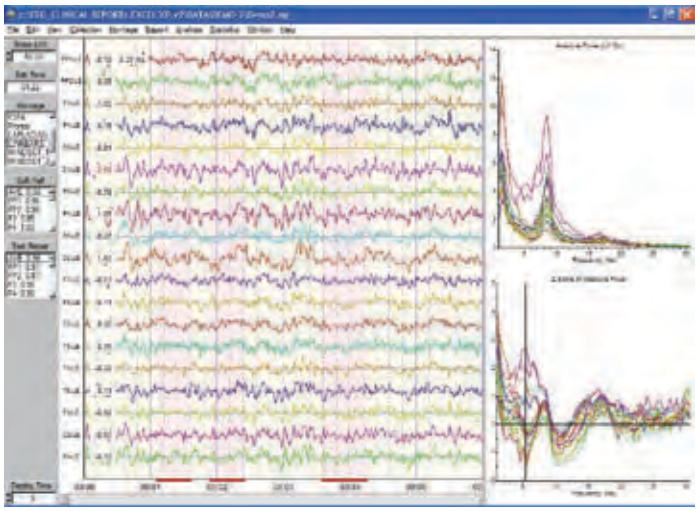

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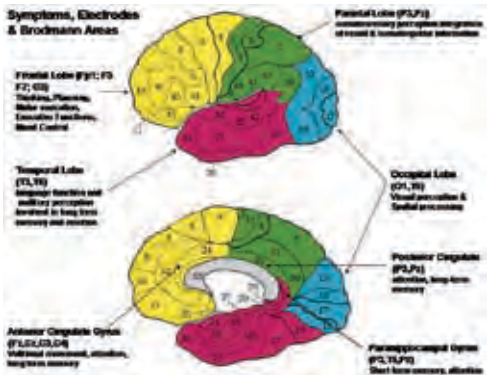

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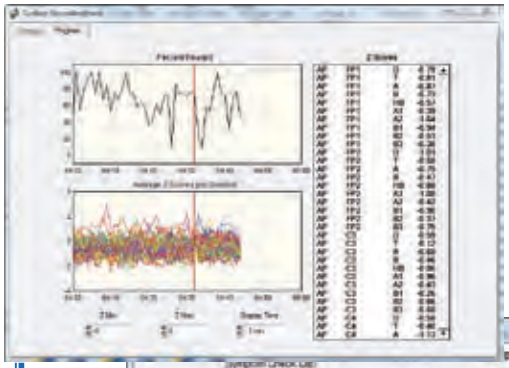

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LENS continued from page 25

From top to bottom, are Delta, Theta, Alpha, Total Amplitude (1 – 40 Hz), and Dominant Frequency band activity. The left-hand block of graphs in each set of maps represents average amplitude and dominant frequency information, while the right-hand block of graphics represents the coefficient of variability at each sensor site, with the bar graphs on the right again representing another possible site-sequence sort that dictates another potential critical path for linearly navigating through the non-linear systems and subsystems of the brain's EEG activity.

The only purpose of these graphics is to indicate the critical path of sensor site placement and the sequences of such placements. It is not the purpose of these topographic maps to use data-based discriminant analyses for drawing diagnostic distinctions. A more detailed discussion of how the LENS maps are used is beyond the purview of this paper.

It is important to remember that the brain is not a disembodied location of electrical impulses. A living brain is, instead, a mass of wet tissues surrounded by liquid-filled bony skull. Rather than use neuropsychological function as tied to the sites of cortical or subcortical nuclei, and rather than even thinking about how sensor site placement affects the neurology at any site, the LENS approach in general, may be more concerned about how puddles of liquid inhibitory neurotransmitters are maintained in the tissues of the brain, which kinds of neuronal activity are needed to give them shape and size, and how the neuronal functioning can be changed to dissipate those pools of liquid inhibitors so that connectivity can be re-established. The LENS may influence the electrophysiological activity underlying production, distribution, and maintenance of the inhibitory neurotransmitters, a hypothesis that may guide future research.

The bar graphs are merely linearized picture-transforms of the topographic maps to their left. They are another way of picturing the several sets of contours in the topography maps so that the clinician has a rational for choosing and placing electrodes on the scalp's sensor sites. By characterizing the topographic activity as a series of bars representing site amplitude and standard deviation, one might be looking at the sensor sites as family members varying in their energy and activity, each having relationships with the others. The bars are rank-ordered from least to highest amplitude. We might, in fact be addressing the family members with the least energy first, and moving right, progressively to family members with greater and greater energy. It certainly appears that working in order of increasing energy from site to site affects others in that site-sort; the sites with greater amounts of energy are affected well before the electrodes are ever directly placed on them.

And while a great deal of discussion might be heard comparing the first set of maps with the second in this case of head injury, for the purpose of this paper it is important to know that all of the topographic information is ignored in the decisions about sensor site placement. For example, if one looks at the Total Amplitude site sort to the right of the top-most topographic maps, the O2-F7-T6-F3-Fp2-C3-Fz sequence was the sequence of the first set of active sensor-site placements when using the map to provide direction for treatment. It is always arguable whether this is the optimal set of sensor site placements for this individual; however a 19-session course of treatment for this client with mild-traumatic brain injury, which took him into a rapid educational program and back into the work force, and which brought him higher, happier functioning, seems not altogether an inefficient evaluation and treatment program.

How the LENS is hypothesized to work: (All of these hypotheses need scientific, objective confirmation or disconfirmation, which will happen as the development of new research tools is completed.)

Several elements of the LENS were designed to reduce or eliminate the chances that seizures will propagate.

1. Destructive interference: The LENS feedback was designed to destructively interfere with the self-perpetuating brain-generated defense mechanisms that interfere with connectivity in the brain. This is not the kind of destructive interference of normal physics involving wave cancellation. This "destructive interference" comes as a result of the LENS feedback being at once unequal to the momentary dominant frequency and yet faithfully reflecting movement in the dominant frequency.

   It is hypothesized that there are brain mechanisms that perpetuate the inhibitory neurochemical barriers in the brain. These barriers appear designed to protect the brain from seizure. (Holt, 1997) The inhibitory neurochemicals that make up the barriers are commonly dumped into the tissues to create firewalls to prevent the spread of seizures. These firewalls either prevent the connectivity through neuroinhibitory blockade, or lock up the physiology to reduce the connectivity that permits higher functioning. It is further hypothesized that the LENS feedback interrupts the self-perpetuated barriers to better functioning. Once the neurochemical blockade is interrupted the LENS feedback process is discontinued to allow the brain to establish or reestablish the adaptive self-regulation needed for its own functioning.

2. The Offset: The LENS feedback generated is based on the running average dominant frequency to which a fixed offset (in Hertz) is added. Thus the feedback to the brain never exactly equals the dominant frequency, and in this way, is made an inaccurate reflection of the brain's activity. Yet the feedback dynamically reflects any changes in the dominant frequency. As suppression of the EEG diminishes, low amplitude EEG activity increases in magnitude, while high amplitude activity drops.

   a. The Offset frequency is shifted away from the dominant frequency. Moving the LENS feedback away from the dominant frequency appears to reduce the high amplitudes associated with seizure frequencies. It was also hoped that the effect of the offset would be to disrupt the immovability of the dominant frequency by re-distributing the energy across the spectrum, and increasing the excursion of the dominant frequency.

   b. It was noticed that 90% of the clients using the LENS approach displayed EEG slowing. The effect of the offset was also to reduce the EEG amplitudes in the energy hump constituting the EEG slowing, thereby raising the average EEG frequency, which often accompanies higher functioning.

3. Self-Regulation: The LENS, itself, does not establish or re-establish connectivity in the brain. It is hypothesized that forming neurochemical connectivity is left to the brain's own self-regulatory capability. The LENS provider does not micromanage or shape the way the brain puts itself back together again. The brain's capability to re-establish its own plasticity and adaptiveness is believed to be innate, given that it is not crippled by some of its own attempts to protect itself.

4. Timing: The feedback pulses from the LENS is metered out in tiny amounts, and not delivered continuously as in traditional neurofeedback. I hypothesize that small amounts of tiny signals are all that is needed to break the established maladaptive patterns of response in the brain by disrupting the timing involved in the neurochemical shutdowns. I believe this accounts for why such weak and brief signals can so rapidly bring about such large changes in the EEG.

Background on Head Injury:

It is often thought that head injury results in injured brain tissue, including the oft mentioned "diffuse axonal shearing" and "torqueing at the midline," as if these are the operative elements bringing about consequent degradations in functioning. It has also been observed that post-traumatic acquired head injury often leads to decreases in awareness, ability to take in information, and concreteness and rigidity of functioning and thought. The return to work of those with mild-to-moderate traumatic brain injury in as little as six sessions, when the client has had high premorbid functioning as well as no comorbidities even when out of work for three-plus years, has led to my working hypothesis that the relatively

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little work needed to be done except the disruption of blockages. I further hypothesized that these blockages to connectivity were put into place by the brain as a protective mechanism to interfere with any anticipated seizure activity that arose from the injury.

The consequences for the use of the LENS approach are as follows:

1. Because the person is shut down from the injury and function is compromised, reasonably strong feedback from the LENS needs to be used initially. This translates into using feedback with a stronger pull on the physiology of the dominant frequency.
2. As functioning begins to increase, the hypothesized interference to connectivity dissipates. Information (i.e. feedback, in this instance) becomes more impactful. The strength of the feedback signal can be adjusted by making adjustments in the offset. The feedback's weakened pull on the dominant frequency becomes a significant part of what is needed to decrease symptom intensity.
3. Once the LENS feedback sessions are stopped, function can be expected to improve as long as premorbid functioning was high and there were no comorbidities.

Discussion:

1. The LENS approach appears to function as a catalyst, providing signals that allow the person to become once again self-regulating and adaptive.
2. The transition from dysfunction to functional is non-specific and not targeted toward traumatic brain injury, or toward the remediation of any diagnosis, for that matter. Instead, the LENS appears to be assisting and supporting the physiology underlying the EEG to once again become adaptive. The transition from dysfunctional to functional is more a characteristic of the brain's own adaptive and plastic processes, rather than due to any controlling or micromanaging of the brain's EEG by the LENS provider.
3. The LENS approach appears to be less directive than is traditional neurofeedback, as it lacks any selective reinforcement, threshold management of reinforcements, or inhibitory influences common to traditional neurofeedback.
4. While there may have been some fears from clients that they were structurally changed by the LENS and its antecedents, they found in every case that either other factors were at play or that the results were functional and transient.
5. It is hypothesized that the LENS approach works by releasing the neurochemical suppression on the physiology that blocks connectivity. It uses signals dynamically resonant (i.e. feedback signals) with the EEG that disrupt the neurochemical blockades to connectivity. The weakness and brevity of the LENS signals might only disrupt the timing signals necessary to perpetuate the blocks to functioning; perhaps explaining how such weak feedback signals can trigger such radical improvements in functioning and changes in the EEG.
6. There is no evidence to date that any of part of the LENS feedback signals are strong enough to surmount photonic scatter to penetrate the tissues and organic and inorganic molecules of the head – any differently than any of the signals ordinarily emitted by any microprocessor-based EEG's signals do. These effects appear not to have been studied outside of the study done on the LENS equipment using J&J EEGs. However while the signals emitted from EEG amplifiers used in neurofeedback are substantially weaker than FDA limits, there is clinical evidence from both clients and therapists using other EEG amplifiers that the training response when the LENS is implemented with other EEG amplifiers can be even stronger than those produced by the J&J.
7. The LENS is an approach that needs research to deepen confidence in it. It needs verification that LENS signals can alter the EEG. There needs to be even clearer physical description of the different signals that are used within the LENS process. Descriptions of the different signals are now clearer and are taught in the LENS training programs, and will appear in a forthcoming NeuroConnections article if this is of interest. The exact method of generating these signals will probably remain a commercial trade secret of OchsLabs, Inc. However the quantified signals can be defined and made public. Finally, outcome studies can be expected within the next few years to also verify that the LENS has merit beyond the claims by OchsLabs, Inc. Until this information appears, it is the unabashed interest to enhance the happiness of the clinicians using the LENS, which can only happen when the providers using it become increasingly skilled at enhancing their clients' self-regulatory skills and happiness.

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Disclosure: Len Ochs is a consultant to OchsLabs, Inc., he receives no income from OchsLabs, Inc., holds no ownership of OchsLabs, Inc, and holds no administrative or directional control in OchsLabs, Inc.

Bio: Len Ochs, Ph.D. continues to develop the Low Energy Neurofeedback System.

Two Channel Low Energy Neurofeedback System and NeuroField with Treatment Resistant Depression: Preliminary Observations

Sara Hunt Harper, PhD and Jill O'Brien, DOM, LAc

Treatment Resistant Depression (TRD) is one of the major issues effecting health care providers today. Although there is no uniformly accepted definition of TRD, there is concern that this population's needs are not being met. (Shelton, Osuntokum, Hemloth, & Corya, 2010). The Sequenced Treatment Alternatives to Relieve Depression (STAR*D) offers guidelines for treatment but found no treatment to be a stand out for TRD and suggests that after two unsuccessful medication treatment trials, there is a significant decrease in likelihood of remission. (Gaynes, Rush, Trivedi, Wisnieski, & Feva 2008). Up to 15% of patients with depression qualify as treatment resistant when defined by two trials of antidepressant medication from different classes of medications with no significant reduction in symptoms. (Berlim and Turecki, 2007).

In the United States there is a lifetime depression prevalence of 13 percent. (Hasin, Goodwin, Stinson, & Grant, 2005). Frequently the first treatment of choice is medications. Of these first line psychotropic drugs, approximately one out of three patients will not respond (Rush, 2007). After several trials of adding more drugs or switching drugs, only 25-50% obtained remission of symptoms. (Connolly & Thase, 2011). At this point more medications are added, subtracted, added again, combined, and whatever new drug on the market is tried. When the medication approach does not work, the patient complains of too many side effects, the patient has switched from doctor to doctor trying to find relief from the symptoms of depression, and/or just plain refuses to take any more drugs, other options are considered.

Psychological therapies are frequently recommended for depression as a stand-alone treatment or in combination with medications,. Little information is available on use of these modalities with TRD. Cognitive therapy (CT), interpersonal therapy (IPT), problem solving therapy, dialectical behavior therapy (DBT), Acceptance and Commitment Therapy (ACT), and mindfulness based cognitive therapy (MDCT) are accepted empirically based psychotherapies and yet, results regarding their effectiveness with TRD are sparse and mixed (Trivedi, Nieuwsma, Williams, & Baker. 2009).

Several non-pharmacological strategies for TRD focus on brain stimulation. Electroconvulsive therapy (ECT) is one of these strategies. However, there are known side effects that may be intolerable to many patients (Fink, 2001) as well as a high relapse after ECT is stopped even with continued medication (Sackeim, Haskett, Mulsant, Thase, Hann, Pettinati, et al, 2001). Vagus nerve stimulation (VNS), an implanted brain stimulation device, was found to be ineffective one year after FDA approval (Cristancho, Cristancho, Baltuch, Thase & O'Reardon, 2011). Repetitive transcranial magnetic stimulation (rTMS) with TRD has produced mixed results. After 15 years of use, rTMS appears to produce only a "modest" response to treatment (Fitzgerald & Daskalakis, 2011).

As documented by the above review of literature there appears to be no universally accepted modality for TRD with data demonstrating a consistent long-term elimination of symptoms. In our offices we explored the use of Low Energy Neurofeedback System LENS (Ochs Labs, Sebastopol, California, USA) and NeuroField (Bishop, California, USA) as a tool for the relief of symptoms of TRD several years ago and have observed some interesting results. For these observations in our practices we used the LENS J&J C2 Plus 6 and the NeuroField 1000. The LENS is available with BrainMaster Atlantis, Bio Explorer, and OchsLabs interface. NeuroField is only available now as NeuroField 2000. The new NeuroField Plus was not available when this protocol was developed.

Neurofeedback Equipment

LENS is a type of neurofeedback equipment which sends a tiny electromagnet signal through traditional feedback sensors placed on the head or body and is a FDA-registered Class II medical device, 510-K exempt. Developed by Dr. Len Ochs (Ochs, 2006), there is ample evidence in the literature about the effectiveness of this style of neurofeedback (Hammond, 2007, Harper, 2009, Larsen, 2006). The authors have been using the LENS in independent private practices for nearly ten years with Dr. Harper at the Stress Management Center in Plano, TX and Dr. O'Brien at Blue Water Healing in Peoria, AZ. Through sharing information at conferences, workshops, emails and telephone conversations, they expanded LENS from a monopolar system to a bipolar/sequential system. As they begin to experiment with two channel LENS placements, Dr. Nick Dogris was asked to write new LENS protocols for this application and Dr. Cory Hammond was asked to validate the placements and come up with suggestions to improve the protocols. The results of this cooperative endeavor were published as advanced LENS protocols. (Hammond, Harper, O'Brien, & Dogris, 2010.)

NeuroField is a stress reduction device that sends low intensity electromagnetic stimulation to the body and was developed by Dr. Nick Dogris (Dogris, 2009). Described as low intensity transcranial magnetic stimulation (TMS), NeuroField is 1/10,000,000 weaker than regular TMS while the electromagnetic fields (EMF) output of the cap can be measured between 1-3 milligauss (Dogris, 2011). There are over one hundred protocols developed by Dr. Dogris, Mike Beasley, M. S. of Neuropath in Austin, TX and others. At this time NeuroField is considered an experimental neurotherapy device.

Subjects

Patients were referred by their family/friends, previous clients, other therapists LENS and non-LENS practitioners, medical doctors, dentists, and chiropractors. Neither office accepts insurance or is on any insurance plans. All patients were self-pay at time of service either by credit card or check. Some were able to obtain insurance reimbursement with a receipt from the practice while others were not. Most had been seen over the years by numerous doctors, therapists, alternative health care providers for relieve of symptoms related to depression and stress, as well as anxiety. All believed they had a poor quality of life or no life at all. Most reported suicide ideation. Several had attempted suicide in the past. Complaints were sleep issues (either too much or not enough), fatigue, appetite issues (either too much or not enough), isolation- with concerns about their relationships, inability or diminished ability to work (either at home or outside home), too many side effects of numerous medications and concerns that the medications were not working. Four clients were referred by their physician before ECT. Other than these four, all other clients felt they had run out of options and were trying neurofeedback as a last resort. Several had tried traditional biofeedback years ago while

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three had seen other neurofeedback therapist using equipment other than LENS and NeuroField with limited results. Ages ranged from 14 to 83. Socioeconomically, the clients were from lower middle class to upper class.

Terms and Tools

LENS map: This was generated by following the 10-20 International System, linked ears at offset of 20, one site at a time with two seconds of hum, one second of feedback followed by one second of hum. The protocol used was Map C2Plus.

Sensitivity: Patients were evaluated for sensitivity, hardiness, and reactivity using the standard LENS guidelines.

LENS Site Sort: The LENS Report Generator arranges each electrode site in order from the most functioning to the least functioning generating one graph each of Delta, Theta, Alpha, Total Amplitude, and Mean Hz. For the purpose of this training the Total Amplitude site sort was used.

Intake Interview: Sara used the intake interview developed by Cory Hammond for QEEG evaluation (Hammond & Gunkelman, 2001) and expanded by him for use in clinical practice. Jill used an extensive intake evaluation which she has developed for her oriental medical practice.

Symptom Rating Form (Hammond, 2006): Sara used this form at the start of each session to set training goals and to track progress of symptoms. Jill used the rating scale every three to six sessions.

Progressive Session Procedures

We began with an extensive history of symptoms, previous treatments, current medications, nutrition, social support, family relationships, informed consent, explanation of LENS and NeuroField, evaluation of sensitivity, patient expectations, and treatment goals. This was followed by a LENS map at offset of 20. We began the site sort using LENS Total Amplitude Map (TAM). Where to set the offset at the beginning of training was a clinical judgment as was the number of sites to train each session. As our patients were hardy the offset ranged from 2-20Hz depending on symptom changes with the number of sites usually at 7 per session. After completion one time of the TAM and assuming the patient was doing well, we added NeuroField. The protocols to start may vary from depression, anxiety, 1-100, emotional calming/nurturing, Schumann wave, brain fog reduction, and improve attention. Assuming patients were experiencing no side effects, NeuroField dehabituation was added. Next NeuroField 10-10000 was added to the mix followed by 2 channel LENS at Fp1-T3 Fp2-T4 with Rocking 7-12 protocol. When the 2-channel LENS Rocking protocol was added, this protocol may be repeated in each session up to seven times until a change in the EEG pattern was seen. With the combination of 2-channel LENS and NeuroField, our experience was that symptoms of TRD were significantly reduced or eliminated within 1-6 sessions.

Cautions:

We want to stress that the above protocols are only guidelines for training. Over the years we have learned that no two brains are alike, neurofeedback is not a cookbook modality, and one size does not fit all. We caution listening to the client, following their lead and slowing down or speeding up training based on their response to procedures. Additionally, we believe it is important to remember that the clients we see are very hardy and that these protocols have not been tested on sensitive clients. We strongly believe that therapists new to LENS and NeuroField should not use these advanced protocols. Again, we developed these protocols using the J&J interface with LENS. Lastly, we want to emphasize that these protocols were developed for the symptoms of Treatment Resistant Depression and were only used with hardy patients when other interventions were not working.

Tomato Effect and Placebo Effect

Longtime practitioners of biofeedback may remember a marvelous book written by Shellenberger and Green (1986). In it they hoped that research on biofeedback would not be viewed as the Tomato Effect whereas a treatment or procedure is rejected by the psychological and medical community because is does not fit into the current mainstream model. Our hope is that new ideas, observations, and research involving neurofeedback are seen as viable options, worthy of investigation and consideration and not just another tomato in the basket.

Over the years the placebo effect has been both negatively and positively viewed regarding the power of biofeedback with much emphasis placed on the clinician. We would like to note that Sara and Jill have entirely different styles of working with clients, different types of clients, and live in different areas of the country. We come to this field from different backgrounds with Sara working initially as an RN and Jill quite successfully in the business world as director of operations for ATT Wireless Division before a career change to Oriental Medicine and advanced studies in China. Jill views patients with a focus on eastern philosophy and oriental medicine and Sara views patients with a western philosophy as a marriage and family therapist. Both look at the mind-body connection but with a different eye.

Conclusion and Need for Research Study

Our observations, feedback from patients, and continued referrals from medical and non-medical professionals lead us to believe that this combination of LENS and NeuroField holds promise as a viable option for TRD. Our next goal is to do a formal pilot study of these combinations using ABA design. Since the qEEG has become the gold standard in tracking changes in the neurofeedback community, we plan to obtain pre and post qEEG, add standardized testing with follow up at three, six, and twelve months.

Berlim, Flock & Turecki, (2008) suggests the need for treatment outcome research and as well as development of "novel therapeutic strategies" for TRD. Perhaps the combination of LENS and NeuroField might be considered one of these novel therapeutic approaches.

Sara Hunt Harper, PhD BCB-Senior Fellow is a Licensed Marriage and Family Therapist (LMFT) with a specialty in EEG Biofeedback for major traumatic brain injury (TBI), depression, anxiety, sleep, autism, CP, and ADHD. Dr. Harper, a pioneer in Animal Assisted Therapy, is assisted in her practice by two therapy dogs, Patricia Marie and Rebecca Marie, Elliot James, therapy cat, and Jacob James, therapy cat in training.

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Letter from AAPB NFB DIV President
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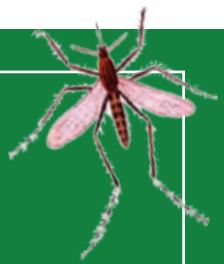
it. Those who have followed that field know that that is no longer entirely true. And neither is it true of our resting state networks. They can be probed with neurofeedback. The technique would be used to subtly induce state shifts, and the resulting changes in resting state functional conformation tracked. This would establish the sound science that would benefit this field. As for the clinical frontier, the research perspective should be broadened, not narrowed. Perspective is needed at top levels on the full dimensions of current diverse practice within the field. An anthropological approach should be taken in which researchers formally study the client-clinician dyad in its state of nature with the full variety of neurofeedback approaches currently flourishing in clinical application. It will be quickly appreciated that neurofeedback will alter the face of mental health profoundly. Given the current state of our society, that cannot happen soon enough.

Siegfried Othmer, PhD



The true meaning of life is to plant trees under whose shade you do not expect to sit.

—Nelson Henderson.

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Hershel Toomim

June 19, 1916 - July 19, 2011

Notes from the BCIA Chair

Fred Shaffer, PhD, BCB, BCIA Chair and Professor of Psychology at Truman State University,
and Judy Crawford, BCIA Director of Certification

I recently returned from a great annual meeting in a lovely desert setting outside of Scottsdale, AZ. I hope you enjoyed your time there as much as I did. If you missed it this year, don't make the same mistake in 2012! It was clear that neurofeedback is alive and well and full of the enthusiasm it needs to carry it forward to its rightful place in the health care community. On the way home, I considered what things I'd like to tell you about and so, in no particular order, here are some things that are important to BCIA.

BCIA has taken an international approach to certification. As was evident at the meeting, interest in biofeedback and neurofeedback is growing rapidly in many other countries. In fact, we are excited to let you know that our international efforts are going very well. More than 60% of the 2011 neurofeedback certificants are from outside of North America. The BCIA credential is now in 22 countries and growing! Many people have asked us why we broadened our focus. The goal for all of us is to promote the credibility and efficacy of the field, based on sound science and research. There is a great deal of cutting edge research and clinical work going on outside of North America. What better way to help support the field than by reaching out with education and training standards that go across geographical boundaries!

One new international program that BCIA is launching is an affiliate relationship with another country or geographical region. We are currently pursuing this relationship in Australia. The goal for an affiliate is to give each country or region the autonomy to help mold certification requirements, based on our blueprint and exam, into what would work best locally to reflect the current health care culture. We anticipate that we don't have all the answers in reviewing which universities are legitimate and which health care professions would be reasonably or legally allowed to practice neurofeedback. Stay tuned for more information on new affiliate relationships.

Education is critical to the promotion and survival of not only the field, but of BCIA as well. This is perhaps the most important work for all of us. Toward this end, we recently launched a formal Education Committee whose function is to oversee and enhance all educational opportunities, whether on the fundamental or the advanced levels. We hope to soon be able to launch a clinical update series which will feature low barrier, low cost, online CE opportunities that can be as simple as an update on research or news in the field, ethics, or a review of the fundamentals to help new clinicians gauge their preparedness for the BCIA exam. If you can suggest a topic or speaker you'd like to see in this venue, please let us know at info@bcia.org. These webinars will be crucial for our international certificants to have access to affordable CE to enhance their skills and maintain certification.

BCIA has taken an international approach to certification.

The top priority for this coming fall is a total program review for both biofeedback and neurofeedback. The hard-working PMDB Task Force is just finishing their work. Neurofeedback is changing rapidly and we want to ensure that our blueprint, reading list, and exam reflect the current accepted science, history, and theory one would need to demonstrate entry level competency. This is a huge task with several layers, but we are lucky to have dedicated volunteers who will break the task into smaller pieces to make it more reasonable. We hope that you are already aware that our own Genie Bodenhamer Davis now has a scholarship named to honor her many contributions to educational excellence in neurofeedback. This scholarship pays all BCIA certification fees for the winner who is chosen by an independent panel who reviews recommendations submitted by a university instructor where didactic course work is taught. In addition, they consider the letter written by the student telling us what they believe neurofeedback will mean to their clinical work. This year, we announced two winners at the ISNR Saturday evening gala. Please join us in congratulating Charity Finch and Noel Larsen, both fine candidates who will be a wonderful addition to the field. Charity Finch was trained at UNT and Noel Larsen at Southwest College of Naturopathic Medicine.

Not only is BCIA grateful to those who teach at a university, but also to those who teach as private vendors. Many of these have been our educational partners for as long as any of us can remember. New training programs are important to keep the field moving forward. We want to welcome two new training partners—Stress Therapy Solutions (STS) and ASET-The Neurodiagnostic Society (ASET). STS offers a unique international platform in that their fundamental 36-hour program is accredited to fulfill the didactic requirement and their staff includes many international BCN professionals who will bring this basic education to several different countries. ASET now offers 10 BCIA-accredited online courses that are perfect for the beginner or to be used to earn low-cost, low-barrier continuing education to be used for recertification. Both new training partners will be helpful to support our international outreach.

Another recent focus for BCIA is our venture into the world of new media. We are proud that our Facebook and Twitter accounts have really taken off, both guiding many new visitors to our website. Recent statistics showed that web visitation figures were up 34% from last year. If you haven't "friended" us yet on Facebook, please do so at @bcia1981. The only way for social media to be relevant is to keep it fresh with good posts, so please send us any "Facebook Fuel"—articles or notices that you think would be of interest to our readers—info@bcia.org.

Another recent focus for BCIA is our venture into the world of new media.

As you can tell, BCIA has a "working" board. Our board terms expire at the end of October each year. This year we will be saying goodbye to three people who have worked hard to support the value and mission of our credential—Susan Antelis, Anita Myer, and Aubrey Ewing. Please visit our homepage at www.bcia.org to read more about how proud we are to have worked closely with them. If you would like to contribute ideas or time and talent to further the value of our credential, please let us know. There is always work to be done to help us all stay "More than qualified—BCIA board certified."

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MindFull

Infinity and Continuum

David Kaiser, PhD

Except for a handful of midline structures, every region of the brain has a partner, a homologue that performs similar as well as different tasks. In the neocortex our homologue is the cortical tissue situated across the way in the other hemisphere at the same location we are located in our cozy corner of the brain, the right frontal lobe compared to the left frontal lobe, for example. Homologues are highly connected to each other, with callosal pathways that provide them an understanding different than that of their more locally connected neighbors. They may hear the gossip across town before they hear what's going on in their own house. Homologues are complementary partners, Bert-and-Ernies, flying squirrels to talking moose, a marriage of opposites. Each brain area excels at certain functions or stimulus types and performs poorly at others. Left and right brain areas in the same location of the cortex are complementary in function, as well as inhibitory, excitatory, and in competition with each other for specific forms of information (e.g., phoneme processing, face recognition, perspective taking). Some believe that homologues provide context for processing content, the surround that makes sense of the middle, background-to-figure. Others think they work opposite sides of the street, looking at the same information from divided perspectives. Diversity leads to discrimination as a team disambiguates sensory input. For example, the left primary auditory cortex is specialized for processing phonemes and behind the right ear is its homologue, a whiz at tones and sounds of nature. This division works well much of the time, allowing us to understand both meaning and intention of a communication. In EEG analysis we have eight homologues or site-pairs in the 10-20 system, and 31 pairs in the 10-10 system: F7-F8, F3-F4, P3-P4, AF3-AF4, etc. Brain damage at one site may slowly damage the homologue, the mirror site (Morrell 1960; et al., 1959). We understand the basics of this process of mirror site lesions, but there are many questions unanswered so it's anyone guess what it means to have partners in disarray, a convoluted mirror.

The role of homologues became a concern in my son's care. His EEG shows excessive delta activity at the right temporal lobe, tissue that much of emotional regulation and episodic formation is finalized away from sensory input. Cortical areas produce delta waves when isolated from sensory information, when they lose subcortical innervation. Delta activity is what we expect for infants, given their limited connectivity, and adults show it during deep sleep, but in the waking state we wouldn't expect this of a teenager, which means this part of his brain has not ripened, has not connected thoroughly with thalamic inputs. It remains infantile and as this area is partnered with the left temporal region via the anterior commissure and corpus callosum, it means his left temporal lobe is developing in many ways alone, in isolation, governed mostly by a feral partner.

Except for a handful of midline structures, every region of the brain has a partner, a homologue that performs similar as well as different tasks.

It took me many years to come to a clear model of cerebral hemisphere function. I knew most of the data and dozens of characterizations, and conceived the right brain as infinite, holding all varieties of personal experience in tow, while the left brain was infinitesimal in that it reduced each event or experience to its basic core. The left brain is expressive, the right receptive, and our world is a reflection of the brain, a balance between infinitesimals within the infinite. Georg Cantor, a 19th century mathematician, was the first to plumb the depths of an infinity larger than the infinity taught to school children, the continuum. He revealed two infinities, one of enumerable parts, elements we can count, and another of unspecifiable and uncountable stuff. The set of natural numbers is a countable infinity, and the set of real numbers is a continuum. In natural numbers we know what follows 2, and 3, and 4,703,182 and this never changes even if we count up to a googol, 1 followed by 100 zeroes, but it's not clear what follows 2.14. Is it 2.141 or 2.1401, or 2.140001, etc? The set of rational numbers (fractions, integers) is infinite yet has fewer elements than the set of real numbers. No two rational numbers can be close enough that a real number cannot cousin between them.

The concept of two infinities, each unlike to the other but similar in nature (endless) is my model of the cerebral hemispheres. The left hemisphere organizes the world into countable elements, dividing the world into knowable parts, and the right hemisphere organizes the world as a continuum, investing the world without parts, into wholes. In many ways civilization reflects increasing dominance of thinking of ourselves as parts instead of thinking of ourselves as wholes. I consider my son to be engrossed by the continuum more than infinity, his reals to my integers. Where I see parts, he sees wholes, where I enumerate, he finds no divisions. He is a mirror to my mirror, only larger and less static. As Plato said, "all things that are even said to consist of a one and a many, and have in their nature a conjunction of limit and unlimitedness." It is in the balance of this conjunction that we all differ, including each of us from our children.

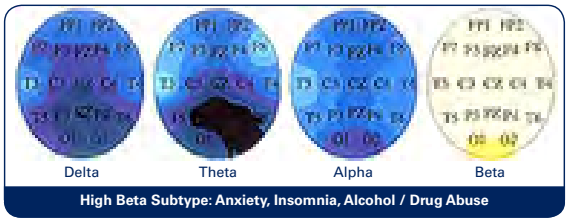
The left hemisphere organizes the world into countable elements, dividing the world into knowable parts, and the right hemisphere organizes the world as a continuum, investing the world without parts, into wholes.

References

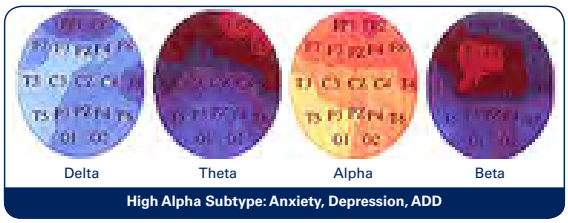
Morrell, F., Sandler, B. & Ross, G. (1959). The "mirror focus" as a model of neural learning. Proc. XXI Internat. Contress Physiol. Sciences, Buenos Aires, pp. 193.

Morrell, F. (1960) Secondary epileptogenic lesions. *Epilepsia 1:538-560.*

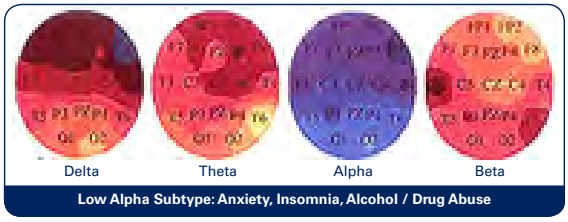
QEEG / TOPOGRAPHIC BRAIN MAPS:
Generalized Anxiety Disorder Subtypes



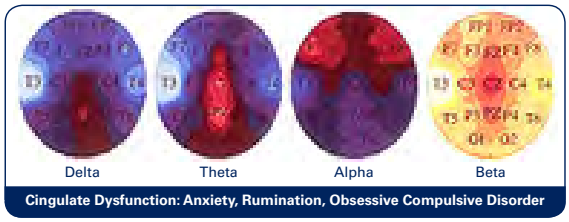
High Beta Subtype: Anxiety, Insomnia, Alcohol / Drug Abuse



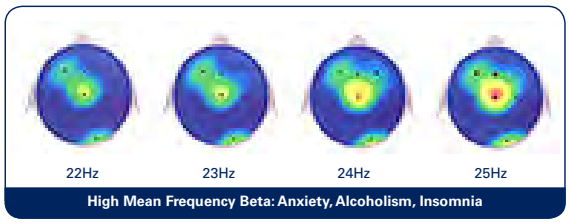
High Alpha Subtype: Anxiety, Depression, ADD



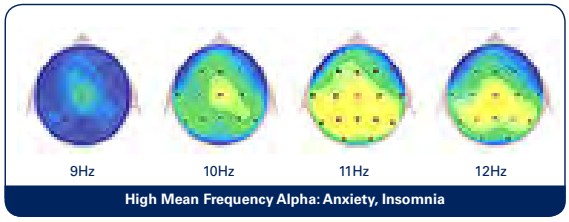
Low Alpha Subtype: Anxiety, Insomnia, Alcohol / Drug Abuse



Cingulate Dysfunction: Anxiety, Rumination, Obsessive Compulsive Disorder



High Mean Frequency Beta: Anxiety, Alcoholism, Insomnia



High Mean Frequency Alpha: Anxiety, Insomnia

AVAILABLE SERVICES

Full Package: #'s 1-7: minimum recommended for Neurotherapy *Includes electronic copy. Priority mail is $20 extra.*	**$225.00**
Full Package: #'s 1-6: Without report (1-5 only) *Includes electronic copy. Priority mail is $20 extra. If one database used the minimum is $75.00*	**$195.00**
01) NX Link - NYU/E. Roy John Normative Database (Eyes Closed) *A) NX Link Discriminant Analyses: ADD, LD, Depression, Memory/Dementia, Substance Abuse, Head Injury, Schizophrenia/Thought Disorders*	$70.00
02) EureKa3! - Nova Tech EEG LORETA Analysis System and Adult Normative *Database - Eyes Closed*	$70.00
03) Neuroguide - R. Thatcher Normative Database *A) Eyes Closed Linked Ears Z-scores // Eyes Closed LaPlacian Z-Scores* *B) Eyes Open Linked Ears Z-Scores // Eyes Open LaPlacian Z-Scores*	$70.00/each
04) Neurorep - W. Hudspeth QEEG Analysis System *A) Eyes Closed - Weighted Average, Z-scores, Magnitude, % Power, LaPlacian, Average Spectrum, coherence, connectivity* *B) Eyes Open - Weighted Average, Z-scores, Magnitude, % Power, LaPlacian, Average Spectrum, coherence, connectivity*	$70.00/each
05) Thatcher TBI Discriminant Analysis and Severity Index	$70.00
06) Thatcher Learning Disabilities Discriminant Analysis and Severity Index	$70.00
07) Clinical Correlations and Neurotherapy Recommendations by Bob Gurnee	$70.00

total value: $630

08) Conventional Medical EEG - Read by Neurologist	**$125.00**
09) EureKa3! – Nova Tech EEG LORETA Analysis - Eyes Open-Non Database	**$70.00**
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MSW, BCIA:EEG, QEEG Diplomate, Director

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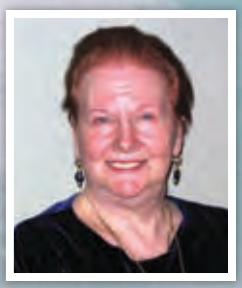
Ride the Waves

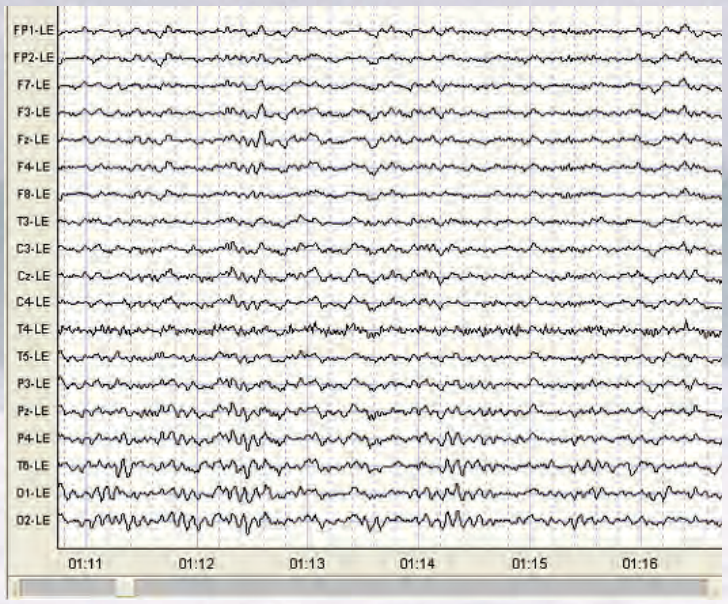
This client is a male, right-handed 23-year-old who had graduated from college a year ago. The first two raw EEG images are of eyes closed and eyes opened conditions. The third is raw EEG images generated during LORETA training for the symptom of executive functioning 10/13/2011. Have fun figuring out what these images tell you about the symptoms and even what treatments might be useful!

Send your answers to us at cynthia@isnr.org or merlyn@nyneurofeedback.com

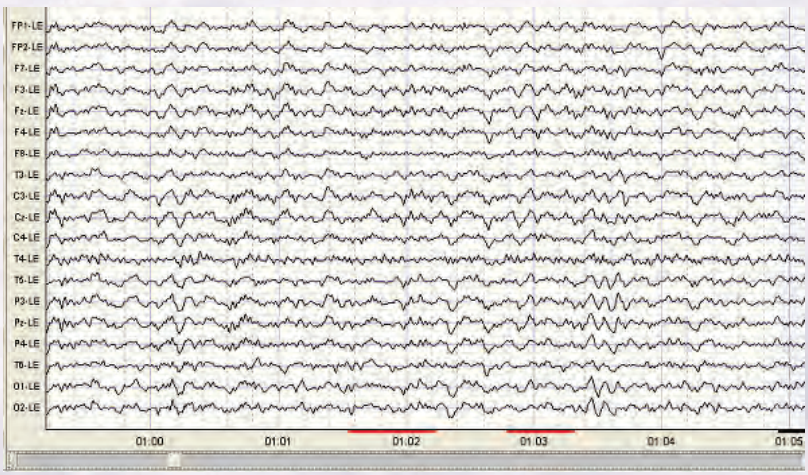
Look for the responses and the correct answer in the spring 2012 issue of *NeuroConnections*.



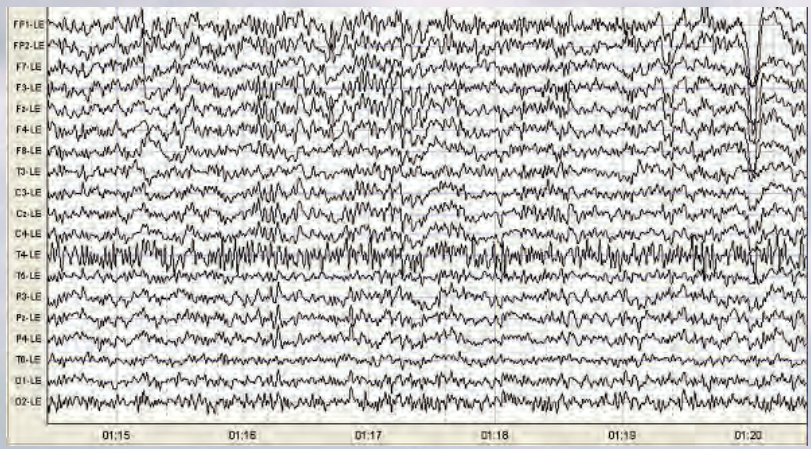
Merlyn Hurd, PhD



Eyes Closed



Eyes Open



Same client during last week
LORETA training
eyes open