NEURO CONNECTIONS Newsletter

Spring 2010

A joint newsletter from the ISNR
& the aapb Neurofeedback Division

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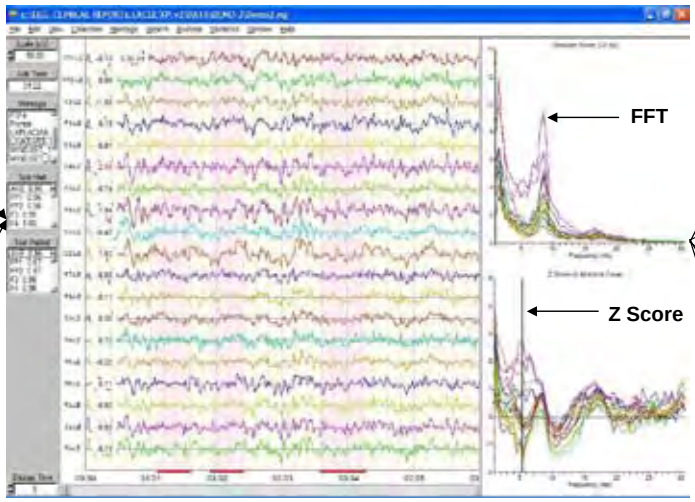

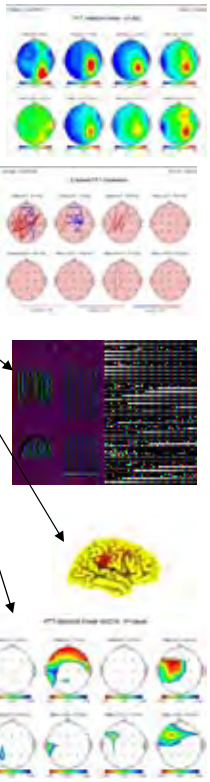

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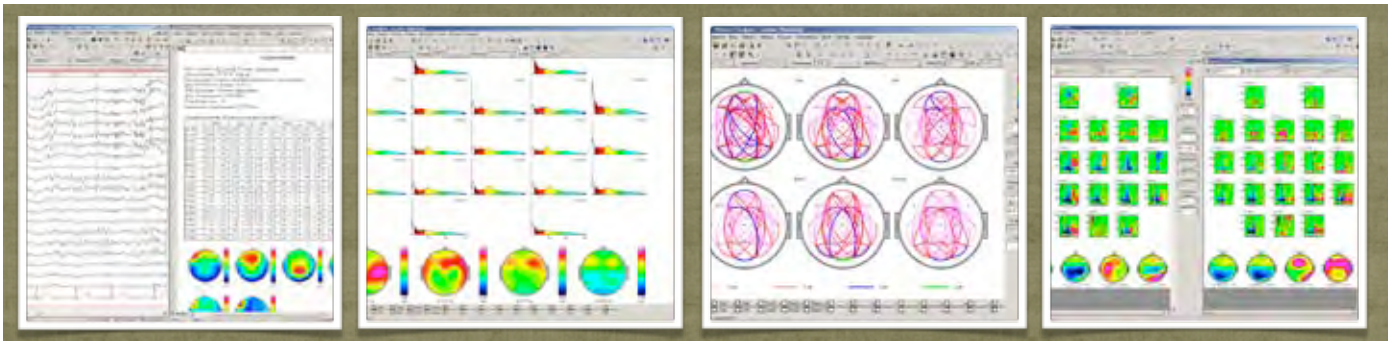

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Letter from ISNR President

Tom Collura, PhD

The Tasks Ahead

While we continue to see progress in the development and acceptance of neurofeedback therapy, this year sees us facing continued challenges, which will define the road ahead. The outstanding issues involve pursuing further acceptance of our work and publications, on a variety of fronts. We enter this decade as neurofeedback remains regarded as experimental, and as insurance companies and other carriers provide minimal or no reimbursement for neurofeedback treatment in many cases. Despite the emergence of more and more books on brain plasticity, brains changing themselves, and related topics, neurofeedback remains notably absent from the thoughts and words of many prominent authors.

In the aftermath of our recent visit and discussions at the CHADD meeting in Cleveland, we were informed that their position remains that neurofeedback is an experimental procedure. They have asked ISNR to provide additional information, which is being prepared, to further answer questions that they have On the positive side, their position on stimulant medication has become harsher, pointing out the limitations, side effects, and lack of lasting benefits resulting from studies including the MTA project. Nonetheless, after reviewing the existing studies, CHADD was unable to conclude that neurofeedback has been shown effective. It is the ISNR

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Letter from AAPB President

Neurocosmology and the Law

David Kaiser, PhD

We have been analyzing human EEG activity in every decade, the 20s, 30s, 40s, 50s, 60s, 70s, 80s, 90s, 00s, and now the teens, and we've accumulated 120,000 peer-reviewed papers on our interesting child, the EEG signal (cf. PubMed; Brazier, 1950). We are approaching an entire century of study, which is a long time to care about anything, even if it is of our own creation, the energy signatures we leave in the air.

The EEG signal was useful scientifically and clinically since its inception. With it we stage sleep, identify seizure, and catch the tails of our mental operations. One of the first experiments with the new technology was feedback: Adrian tried to match his inner experience with the sounds he was hearing from the EEG translation unit he and Berger created (Adrian & Matthews, 1934). Studying and playing with the volitional-physiological correspondence has progressed ever since, through human and animal trials, and under a variety of names like neurotherapy, EEG biofeedback, brain-computer interface, and brain training.

As of this morning, PubMed indexes 5,565 papers for neurofeedback, which is an order of magnitude increase since the last time I searched, in the aughts last December, when it was a sparse 400. (PubMed widened its standard search to include biofeedback as part of the neurofeedback key-

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Letter from ISNR Co-Editor

Merlyn Hurd, PhD

Hello and welcome to the Spring Edition of NeuroConnections.

You may remember that I forecast in my letter in the winter edition that this edition would be about Epilepsy and as you can see, it clearly is about Clinical Discussions. At this point, the Summer Edition will focus on Epilepsy and will have some really strong and pithy essays from well known researchers and clinicians.

On to the present Spring Edition.

Dr. Ochs has a most informative article about the issue of suppression and the clinical implications. He uses epilepsy clients' cases to illustrate his points. We have all grown up with the idea of suppressing amplitudes of certain frequencies. Dr. Ochs' viewpoints are worth your appraisal and will give you much to think about in your research and practice. Dr. Othmer sets forth the issue of Low Frequency training and how this affects the clients' health and functions. If you have wondered about the effect of Low Frequency training, here is the article to help you delve into this line of neurofeedback. Dr. Ryan brings us a personal experience that illustrates how we can sometimes think the client is experiencing anxiety when it may be something more profound and dangerous. Do read to become acquainted with this disorder, especially, if you work with older people. Dr. Abbot gives us an experience that has some of the brain teasing we all experience when we come up against neurofeedback data that looks odd.

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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.

Letter from AAPB Co-Editor

Roger Riss, PhD

Last year, 126 years after his death, the Church of England published an official apology to naturalist Charles Darwin for their initial denunciation of his theory of evolution. *"Charles Darwin, 200 years from your birth (in 1809), the Church of England owes you an apology for misunderstanding you and, by getting our first reaction wrong, encouraging others to misunderstand you still,"* wrote Reverend Dr Malcolm Brown, director of public affairs of the Archbishops' Council. *"When a big new idea emerges that changes the way people look at the world, it's easy to feel that every old idea, every certainty, is under attack and then to do battle against the new insights."*

Controversies surrounding 1859 publication of Darwin's *On the Origin of Species by Means of Natural Selection* came to a head during an 1860 meeting of the British Association for the Advancement of Science. Samuel Wilberforce, Lord Bishop of Oxford, a member of the House of Lords and a Fellow of the Royal Society, was an outspoken defender of the old order. During the debate, he famously ridiculed Darwin's new paradigm with a pointed query: Was it through his grandmother or his grandfather that Darwin considered himself descended from a monkey?

As scientist-practitioners within an emerging, and still controversial, health care field, perhaps we can be forgiven, if at times we suspect that the intransigence of our critics (like Darwin's), while cloaked in the language of science, at times appear to reflect little more than the predictable resistance faced by any idea big enough to challenge the entrenched interests of older world view.

Perhaps we can take some comfort in the final chapter of Darwin's story.

Upon his death some twenty-five years later, Darwin's family arranged for him to be buried in a local churchyard near the family village of Downe. However, William Spottiswoode, then President of the Royal Society, had greater aspirations for Darwin. He contacted the Dean of Westminster Abbey to request that Darwin be accorded the honor of burial in its cemetery. In recognition of the wide acceptance his ideas had gained, even among former critics, the Dean of Westminster granted Spottiswoode's request. Following services at the prestigious Abbey, Darwin was laid to rest close to two other famous scientists, Sir John Herschel and Sir Isaac Newton.

As in Darwin's era, it is increasingly clear that time and the progress of science is on our side. Elsewhere in this issue, David Kaiser points out that a Medline search of the term *neurofeedback* currently yields a heartening 5500 peer-reviewed scientific studies. While there is no evidence that the continued growth in scholarly enquiry into neurofeedback and related disciplines such as brain computer interface is likely to slow, much work remains.

In a recent meta-analysis, Arns and colleagues reported that evidence emerging from a recent series of randomized controlled studies incorporating credible sham active control conditions is of sufficient merit to conclude that neurofeedback treatment for ADHD can now be considered "Efficacious and Specific" (Level 5) with a large ES for inattention and impulsivity and a medium ES for hyperactivity. This body of work now serves as a model for future efficacy research related to other neurofeedback-responsive conditions. As we enter a new decade, ISNR and AAPB plan to continue to play an integral role in supporting and encouraging that work.

Roger Riss, PhD
AAPB Co-Editor

Letter from ISNR ED

Cynthia Kerson, PhD

This spring issue started out as one about epilepsy and seizure disorders. Due to many factors, it evolved into one that looks at many different clinical indications. I've left those details for Merlyn to further discuss in her letter. However, this occurrence brings to mind just how important it is for an Executive Director to "go with the flow," which is often not easy. I constantly juggle vendors' needs, organizational problems, issues with NeuroConnections, issues with the Journal, legal and financial concerns and the special projects that require particular care. In Tom's letter from the President, you're filled in about the special projects —the IEEE, the CHADD dialog, the ongoing struggle to Medline-index the Journal, academic acceptance, etc.

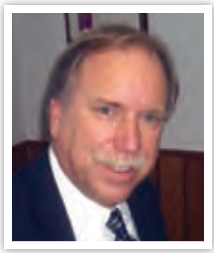
Lastly and certainly not least of the challenges of a member organization is those of our members. Thus, I wish to take a moment to thank our membership coordinator, Ann Marie Horvat. Ann Marie has a singular ability to deal with the unique matters of each member of the association on a very personal level. Her venerable interest in resolving each individual issue is often beyond the call of duty. Admittedly, I regularly admonish her for coddling. That said, our membership is growing. In these economic times, that's a real luxury to say. I can easily and proudly say that a big part of this is due to Ann Marie's efforts. I'm sure I speak for everyone who has any official capacity in the organization that this is true. We cannot thank her enough for overfilling her position.

The ISNR Research Foundation efforts continue. We've moved forward with the TBI and EEG/fMRI projects. Find more information about these two projects on the Foundation's Web page at www.isnr.org/ResearchFoundation.cfm. I would like to thank Dr. Boba Stokic at the Methodist Rehabilitation Hospital for taking neurofeedback and ISNR on as a viable research modality and entity in the treatment for their TBI population and to Dr. David Hubbard for offering complimentary fMRI scan time at the Applied fMRI Institute in San Diego to any ISNR member who can provide a project that facilitates simultaneous EEG/fMRI recordings that furthers the mission to assimilate the modalities. BrainMaster Technologies, Inc, Mitsar and NovaTech EEG have donated equipment and services to these projects. The Foundation, working on very limited funding, is very grateful to all of these entities for their support. Care to participate? Please contact me at office@isnr.org.

Cynthia Kerson, PhD, BCIA-EEG
Executive Director, ISNR

Letter from AAPB ED

Members—The Life Blood of AAPB



David L. Stumph

For years, AAPB has developed and nurtured a core set of services that are valued by its members. Such services include two outstanding publications: a scientific journal, *Applied Psychophysiology and Biofeedback*, and a newsmagazine/clinical journal, *Biofeedback*, a highly regarded annual conference, its teleseminar series, Clinician's Tool Kit, and a host of other important member services.

As with any "business," it is important for AAPB to evaluate its members' needs and to fully understand how interests change from time to time. In our rapidly changing world, it should be no surprise that member interests change quickly as well. In the world of AAPB, members are faced with new and exciting equipment options every day. How we communicate with our members and with each other is also changing rapidly. As younger practitioners enter the field, what mechanisms will best serve their needs educationally, how will we reach and engage them, and how will we take advantage of new media opportunities that are presented by the evolving online environment?

To validate the importance of its current services and to evaluate new potential activities, products, and how we conduct the business of the organization, AAPB is embarking on an extensive project to survey its member's needs and interests. The membership committee is putting the final touches on the survey and we expect that it will have been launched before this article is published. The survey will evaluate all aspects of the association from its customer service to the value of its publications, from the annual conference to its website, and from overall communications to future projects and services.

You do not need to be a member to participate. In fact we hope to hear from a wide variety of individuals including members and non-members so that we receive a wide variety of feedback. You can access the survey from the AAPB website at www.aapb.org.

The new decade brings new opportunities along with new challenges. Making sure that we understand our members and what is important to them is paramount. Understanding the impressions that others have of our organization is important as well. We welcome everyone's participation and look forward to an outcome that will provide us with the information and tools to set a new and revitalized course for AAPB.

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

AAPB President

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word). PubMed indexes mostly wet-science journals -- biomedical, psychological, and biomedical engineering, with a few physics journals thrown in, and has 19 million papers indexed since 1950, 10 million from the last two decades. Google Scholar goes back only two decades instead of six like PubMed, but it includes both wet- and dry-science journals including the range of physics, mathematics, and information sciences. Google has 5,340 articles with *neurofeedback* mentioned somewhere in the article and an amazing 69,000 articles for *biofeedback*. PubMed does have some dry-science journals in its database; for instance, a search of *supersymmetry* provides a respectable 370 listings but the same term in Google Scholar provides 164,000. In physics *supersymmetry* refers to elementary particles and basic forces, but we can use the same term as well and apply it to the elementary particles of our world of mental health and healing. We want to create a supersymmetry between mind and body, a transparency between volition and physiology, a balance of heart and head. The Neurofeedback Division of AAPB are those members of the neuroscientific care community who are doing just that, working at the cutting edge of biovolitional supersymmetry and it's an amazing field to be part of.

David Kaiser, PhD

References

Adrian ED & Mathews BHC (1934). The Berger Rhythm: Potential changes from the Occipital Lobes in Man", Brain, 57, 355-385.

Brazier MAB (1950). Bibliography of electroencephalography 1875-1948. Montreal International Federation of Electroencephalography and Clinical Neurophysiology, 1950.

ISNR President

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board's opinion that while medication may be an effective short-term solution, neurofeedback should be paramount among the long-term treatment modalities that should be pursued to secure lasting benefits, and to produce lifelong freedom from the symptoms of attention related disorders.

It is curious that we are being held to very high standards, particularly the requirement for placebo-controlled, double-blind, randomized trials. There is division within our ranks as to whether such studies are necessary or even possible, which, along with the cost of such trials, remains a substantial impediment. Curiously, the CHADD publication ADDitutes, recently endorsed an arguably neurofeedback-like intervention (LENS), based on outcome studies, plus the positive experiences of one of the editors with this system. This endorsement was published without citing studies of the type that the CHADD scientific advisory board indicated were necessary, to the ISNR board. This points to the fact that much of our challenge is political and practical, not scientific or academic.

In addition to acceptance by organizations, insurance carriers, and large health care entities, we need to see our publications recognized by the scholarly communities. To this end, we have sought to have the Journal of Neurotherapy accepted by PubMED and MEDline. Having this acceptance and listing will enable a broad range of investigators to find our output, by conducting standard searches in the library systems and the internet. In the past, we have met barriers to this acceptance, due to limitations in the type, quantity, and quality of material. The board has recently instituted a revision in the JN editorial board membership and charter, putting acceptance by the medical indexing services on the front burner. We look to Martijn Arns and Randy Lyle, as co-editors, along with an expanded slate of reviewers, to stimulate high quality submissions that will lead to further acceptance of JN in the future. We wish to thank David Kaiser for his past contributions, and look forward to his continued participation in ISNR projects.

I would also like to encourage all ISNR members to consider submitting material for the journal. A range of topic areas is open for submissions. In particular, results of clinical trials or case studies, theoretical or concept papers, and scientific and technical papers are being requested. Please contact one of the JN editors or a board

member if you believe you have material that could be submitted for publication.

Growth is also a priority for ISNR. Recent efforts in international membership programs have produced a significant increase in membership, and more global visibility for ISNR. Another initiative is the further incorporation of the academic community into our ranks. We are undertaking specific efforts to introduce academic memberships in ISNR, and to stimulate faculty as well as students to contribute to our meetings and publications.

On the technical front, ISNR is pursuing, along with the Institute of Electrical and Electronics Engineers (IEEE) and leading neurofeedback systems developers, a standards effort for neurofeedback equipment. The initial standards effort has begun, and defines the scope of the new standard to ensure quality and availability of the data and feedback provided. This is intended to ensure that neurofeedback systems operators and trainees are provided with devices, documentation, and training that provide clear and useful results. While this effort is not intended to dictate any particular philosophy or approach to neurofeedback, it should help to ensure that terminology and technical provisions are uniform and comprehensible, thus providing consistency and quality in the field.

Overall, neurofeedback, which has been "out of the box" for the past couple of decades, needs to find its way into one or more boxes, be they new or old boxes, in order to see increased acceptance and reimbursement. Our goal remains the same, that neurofeedback as a field becomes a vital, well-supported, profitable, and beneficial endeavor in the mental health care community. Most of us have seen neurofeedback in action, and have no doubts about its value. The challenge is to convey this understanding to the uninformed, and to convince the unconvinced. This is a task that requires action on many fronts, including scientific, clinical, political, economic, and educational. To replay one of my "broken records," it is by working together in a cooperative and supportive manner that we will achieve these goals. We need to listen to each other, strive to understand and appreciate differing viewpoints, and respect diverse contributions.

One year is a short time. My tenure as ISNR president has hardly gotten into full swing, and the transition to a new president is already in sight. I want to thank past President John Nash for his contributions, and in particular for his leadership in professional and technical standards efforts. I look forward to president-elect Leslie Sherlin taking the helm later this year. It is one of my goals that we see continuity as well as growth during these transitions.

As is true of any professional organization, we are only as strong as our membership and our members' contributions. We continue to look to members for ideas, contributions, and efforts to build our field and its acceptance. There is no time like the present to ask yourself what you can do for the field, and how your participation in ISNR can be part of your plans. Ask yourself what you can do, what you can write, what you can publish, who you can call, or how you can use your time and talents to benefit the field of neurotherapy, and thus help those who can benefit from its capabilities.

Tom Collura, PhD
ISNR President

ISNR Co-Editor

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The issue of scar tissue and the implications of the effect in using neurofeedback on the site is interestingly presented. My Physical Therapist told me to think of scar tissue as molasses, so when one is trying to break it up it evolves into different forms and thus causes pain in different regions. Obviously, this could influence neurofeedback data.

Dr. Donaldson provides an in depth case of treating Schizophrenia with neurofeedback. The case is presented by the client with clinical observations by Don and his wife Mary, both of whom provided the analysis and treatment of the client. This is a fascinating journey and gives hope to all of us to look at providing treatment for a population that has mainly been treated with drugs and hospitalization. I can remember over the years, the admonition that we, in the biofeedback world, never treated schizophrenia. This article will certainly cause one to take the "never" out of that admonition.

Hope you enjoy this edition. Also if you wish to give copies to the clients, we sometimes have a few in the office that we could send you. Otherwise, have the client or yourself go to ISNR.org and click on NeuroConnections. All the articles for the last 3 years are available. As a member, you have access to the 2 latest editions through the member log-in portal. After six months the edition is available to all who visit the website. This service gives you the opportunity to download and make available, in your waiting room, in your communications and even as handouts, articles relevant to the case(s) you are treating.

Hope you enjoy and gather some tidbits of information from this Clinical Discussions Edition.

Warmly,

Merlyn Hurd, PhD, BCIAC/EEG Fellow
ISNR Co Editor

Not Your Ordinary Panic Attack!

Patricia Jo Ryan, PhD

Last year, I walked into my bedroom to go to bed and found my husband shaking violently, barely able to speak, complaining that he was freezing to death, showing severe difficulty with breathing, and hyperventilating in a way that he could not stop. His teeth were chattering and he was shaking so hard it was frightening to see. He was unable to alert me about what was happening to him because he could hardly speak. It was quicker to bundle him up and get to the local ER about 6 minutes away, so that is what we did. By the time we got there, his shaking had stopped and he was appeared to have weathered the storm. Many tests were taken - MRI's, CT scans, blood work and they revealed nothing, so he was sent home with an appointment to see his primary physician. This might appear to be a panic attack as evidenced by more than four of the symptoms in the DSM-IV.

However, a few months ago, this same kind of incident occurred again with more intense expression of the same symptoms, and he called 911 (I was not home at the time). He reported that the EMT technician could not find his blood pressure, his pulse was weak, and the EMT team had difficulty establishing current heart rate functioning. His pulse had slowed, and blood pressure was very low, but these functions began to normalize during the time he was in the ER. Again, he was sent to his physician for follow-up. This time I wrote a letter to his doctor with my thoughts and concerns. She dismissed it by saying she had to look for a "physical cause." She called for multiple blood tests that yielded no results. These personal events led me to revisiting the concept of panic attacks and their origin.

Panic disorder, in the DSM-IV, is divided into three categories; unexpected (uncued); situationally bound (cued), or situationally disposed. The events that I witnessed, and later, through self report, appeared to be sufficient to explain this phenomenon. After the second episode, I began to suspect that what might be occurring was a phenomenon called Parasympathetic Rebound which is described as an "unpleasant physiological reaction of the body" in a book I was reviewing on Autogenic Training (Sadigh, 2001). In the second episode, the symptoms were described as much more intense though he did not go to the hospital that time. His voice became almost inaudible and hoarse and stayed that way for the next week. The description in the DSM-IV suggested that the intensity of the symptoms diminish with recurrent attacks. This was not the case here; the physiological manifestations intensified and the last episode was reportedly the longest (55 minutes) before returning to a normal state.

I then attempted an internet search on Parasympathetic Rebound. Very few citations were available and the descriptions were overlapping and scant. However, the symptoms observed and reported, suggested a serious over-reaction of parasympathetic nervous system (or overcorrection) in returning the body to homeostasis and could, in fact, cause death. Parasympathetic Rebound apparently can follow prolonged and sustained sympathetic nervous system activity. My husband had been suffering from a severe bout of sciatica for several months prior to this last episode, refused to take pain medication due to its side effects and endured the pain. At that time my suggestion for chronic pain intervention by a local psychological specialist went unheeded.

After these events and looking for more extensive information about this activity, I called and e-mailed some of my colleagues to inquire if they have ever heard of this phenomenon. One colleague, Dr. Ed Hamlin, a neuropsychologist in North Carolina, did, in fact, know about this phenomenon and identified it as a dangerous condition as well. Another colleague described experiencing similar symptoms during an Alpha-Theta session she was having and had to stop her session. At that time, she didn't know what her symptoms meant. In the past, I have had reports from clients of some unpleasant reactions to Alpha-Theta training and/or SMR training with suspected over-aroused sympathetic activity. They described the symptom as "flu-like," but there were no reports of the extreme manifestations of the physiological symptoms I observed in the first episode.

One definition of the activity of the parasympathetic nervous system included narrowing of air passages, constricting the pupils, decreasing heart rate, and lowering the blood pressure in the body, to name a few. All of these activities are the very ones that seemed to have occurred. One of the major components of panic attack is that it can occur without an obvious precipitator. While the physiological symptoms are part of the criteria of panic episodes, it appears to me that the severity of this phenomenon is not just your ordinary panic attack. My hypothesis is that ***the automatic rebound or over-correction of the parasympathetic nervous system precipitates the panic episode!*** My other hypothesis is that medication changes this dynamic in the body and may preclude activation of an effect.

These events have given me a much healthier respect for the automatic activity of the autonomic nervous system of the body. I am certainly being more careful about how I treat clients that have long-term excessive autonomic arousal or arousal levels that are not always registered in SMR training. Luckily, it served as a wake -up call to my husband and he is now practicing autogenic training, seeing a colleague, and attending NFB sessions. An interesting piece of information gleaned from Sadigh's book describes Autogenic Training as engaging the state between "pre-drowsy and drowsy" on the wake-sleep continuum. We attempt to access same state through Alpha -Theta training using a different delivery system. This juxtaposition of concepts from different sources may explain some of the puzzling reactions some clients have reported on occasion since adding NFB to my practice. I now pay a lot more attention to a history of unremitting, sustained stress on the body. That said, whoever stated that panic will not kill you may be wrong (it may not be just an ordinary panic attack)!

Sadigh, M, (2001) Autogenic Training - A Mind-Body Approach to the Treatment of Fibromyalgia and Chronic Pain Syndrome. The Hayworth Medical Press, New York, London, Oxford

A Single Case Report of TBI and Depression

Kathy Abbott, PsyD

Mr. Client came for neurofeedback to address symptoms of depression, anxiety, insomnia, and an attention deficit. While he had previously had success at work in sales, at this time he was having significant difficulty getting and keeping jobs. It was apparent that he was very gifted in his ability to socialize but was also very angry which showed in his being critical of others. He expressed loneliness and tried to be friends by pushing his needs onto others. He was using a golden rule literally – if I need this, I think you must need this. He was unable to accept "no" to his offers of help without feeling offended and he would respond with indirect anger sometimes in the form of criticism.

The initial protocols were those typically used for depression and an attention deficit: enhance 15-18 Hz, inhibit 4-7 Hz and 20-30 Hz at F3; enhance 12-15 Hz, inhibit 4-7 Hz and 20-30 at Cz. He reported some limited improvement with these protocols. However, the success was frequently short lived. He was encouraged to have a qEEG (quantitative EEG) but refused for quite some time. Eventually he agreed to have the qEEG which was helpful in establishing more effective protocols.

The qEEG (see Figures1a and 1b) revealed an excess of low beta central/parietal which is associated with anxiety. (Gurnee, 2000; Arns, (personal communication, *SNR Members Forum*, January 24, 2008; Coger et al, 1999; Brownback et al, 2009). I had previously seen a significant scar on another client that was in the region where I was training with successful decrease in symptoms. Mr. Client had some scar tissue just outside of and above his left eye. The qEEG showed a slight excess of theta at F7 but the qEEG finding was around 1-1/2 to 2 inches from his scar which was not in the area where the EEG was collected. Dr. Cory Hammond had reported using F9 and F10 as a way of getting to the orbitofrontal cortex (personal communication, January 6, 2010). This area is close to the one found on

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TBI and Depression
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Mr. Client. Drevets (2009) indicates that the orbitofrontal cortex is associated with mood. There was some low coherence (see Figure 2) in all frequencies but more pronounced in the lower frequencies. The low coherence was emanating from T3 to Fp1, F3, F7 and C3 which would be consistent with a head injury but not diagnostic.

We decided to train here. The response was immediate. Mr. Client was able to train down his theta quickly in his first session and successive sessions. His mood improved. He became more insightful and realized on his own that he had been pushing his needs onto others. Previously Mr. Client was sometimes enjoyable and other times negative and critical. Following treatment, his attitude was pleasant nearly all of the time. His motivation increased and he was able to start and complete projects. Although he remains affected by weather and sunlight conditions, he is able to accomplish work on sunny days. Some of the symptom improvement such as lower anxiety and improved sleep are probably due to training down low beta mentioned previously. But the improvement in motivation, attention and mood appear to be due to training over the scar in the left frontal area. Further, he had been trying to lose weight for a year and following a few sessions, he lost 30 pounds. Additionally, at one-year post treatment Mr. Client has not only maintained the weight loss but lost another 20 pounds.

Discussion of Head Injuries in General and Scars.

While scars are not a definite indicator of a traumatic head injury (TBI) they may indicate the presence of one. Additionally, TBIs are frequently under-diagnosed. They do not show up on many medical scans (Hoffman et al). However, there are findings associated with head injuries that show up on a qEEG. Mr. Client did not have a memory of a head injury in this region but reported extreme emotional abuse as a child. Another problem with head injuries is that the symptoms may not show up for years, or there may be symptoms initially but the brain may organize itself around the injury so the person can function better. This may explain why Mr. Client was able to do well on the job earlier in his life and not later.

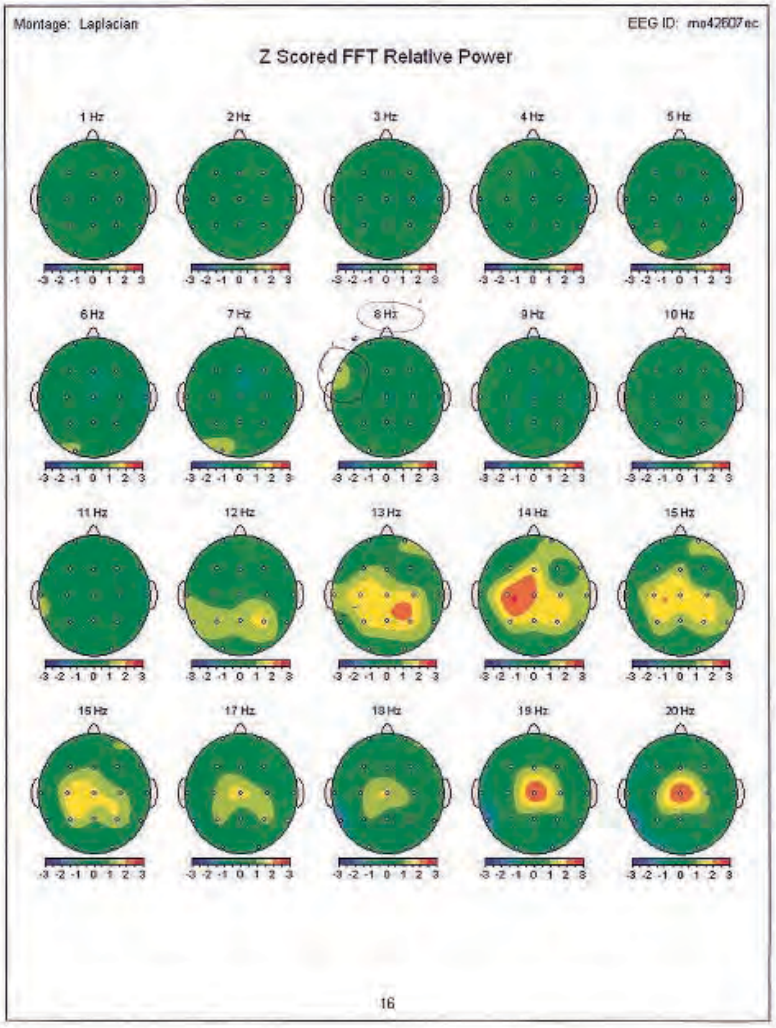

Figure 1a. Z Scored FFT Relative Power: 1 Hz to 20 Hz.

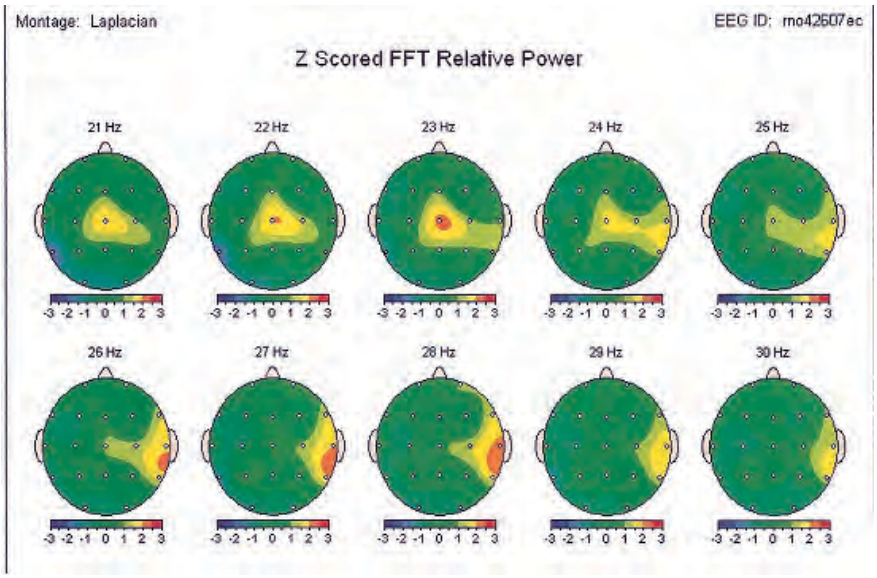

Figure 1b Z Scored FFT Relative Power: 20 Hz to 30 Hz.

Some symptoms of head trauma include the following:

- Difficulty concentrating
- Loss of memory, vision, hearing, or movement
- Seizures or short period of "absence"
- Increased irritability and/or difficulty with anger management
- Depression and anxiety

Neurofeedback can be used to decrease the following symptoms associated with MTBI:

- Vision (Nash, 1997)
- Headaches (Nash, 1997; Packard et al, 1997)
- Energy level (Ayers, 1987) and sleep (Salerno, 1997,)
- Cognitive functioning (Hamilton, 1997; Packard et al, 1997)
- Attention (Salerno, 1997)
- Emotional functioning (Salerno, 1997)

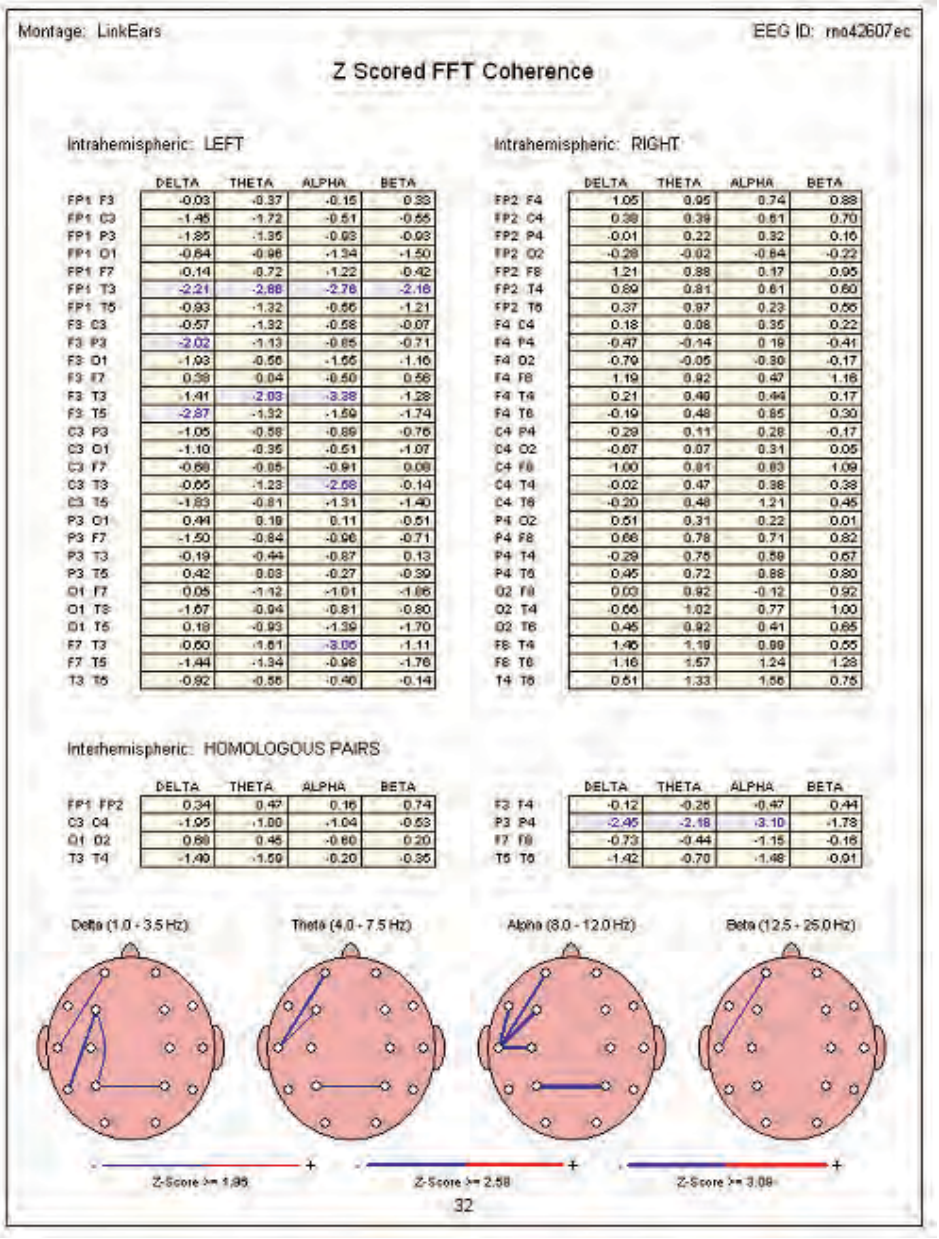

Figure 2. Z Scored FFT Coherence.

A more than a fifty percent improvement was found in 88% of TBI cases reported by Walker, et al (1997). The percent of cases reporting improvement in the following symptoms were: vision (20%), headaches (84%), energy level (12%), sleep (12%), memory (72%), attention (44%), depression (44%), anxiety (16%), dizziness (12%), and confusion (8%).

Ayers (1987) working with 250 people with a head injury. She found a decrease in depression and temper outbursts and an increase in energy in the first six sessions. After 12 sessions, the clients reported a decrease in light and sound sensitivity, and better ability to focus. In the next 6 sessions, the clients reported a decrease in dizziness and headaches. With the following 6 sessions, there was a report of increased libido, and a decrease in reversal of letters and words. Of the 250 clients, short term memory was reported to return in 150.

Mr. Client responded to qEEG-guided neurofeedback, plus work on the area on his scalp with scar tissue consistent with the above reported symptoms and response to treatment. The case is a clear indicator that the qEEG does not cover all locations that may need training.

Dr. Kathy Abbott is a Licensed Clinical Psychologist *with 20 years of experience working with individuals (children, adolescents, and adults) and couples. She has specialized training and experience working with trauma and addiction.*

Dr. Abbott has been doing neurofeedback since 2003. Her focus is on learning issues, traumatic brain injury, PTSD and anxiety, and Asperger's disorder.

Dr. Abbott earned her Doctorate in Clinical Psychology from the Adler School of Professional Psychology. She is a member of the International Society for Neurofeedback and Research, and the American Association of Psychophysiology and Biofeedback. Dr. Abbott is board-certified in EEG Biofeedback by the Biofeedback Certification Institute of America.

Prior to entering independent practice, Dr. Abbott worked in outpatient and hospital settings. She helped start the EEG biofeedback program for addicted health professionals at Rush Behavioral Health. Dr. Abbott is a prime organizer of the Neurofeedback Centers of Greater Chicago (EEGChicago.com), a group of professionals educating the public about neurofeedback. Dr. Abbott works in Evergreen Park and Oak Park.

Continued on page 13

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TBI and Depression
continued from page 9

References

Ayers, M. (1987). Electroencephalographic neurofeedback and closed head injury of 250 individuals. *National Head Injury Syllabus*. Washington, DC: Head Injury Foundation, 380-392.

Brownback, T. S., Mason, L. et al (2009). *The Brownback Mason & Associates Neurofeedback System (BMANS) Manual 2: Neuropathologies.*

Coger, R. W., Dymond, A. M., Serafetinides, E. A. (1999). Electroencephalographic similarities between chronic alcoholics and chronic, nonparanoid schizophrenics. *J. Neuropsychiatry Clin Neurosci, 11, 190-208.*

Drevets, W. C. (2009). (Abstract) Orbitofrontal Cortex Function and Structure in Depression, *Annals of the New York Academy of Sciences, (1121),* 499-527.

Gurnee, R. L. (2000). EEG based subtypes of anxiety (GAD) and treatment implications. http://www.snr-jnt.org/topics/abstract.htm

Hamilton, P. M. (1997). The effects of EEG biofeedback on vigilance in adults following mild traumatic brain injury, *Dissertations Abstracts International, 57, (9-B)*, 5969.

Hoffman, D. Steven Stockdale, S., Hicks, L. L., Schwaninger, J. E. (1995). . Diagnosis and Treatment of Head Injury. *Journal of Neurotherapy, 1*,(1).

Nash, J. K. (1997). *Binocular vision and mild traumatic brain injury.* Poster Presentation at the Annual Meeting of the International Society of Neuronal Regulation.

Packard, R. C & Ham, L. P. (1997). EEG biofeedback for post-traumatic headache and cognitive dysfunction: A pilot study. *Headache quarterly, 8*, 348-352.

Salerno, J. A. (1997). Neurofeedback in closed head injury: A multiple case design study. *Dissertation Abstracts International: Section B: The Sciences and Engineering. May*, vol 57, (11-B) 7266.

Walker, J, Norman, C. A., & Weber, R. K. (2002). Impact of qEEG-guided coherence training for people with a mild closed head injury. *Journal of Neurotherapy 6,* 31-43.

Additional Reading:

Hughes, J. R. & John E. R. (1999). Conventional and quantitative electroencephalography in psychiatry, *Neuropsychiatry Clin Neurosci, 11,* 190-208.

Schoenberger, N. E., Shiflett, S. C., Esty, M. L., Ochs, L., Matheis, R. J. (2001). Flexyx neurotherapy system in the treatment of traumatic brain injury: An initial evaluation. *Journal of Head Trauma Rehabilitation, 16,* 260-274.

Tinius, T. P. & Tinius, K. S. (2000). Changes after EEG biofeedback and cognitive retraining in adults with mild traumatic brain injury and attention deficit hyperactivity disorder. *Journal of Neurotherapy, 4,* 27-44.

Conferences and Calls for Papers

Association for Applied Psychophysiology and Biofeedback 41st Annual Meeting

March 24-27, 2010, San Diego, California.
http://www.aapb.org/

Biofeedback Foundation of Europe, 14th Annual Meeting

April 13-17, Rome, Italy.
http://www.bfe.org/

American Academy of Clinical Neuropsychology, 8th Annual Conference & Workshops

June 17 - 19, 2010, Renaissance Chicago Hotel, Chicago, Illinois.
http://www.theaacn.org

International Neuropsychological Society 2010 Mid Year Meeting

June 30th-July 3rd . Krakow Poland.
http://www.the-ins.pl/

Applied Neuroscience Society of Australasia, 2010 Annual Conference and Workshops

July 12- 16, 2010, Mantra at Salt Beach, South Kingscliff New Tweed Coast, Australia
http://www.ansa.au.com/

International Society for Neurofeedback and Research, 18th Annual Conference

September 29-October 3, 2010, Omni Interlocken Resort, near Denver, Colorado. Preconference Workshops Sept 27-29. Submission deadline April 16, 2010.
http://www.isnr.org

National Academy of Neuropsychology 2010 Conference

October 13 - 16: The Westin Bayshore, Vancouver, BC.
http://nanonline.org/

American Academy of Child and Adolescent Psychiatry (AACAP)

October 26-31, 2010, New York City
http://www.aacap.org

Introduction to Infra-low Frequency Training

Siegfried Othmer, PhD and Susan F. Othmer, BA

The first convincing evidence for EEG feedback efficacy in the management of pathophysiology was with regard to generalized seizures. The early work by Sterman, Lubar, as well as the subsequent follow-up by others, therefore remains a crucial point of reference for the various feedback techniques that have built upon the early protocol of SMR reinforcement combined the theta-band and high-beta band inhibition. Remarkably, the essential features of the early approach have been retained in the various evolutionary pathways that have emanated from the early work. This essential similarity has perhaps obscured other aspects of the training approach that have changed substantially over time, the significance of which may not have been fully appreciated except in reflection. In this newsletter we consider some of these changes and their implications generally, as well as for seizure management in particular.

The common thread in most modern neurofeedback approaches is the combination of a reinforcement strategy on one EEG frequency or another and an inhibit strategy based on detection of excursions into dysregulation. Some issues relating to the inhibit side have been relegated entirely to the software, thus removing them from ready visibility. Artifact detection and the division of labor between that and the conventional inhibits is a case in point. Specific targeting strategies typically remain to the discretion of the practitioner, as for example with respect to thresholding, placement, and frequency band selection. The general thrust over time has been to broaden the "field of view" of this EEG-based disregulation detector, mainly with respect to the frequencies being targeted, but sometimes also in terms of placement. Multi-channel instruments allow independent choice of placement for the reward and the inhibit strategies. Fortuitously, the various inhibit strategies being actively used—though differing significantly from each other—have not been wrapped up in much controversy.

Far more discretion prevails with regard to the reward strategy, and unsurprisingly most of the proliferation of alternatives, and hence most of the unresolved issues in the field, relate to that aspect of the protocol. In the early work, the discrete detection of an SMR bursting response in cats, combined with discrete rewards, firmly established that operant conditioning of the EEG could be routinely achieved. In the subsequent training of human subjects, the dollop of food reward was replaced by a mere beep tone. More significantly, the waking human EEG did not typically exhibit the distinct SMR bursting response that could be seen in cats. The distribution of SMR amplitudes was instead well-behaved. With a continuous, quasi-Gaussian amplitude distribution, the choice of threshold was left somewhat arbitrary, whereas in cats the threshold level had been largely dictated by the double-humped distribution.

The common thread in most modern neurofeedback approaches is the combination of a reinforcement strategy on one EEG frequency or another and an inhibit strategy based on detection of excursions into dysregulation.

In the clinical setting, as distinct from the research setting, a second feedback loop is always operative, namely on the clinician monitoring the feedback process of the client. As it happens, the behavior of early clinicians was gradually shaped to offer rewards more generously than had been the case for the underlying research. This was done through easing the reward threshold. By way of contrast, both Sterman and Lubar had maintained high thresholds in order to discriminate the rare, large excursions in SMR burst amplitude. The reward incidence was correspondingly low, by analogy to what had transpired in the cats. With the emergence of more generous thresholding, the discrete reward (beep tone) eventually became so plentiful that hearing it became an expectation. The dropout of the tone became the 'odd-ball' event to which attention would be preferentially devoted, the reverse of what had originally been intended, and the reverse of what had been done in the original research.

The discrete reward had come to function like an inhibit. On the other hand, in terms of clinical results the reward functioned just as it had before. If anything, the training had become even more efficient with the relaxation of the reward criteria. The mystery was resolved with the realization that the principal source of information to the client lay in the accompanying continuous information flow on the instantaneous reward amplitude. The beeps had been reduced to mere accompaniment for most sighted individuals.

Years later Sterman appealed to the neurofeedback community that in the spirit of learning theory one ought to allow a sufficient refractory period between discrete rewards to permit consolidation, but by the time he forcefully put this forward the die had already been cast, and most practitioners were using instrumentation that implemented the new regime. The emphasis had shifted toward the analog reward signal as the bearer of the principal burden of the feedback. There had to be a heightened information density to explain the quicker responses of trainees, and the rapidly fluctuating analog signal was the only explanation.

It has largely gotten lost in the flow of subsequent developments that both Sterman and Lubar used bipolar montages in all of their early research on human subjects.

In our work we used both bipolar and referential montages, and a general impression emerged that the bipolar montages yielded stronger effects. This could, however, also have been due to placement. One could never be sure. Confirmation of sorts was provided subsequently by the general observation that two-channel coherence training is typically more powerful than referential amplitude training. Both bipolar placement and two-channel coherence training weight the relative phase of activity at the two sites more heavily than referential training, which involves us directly in the issue of connectivity. This follows from the observation that if the reference were entirely neutral, then phase would not enter the picture at all. In between, we are on a continuum.[1]

Additionally, it was observed that clients differed in their response to particular reward frequencies, and that their clinical response could be optimized by small adjustments in the reward frequency. In some instances, small adjustments became large adjustments, and eventually this approach could no longer realistically be referred to as SMR-training. Remarkably, however, the optimal reward frequency (ORF) for a client was largely unrelated to the underlying clinical condition. And just as SMR-training had come to serve a variety of objectives having little or nothing to do with the motor system specifically, the optimized training was if anything even broader in its clinical impact.

At one of our training courses, an attendee who remained to be persuaded was watching a demonstration of the optimization procedure on a volunteer from the class, his critical faculties on high alert. As the trainee reported changes in alertness, in feelings, and in symptom severity after mere minutes, the skeptic exclaimed: "This cannot be operant conditioning!" And he was right. Significant learning cannot have been accomplished on this time scale. All we had really done is to shift the client's state in the moment. Still, that required a lot of information, which only the analog signal was capable of providing. The analogy here is to traditional biofeedback, where attending to analog GSR and temperature signals in first instance simply induces state shifts. The learning of new behavior is then the consequence of numerous repetitions.

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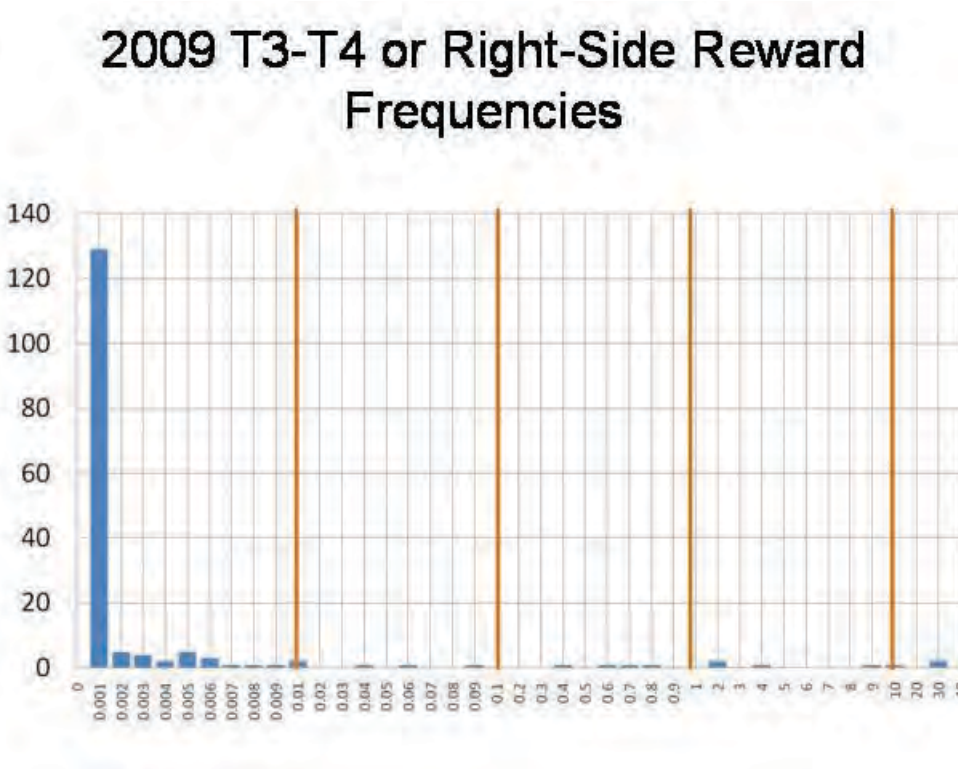

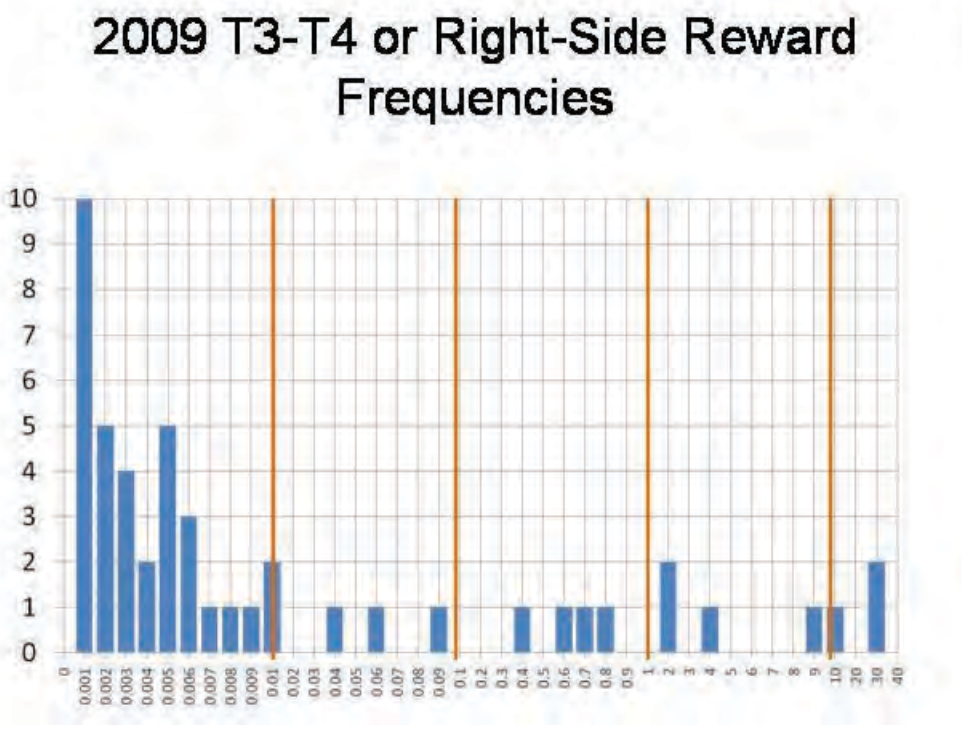

Figure 1 shows the distribution in optimum reward frequencies observed in 167 clients seen in our office during 2009. Figure 1a shows the full distribution; Figure 1b shows the same data on a finer scale. The abscissa scales logarithmically in decades per division, but within each division the scale is linear. The plot is therefore best seen as a concatenation of several linear plots each covering a decade in frequency. The strong dominance of the lowest available reward frequency of 0.001 Hz is apparent. This data point is truncated in Figure 1b.

Infra-low Frequency Training continued from page 15

The Trend toward Infra-low Frequency Training

In the continuing exploration of the optimization procedure with each client, it was observed that we often bumped up against the lower limit of the frequency range that our software provided for. Early on, we allowed for reward-based training down to 4-7 Hz. (Three-Hz bandwidth was standard for the reward band.) Over time it became clear that many clients were bumping up against the lowest frequency, and one had the impression that many needed to train even lower. When the software was extended down to 0-3 Hz, the distribution changed further, and revealed the lowest center frequency, 1.5 Hz, to be the most populated. Again one had the impression that some clients needed an even lower reward frequency. When the software was further extended to allow operation down to 0.1 Hz cutoff frequency, over time the center frequency of 0.05 Hz became the modal value. The same occurred when we extended the range down to 0.01 Hz in center frequency. And finally we extended the range to the 'absurd' value of 0.001 Hz in November 2008. The lower range that had been opened up immediately became populated, and once again the modal value became the lowest we had available, 0.001 Hz.

Looking over the past year of clinical experience in our office, 77% of all clients optimize their response at the lowest frequency (129/167), and 90% optimized below 0.01 Hz (151/167). The distribution is shown in Figure 1. Observe that the abscissa scales logarithmically in decades per division, but within each division the scale is linear. As our current experience is compared with the earlier "trials" with higher cutoff frequencies, an interesting pattern emerges. As we went down in minimum cutoff frequency through all of the stages listed above, the distribution became ever more skewed in favor of the lowest frequency rather than less. This is contrary to what one would expect. One would have expected the equivalent of unrolling a carpet, gradually exposing more of the pattern of nature as the software progressively made this possible. But in fact the whole distribution kept changing as we went. Note in this regard that in the current distribution the earlier peaks at 13.5 Hz (the 12-15 Hz SMR band) and 16.5 Hz (the 15-18Hz low beta region) are no longer discernible, nor are the subsequent peaks of (successively) 5.5 Hz (4-7 Hz band), 1.5 Hz (0-3 Hz band), 0.05 Hz, and 0.01 Hz.

At the top level one must assign the changing distribution to our clinical learning curve. It took us years to become fully conversant with this new frequency domain. But more specifically it also became very clear that the training was stronger at the lower frequencies, and correspondingly more frequency-specific. The more deeply we penetrated into the ILF range, the more precision was called for in the choice of reward frequency. Reflecting back on our earlier history from our current perspective, it is apparent that optimal frequency training was not available for some 90% of our clinical population even as we were finding our way with the optimum reward frequency model. All we were in a position to accomplish along the way was a kind of 'local optimization' that should not be expected to reflect the distribution once global frequency optimization became a possibility.

Over time it became clear that many clients were bumping up against the lowest frequency, and one had the impression that many needed to train even lower.

The Clinical Role of ILF Training

The defining characteristics of the protocol are bipolar placement and optimization of the reward frequency without restriction (combined, of course, with an inhibit strategy). However, since over 90% of all our clients optimize in the ILF range, and since novelty attaches to that aspect specifically, this training approach has come to be referred to as ILF training as commonly as ORF training. The frequency-optimized bipolar training has unambiguously improved our outcomes across the board with our clinical population, and this includes in particular medically refractory seizures, which remain a benchmark for comparison purposes across the decades.

Our challenge is to provide a theoretical framework in which these results may be understood. The bipolar placement can be seen as having a bias toward network desynchronization, which in turn is thought to be intrinsically stabilizing in the case of seizure susceptibility. This could equally have been the operative mechanism in Sterman's and Lubar's classic papers in which bipolar placement was used. On the other hand, the target in the cat work was clearly network synchronization in the SMR band. These disparate approaches, both leading to a common end result, can be reconciled by thinking of certain protocols in terms of setting up challenges as opposed to prescribing destinations. After all, neither network synchronization nor desynchronization represents a desirable steady-state condition.

It is helpful in this regard to draw on the collective wisdom from our sister disciplines of peripheral biofeedback. Even in temperature training, where a clinical objective was unambiguous, clinicians often relied upon alternating up- and down-training. The immediate objective was enhanced control. Peripheral biofeedback also helps us to understand working in the ILF region. Here we are tracking the slow cortical potential through its (differential) migrations on very long timescales. Threshold crossings in the traditional sense are extremely rare at the lowest frequencies. Much can happen between one threshold crossing and the next, so the threshold can no longer be critical to the proceedings. It has become entirely a matter of process, and that's where traditional biofeedback has already been----for example with Heart Rate Variability (HRV) training. Merely by focusing on the instantaneous beat-to-beat interval, the trainee affects the entire HRV spectrum, all the way down to the ILF range.

The journey has become much more important than the destination. Goal setting has become less relevant. The idea of discrete rewards has lost meaning in this context. Rather, the watchword now is engagement with the process. Engagement here refers to the brain rather than to the client, and in practice this means the brain must recognize its agency with respect to the proffered signal. Such recognition is obviously favored by a continuous rather than episodic signal stream.

Now when it comes to promoting engagement, we seem to be better off at the optimized reward frequencies in general, and at the infra-low frequencies in particular. At first glance this seems entirely counter-intuitive because in the ILF range we have

much less 'information density' to convey back to the client than we do in the SMR/beta range, where the training can be highly dynamic. There must be a compensating factor, and it is likely that the ILF region ties us in much more directly to the core regulatory functions of arousal regulation, affect regulation, autonomic regulation, and interoception that are foundational for our enterprise. Even more fundamentally, it is necessary for the regulatory system to maintain unconditional stability, and in this regard the ORF training has been a clear step forward in our clinical experience.

It is not only our experience with seizures that testifies to this. Indeed, we have not yet seen many seizure cases in the 400 or so days since we've had the 0.001 Hz capability available to us. The proposition is also supported by our experience with other brain instabilities such as migraine, panic attacks, vertigo, episodic tinnitus, rage behavior, and Bipolar Disorder, all of which respond nicely to ORF training with the same placement we use for seizure management.

It is tempting to suggest that a kind of 'hierarchy of needs' applies to neurofeedback, one in which brain stability is the paramount issue, and in which the foundational regulatory mechanisms should be normalized before one addresses higher-level issues. The latter include specific learning disabilities, specific sensory processing deficits, working memory, and any localized deficits such as those attendant to organic brain injury, all of which may benefit selectively from highly targeted, QEEG-guided training.

At the outset above a distinction was made between merely evoking state shifts and actually acquiring learned control. As the technique gained in effectiveness over time, this distinction has become blurred. Mere state shifts can effect major enhancements to functionality and to the brain's capacity for self-regulation. This means that one cannot use this method on a 'set-and-forget' basis. Constant vigilance on the part of the clinician is required in sensitive responders and in unstable nervous systems, in order to maintain optimization of the reinforcement parameters and to guide the process to the most propitious outcomes.

The method shapes the available brain plasticity in real time within each session, so the target of our exertions is always moving. A corresponding level of attentiveness is obviously required in connection with seizure disorder in particular, so this method should not be casually deployed. If the reinforcement parameters are not matched to the situation the person may be further moved into dysregulated states, giving rise to adverse effects which are characteristic of that particular nervous system. Unsurprisingly, this can be problematic as clinicians first encounter such a powerful method. It is therefore advisable for practitioners newly adopting this approach to get specialized training and to establish a consultative relationship with a seasoned practitioner.

In important ways we have come a long way from the early days of SMR training, and yet a basic kinship remains with the early seminal work that launched this field. At the same time, we may be re-establishing a kinship with peripheral biofeedback that has been lost along the way.

Reference:

1. Phase Sensitivity of Bipolar EEG Training Protocols John A. Putman and Siegfried Othmer Journal of Neurotherapy, 10(1), p.73-79 (2006)

Additional Resources:

Post-Traumatic Stress Disorder—The Neurofeedback Remedy, Siegfried Othmer and Susan F. Othmer, Biofeedback, 37(1), 24-31 (2009), AAPB http://www.eeginfo. com/research/ articles/ PTSD-Neurofeedback-Remedy. pdf

Neuromodulation Technologies: An Attempt at Classification, Siegfried Othmer, In Quantitative EEG and Neurofeedback, Second Edition, Budzynski, Budzynski, Evans, Abarbanel, editors, Academic Press, San Diego (2009) http://www.eeginfo. com/research/ researchpapers/ Neuromodulation_ Technologies. pdf

Infra-low Frequency Training, http://www.eeginfo.com/research/infra-low_neurofeedback.html

Protocol Guide by Sue Othmer, http://www.eeginfo.com/shop/product_info.php/products_id/47

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- Pioneer in the field of neurotherapy research and treatment, he has used neurofeedback in his medical practice for over 20 years

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Schizophrenia in Retreat

Mary Donaldson, M.Ed, Doneen Moran, BA, Stuart Donaldson, PhD

Elizabeth's Story

I was normal until the age of twelve. From then on, I descended into the pit of schizophrenia. Schizophrenia is a disorder of the nervous systems of the body which can result in many symptoms. A person who has schizophrenia may suffer from one or more symptoms to a greater or lesser degree. I suffered from a number of symptoms to a mild, yet disabling, degree.

Schizophrenia does not involve multiple or split personalities. That illness is "Multiple Personality Syndrome." Schizophrenia is not caused by poor parenting, although in many cases it is inherited. Schizophrenia is not the result of childhood trauma.

Doctors don't expect a patient's condition to get better over time. They warn you that you will have to be on medication for life. Some people experience a remission when they go through menopause or andropause, but not usually a full recovery. I am of that age, but I believe that biofeedback has helped me more than a possible remission due to menopause has.

What follows is a discussion of some of the symptoms I have suffered and how biofeedback has alleviated these symptoms. I also discuss what my life was like before biofeedback and how it has changed as a result of treatment. I will discuss subjectivity, poor rapport, poverty of thought with lack of spontaneity, pain, depression, going off medication, lack of emotion, lack of motivation, and delusions.

I was diagnosed with manic-depression, now called bipolar disorder, in January 1983 and finally diagnosed schizophrenic in 1993. In 1983 I was in the middle of completing a Bachelor of Arts degree in English Literature. I finished my degree and fell into low-paying part-time jobs (I was not expected to be able to work full-time again) punctuated by almost yearly trips to the hospital where I would be put back on medication over a period of six weeks to two months. Most of these trips were brought about because I had gone off my medication after trying one cure or another. I thought that if I could do without the medication for long enough, I could prove I did not need it at all. Except for one year when I took large amounts of niacin, I have taken medication to control my schizophrenia.

Going off medications has its price. When you experience a psychosis and then get back on medication, you never return to your former level of functioning. You return to a lesser level than what you were at before you went off the medication. As a result of going off my medication sixteen times in sixteen years, I was functioning at a much lower level in 1999 than I had been sixteen years previously. To cap if off, in 1999 I went off the medication twice and spent several months in the hospital.

As a result of going off my medication sixteen times in sixteen years, I was functioning at a much lower level in 1999 than I had been sixteen years previously.

My condition in July 2000 was pitiable. Every moment of every day I was afraid. I was either afraid or more afraid, never without fear. Think of all the symptoms of fear: stomach churning, legs tat and ready to run, mind racing. I had all of those symptoms constantly because I was constantly afraid. I also suffered from other delusions (paranoia is considered a delusion) such as thinking that people were following me, that I was being watched, that people were talking about me and other delusions. I suffered from nightmares of frustration and failure every night. I suffered from terrible memory problems. I felt my life was a failure.

I used to "research" things. I looked up words or ideas in books. At times I would go through the yellow pages looking for ideas, things that would become obvious to my tormented mind as I went through the advertisements. It was when I was looking through the yellow pages that I decided, uncharacteristically, to look under "Psychologists." I didn't think I needed any more ideas from psychologists. However, I found an ad for EEG therapy (electroencephalogram therapy). That was the beginning of biofeedback for me. The ad lead me to Dr. Stuart Donaldson, a psychologist who uses biofeedback and other therapies to treat patients' pain, and his wife, Mary, a teacher who also conducts biofeedback sessions and who is extremely knowledgeable and capable.

I knew biofeedback was what I wanted because I had had three EEG's done in 1986 which had restored my thinking quite well. I was in the hospital and had lost my memory for two weeks. My psychiatrist decided he need an EEG to determine what damage had been done. The EEG technician asked me to sit for two additional EEG's because I was able to sit quietly and she needed a portfolio of EEG's, so that she could apply for a Master's program. After the third EEG I felt more like myself and the psychiatrist said I should probably have more of them. I t was a joke, but he had noticed a difference. I tried to get EEG

Continued on page 20

Schizophrenia in Retreat
continued from page 19

or biofeedback therapy in the city where I was living, but was unable to. I moved and eight years later found Dr. and Mary Donaldson, a miracle.

I began biofeedback in July 2000. I was working two days a week, four hours altogether, as a companion to a stroke victim in a nursing home. I could understand what he was going through. Dr. Donaldson said he had never treated anyone who had schizophrenia, but if I was willing, he was willing. He gave me a deep discount on the sessions, which his wife, Mary, carried out. I more than hoped.

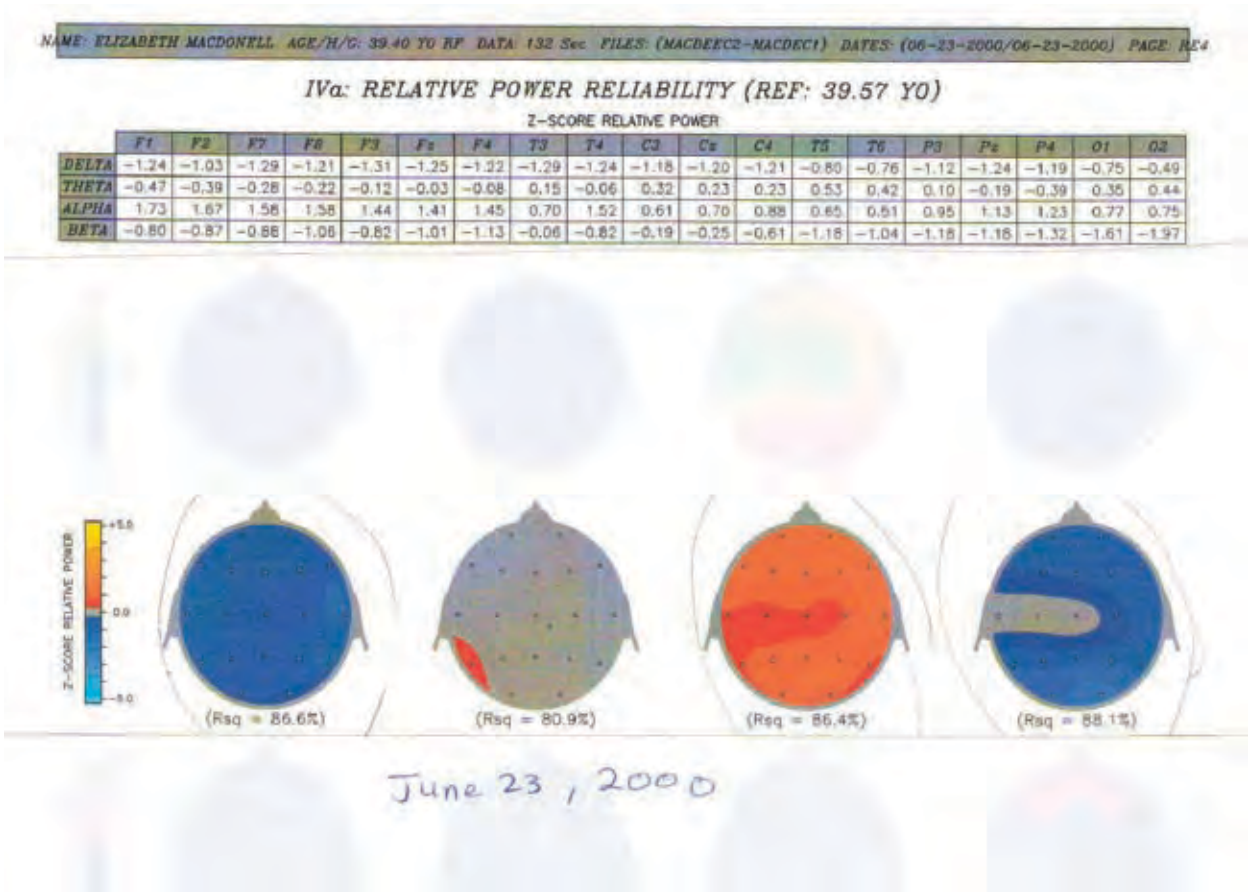
One of the first changes was that I slowly began to comprehend what other people might be feeling. From the time I first entered a psychiatric hospital, I thought principally of myself. With biofeedback the vise that held my thoughts down and kept me entirely subjective was opened and I was able consider what others might be going through, thinking. When I talked to other sufferers of schizophrenia, they said that the alleviation of this one symptom would be a great step for them. This symptom has continued to abate and now I can, at times, be entirely objective about myself, others, and given situations.

Three symptoms: poverty of thought, lack of spontaneity and poor rapport can be taken together. Poor rapport is not being able to get along with people. It comes from not understanding how another person might feel, from a lack of being able to think in an instant what to say which is what poverty of thought is and an inability to react in a timely fashion which is lack of spontaneity. These handicaps affected what work I could do. For instance, I worked in a day care in 1998 before I started biofeedback. Imagine not being able to think of the right thing, or anything, to say to a three-year-old to dissuade him from performing the wrong activity or to start the right one. I asked the other staff how they thought of things to say to the children and they said they didn't know how they did it.

Not content with their explanation, I tried to memorize what the other workers said to the children in given situations, but so often the situations were not the same. I was unable to create a speech which would suit the new situation. Lack of spontaneity is like poverty of thought. You cannot react properly on the spur of the moment to another person. I was able to speak, but not to participate in give-and-take.

Through biofeedback I began to be able to respond to what other people did or said.

Lack of emotion also contributes to poor rapport. I gradually lost the ability to feel emotion as I entered schizophrenia in my teens. Eventually I lost everything except the power to laugh. I did not cry, never regretted, was never happy, was never unhappy. I could show sympathy however. I pitied people who I thought were ruled by emotion. I easily expelled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IVa: RELATIVE POWER RELIABILITY (REF: 39.57 YO)

Z-SCORE RELATIVE POWER

	F1	F2	F7	F8	F3	Fz	F4	T3	T4	C3	Cz	C4	T5	T6	P3	Pz	P4	O1	O2
DELTA	−1.24	−1.03	−1.29	−1.21	−1.31	−1.25	−1.22	−1.29	−1.24	−1.18	−1.20	−1.21	−0.80	−0.76	−1.12	−1.24	−1.19	−0.75	−0.49
THETA	−0.47	−0.39	−0.28	−0.22	−0.12	−0.03	−0.08	0.15	−0.06	0.32	0.23	0.23	0.53	0.42	0.10	−0.19	−0.39	0.35	0.44
ALPHA	1.73	1.67	1.58	1.58	1.44	1.41	1.45	0.70	1.52	0.61	0.70	0.88	0.65	0.51	0.95	1.13	1.23	0.77	0.75
BETA	−0.80	−0.87	−0.88	−1.08	−0.82	−1.01	−1.13	−0.06	−0.82	−0.19	−0.25	−0.61	−1.18	−1.04	−1.18	−1.18	−1.32	−1.61	−1.97

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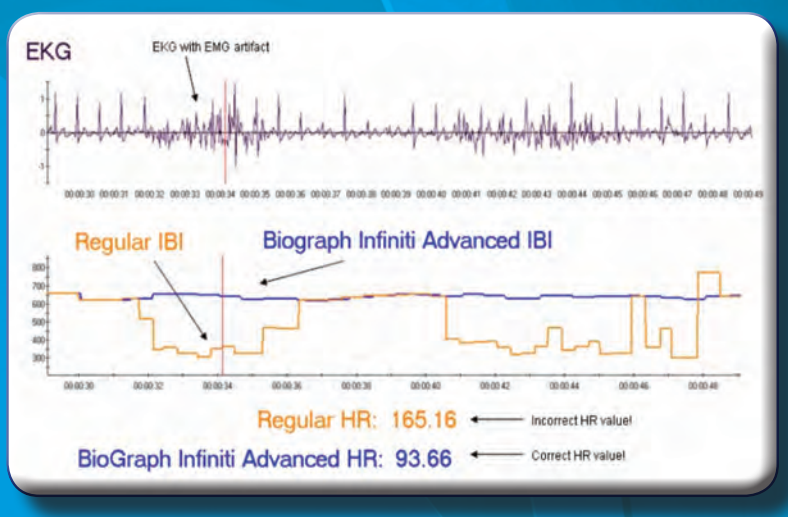

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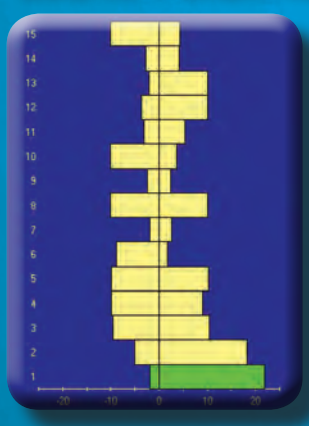

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emotions if they threatened to appear in me or if I waited long enough, which was usually not long, the emotion went away.

Imagine trying to communicate with individuals and, at times, classrooms of individuals when I presented talks about schizophrenia for the local chapter of the Schizophrenia Society, without being able to address the emotions of the people listening. People begin to treat you like you're a stick of wood. You feel awkward and apart from the world.

Through biofeedback I began to be able to respond to what other people did or said. I began to be able to make jokes. People who never asked my opinion before now listen when I have one. When I began to respond to people, they were taken aback and surprised. My mother thought I was off my medication since that was the only time I usually joked. Now they've accepted the new me which is the logical extension of the me that promised to be when I was young.

I am a more effective music teacher. So much of music is emotion and feeling and so much of teaching is finding the right thing to say at the right time. I can now address the problem of how to capitalize on what the student does well by asking them good questions, not just the only question I can think of. I have a range of choices when I am deciding what to say next. That's a far cry from having no thought at all in my mind.

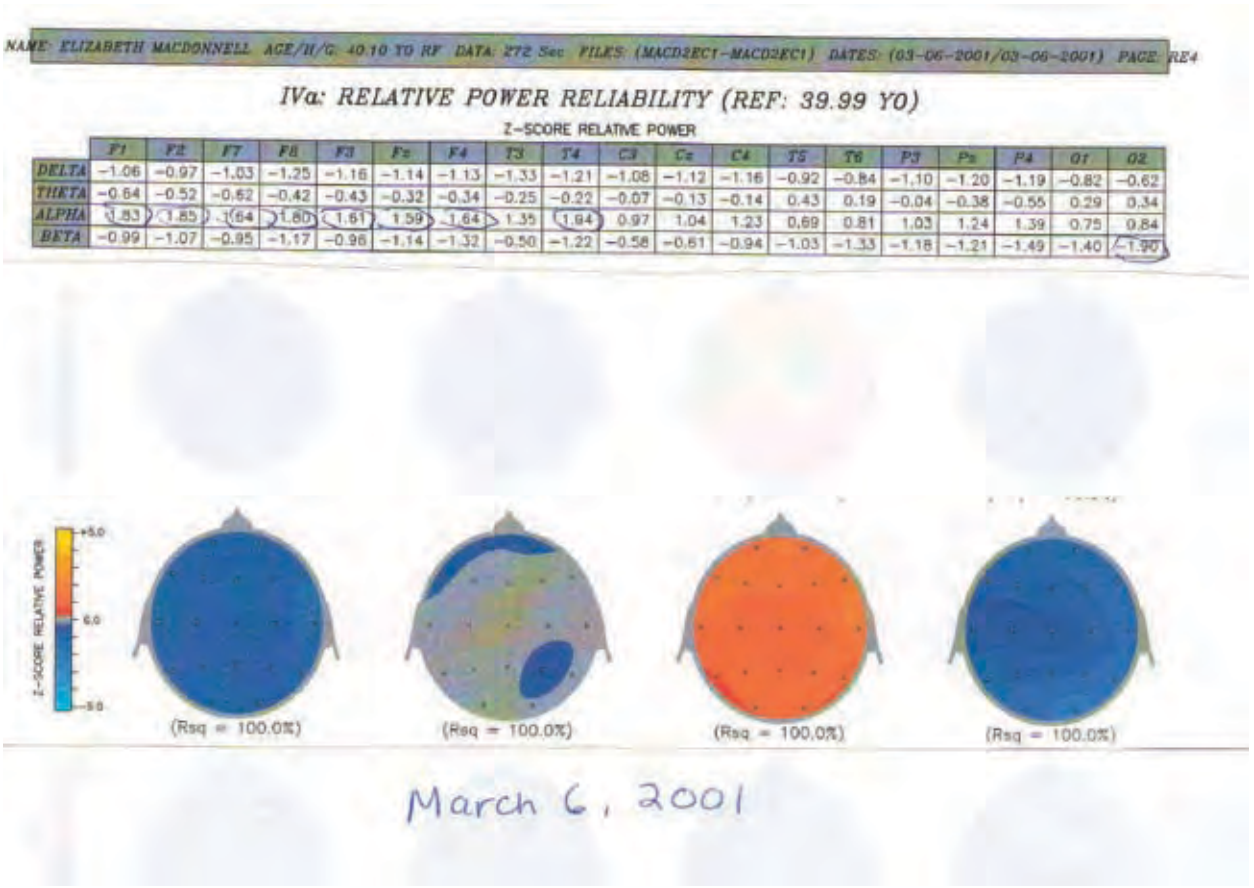
I'm again part of that world of words and interactions which, like the rings of Saturn surrounds us in our everyday world, those particles of words exchanged that surround our planets, ourselves, which look like nothing taken piece by piece, but which form a fabric, like the rings, when you see them all together. Biofeedback has given me back a world.

Biofeedback has given me back a world.

Physical pain was a part of my schizophrenia, a part I did not know I had until it began to go away. Dr. Donaldson uses biofeedback to treat people who have fibromyalgia, inexplicable pain. My pain first left my extremities and lastly left my solar plexus. I wonder how many schizophrenics have complained of pain (I know I complained of stomach pain) and doctors have either found no cause or have attributed it to the schizophrenic's craziness. Thanks to biofeedback I am now pain-free.

Depression underlies schizophrenia. At times I have taken anti-depressants with reasonable results, but after I started biofeedback, I no longer needed anti-depressants even if I was going through a tough time. Darkness and the feeling of a heavy weight on me has changed to lightness in both senses of the word. Results from biofeedback have been better than the results I got from anti-depressants since with anti-depressants I could get to a point where things were OK, but not spectacular and with more medication I

Continued on page 22



Schizophrenia in Retreat
continued from page 21

became agitated. With biofeedback I am calm, but have good energy and am reasonably optimistic.

From the moment I started on medication I wanted to go off it. I went off my medication almost yearly from 1984 until 1999, the year I went off the medication twice.

Two years after I started biofeedback, my desire to go off my medication had gone. For me and for millions who have schizophrenia this is saying a lot. Going off medication is a huge issue in the schizophrenic and psychiatric communities. To have a non-invasive technique like biofeedback available that deals effectively with even that one problem is a tremendous advance. Probably it would have taken less than a year to effect this change if I had been doing biofeedback two or three times per week instead of only once per week. Even so, once per week worked and I still have no desire to go off the medication nor have I had even during the times, once as long as three months, when I have not been doing biofeedback. The results have been permanent.

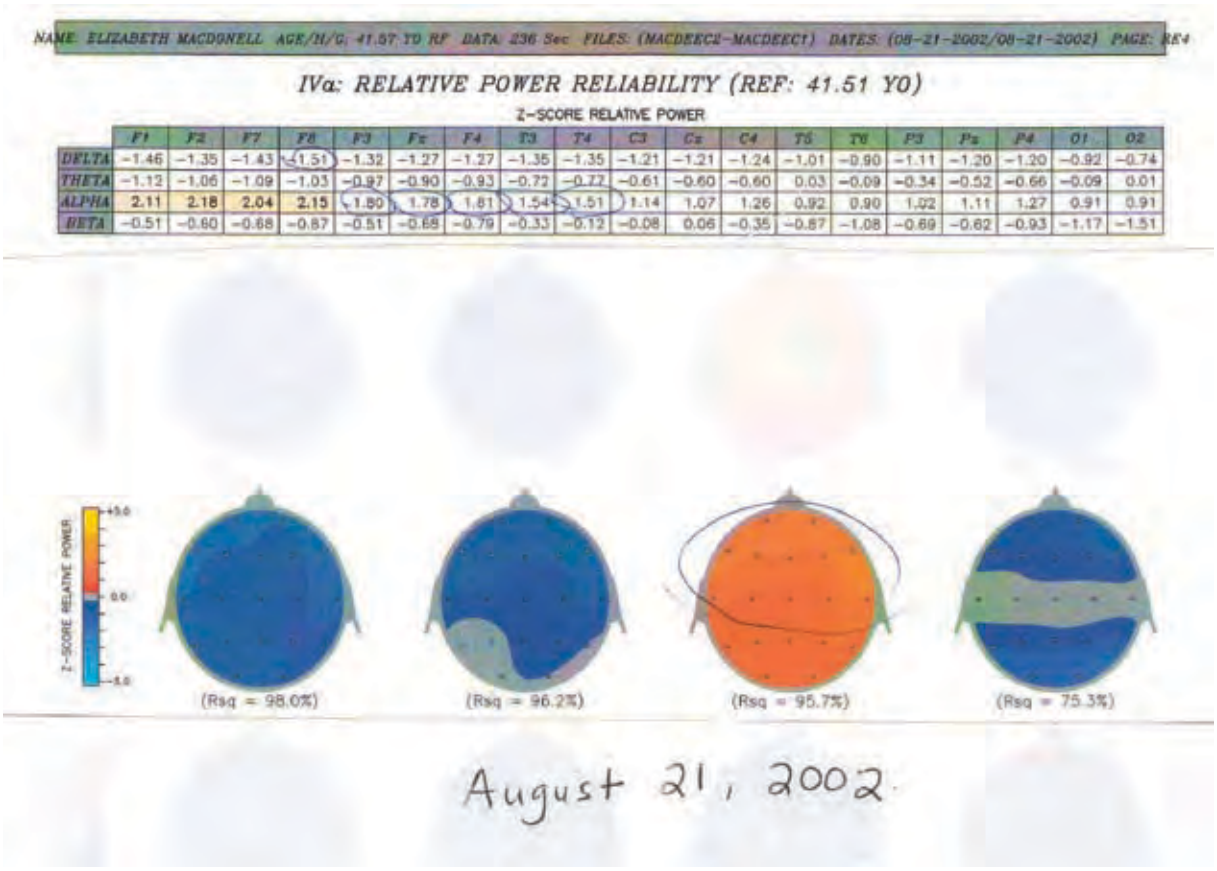
Lack of motivation is a symptom of schizophrenia that is rarely explained, but from which I suffered terribly. It means you have no inclination to do anything. You have to push yourself to accomplish everything. From brushing your teeth to going out with a friend, everything takes effort. If you are to do anything except lie in bed all day, you have to push yourself and push hard. I used to go to bed at 10:00 p.m., set the alarm for 10:00 a.m. and spend two hours every morning convincing myself to get out of bed and get started with my day. When I could get up by eleven in the morning I was doing well.

I suffered from delusions, particularly paranoid delusions, since 1980. Medication never took care of my delusions. I was afraid and delusional all day every day. At times I tried to fight against the delusions, but it never worked. Usually I just let them roll over me and tried not to base my actions on them even though it was difficult to tell what was delusion and what was fact.

After having done biofeedback for a year or so, I was looking at the two lanes of traffic on a one-way street coming at me and wondered that I was not afraid that they were coming toward me because they meant to harm me. Now I feel no fear except what a person would feel normally and I control it when it does come up. My other delusions have disappeared too.

These are only some of the changes brought about by biofeedback. Biofeedback has given my life back to me. After twenty years of continuous fear and confusion, I can now think of beginning a new career, of being capable and happy. The journey from living death to where I am now has been wondrous and there is more to come. I am still improving.

Although going off my medication with my doctor's approval was not a goal the Donaldsons' set for me, it is coming about. First I was able to go on to a milder main medication and now I have reduced that medication by nearly thirty percent and another sleeping medication by thirty percent. I can't thank Dr. Stuart and Mary Donaldson and the staff at Myosymmetries enough.



Neurotherapy of Elizabeth by Mary Donaldson M.Ed., Doneen Moran B.A., & Stuart Donaldson PhD.

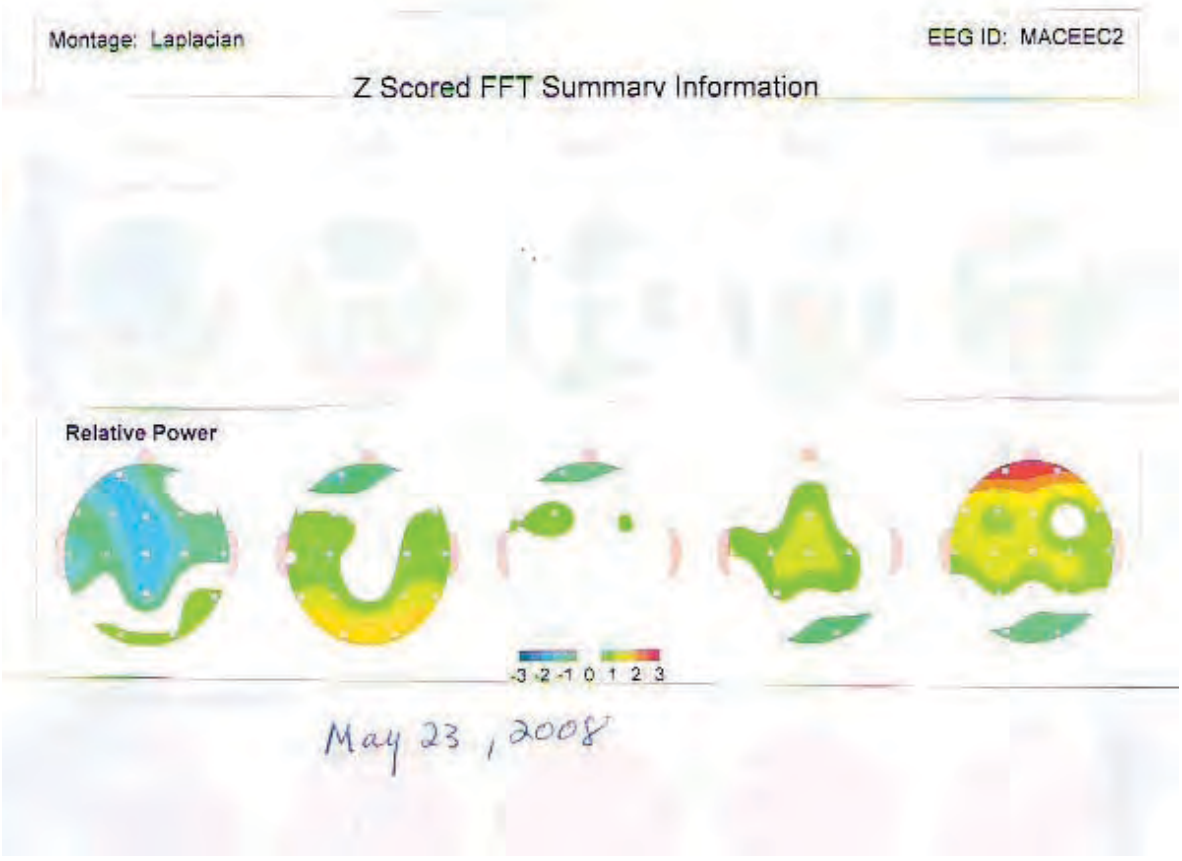
As is standard practice at the Myosymmetries clinic EEG neurotherapy treatment is based upon data generated by a qEEG using the NeuroGuide Database by Thatcher. The general strategy utilized in this situation was to downtrain sites with excessive activity and uptrain those sites with decreased activity. Therapy was initially conducted using the Autogen system, then latterly the BrainMaster 2x2 system. Therapy lasted over a period of several years starting in May 2000 and continuing at this clinic until November 2006. After 2006, therapy was continued using a home training system (BrainMaster) with contact only as needed. While an attempt was made to keep the appointments regular over this time period there were several interruptions due to hospitalizations, illness and holidays. In total Elizabeth was seen 297 times over the course of attendance at the clinic.

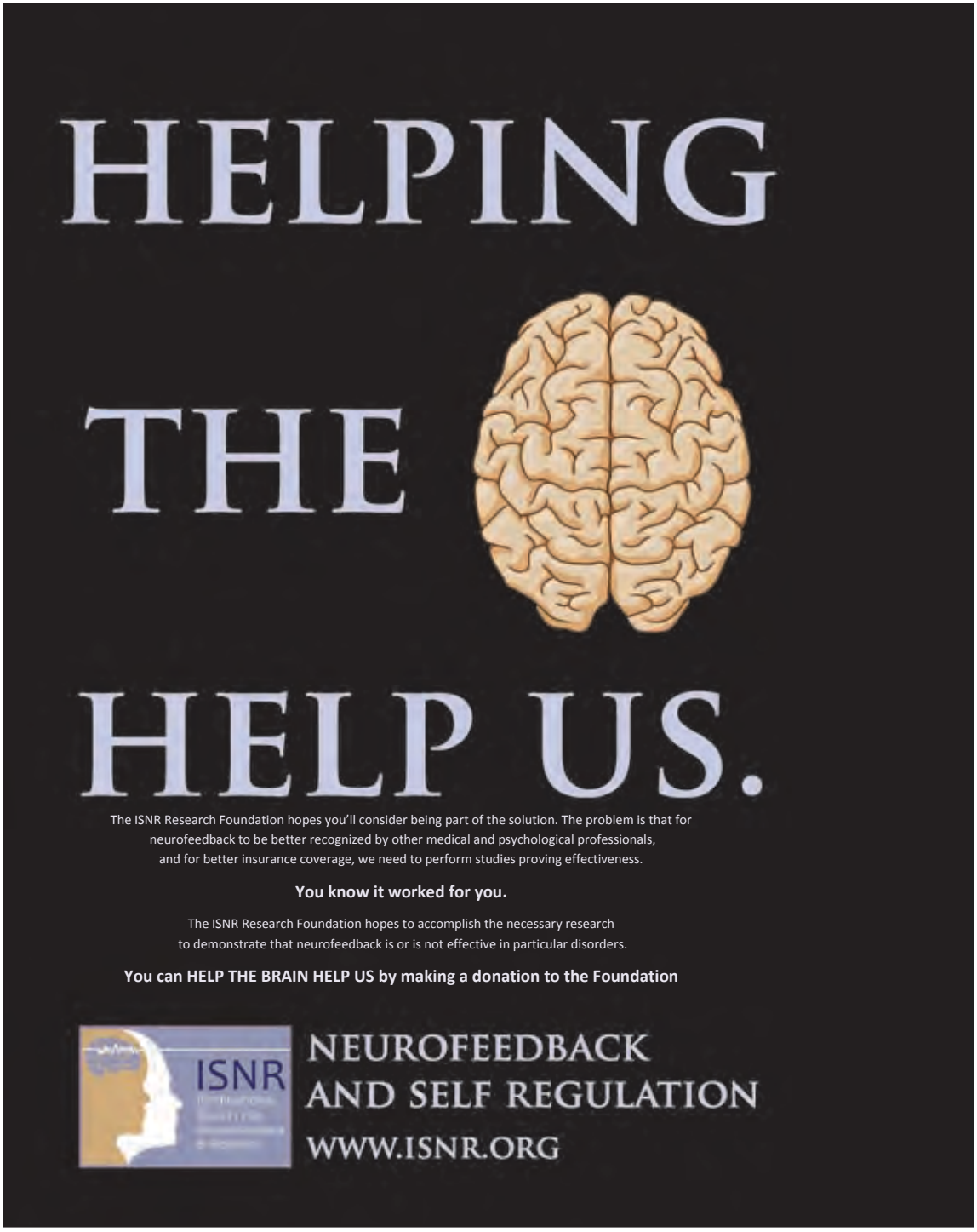
Four qEEGs were conducted over the time period of June 2000 to August 2002 with the client on and off medications. The Z score power was remarkably consistent over the testing showing little change except for some changes in activity along the sensory motor strip. In general Delta was decreased 1 to 2 standard deviations (SDs) throughout the brain; Theta was within normal limits for the first 3 qEEGs decreasing slightly on the fourth. Alpha was significantly elevated (1 to 2 SDs) throughout the entire brain to all tests. Beta was significantly decreased (1 SD) throughout the entire brain. As mentioned the only change occurring was along the sensory motor strip which gradually increased in activity level in Beta from the first qEEG to the 4th. Interestingly phase and coherence were never significant for any site for all 4 tests.

During this time period routine EEG neurotherapy was conducted at CZ with the goal to increase SMR (12 - 14 Hz). Delta was never altered due to a belief that it should not be played with. As mentioned progress was gradual but clinical observation suggested improvements as outlined above.

A qEEG was repeated again in May 2008. Due to concerns about the effects of medications Laplacian data analysis was utilized. The results showed a normalization of all frequencies (except Delta) for the sensory motor strip. Incidentally home training was conducted at CZ and more recently at FZ. The improvement or normalization showed as a pattern of normal activity in the frontal, central and parietal areas forming an H pattern when viewed from above. This pattern was found in Theta, Alpha, and Beta frequencies. High Beta showed improvement frontally at F3, FZ and F4. Delta continued to show decreased activity especially at FZ and to a lesser extent surrounding FZ. High Beta was significantly elevated at PZ.

Neurotherapy continues today with Elizabeth doing home training and calling for help as needed. Some people argue that age produced these improvements while others suggest it was due to the medication changes. Regardless it is nice to be able to help improve someone's life. Elizabeth is presently taking courses to help her get started with a new career and life.





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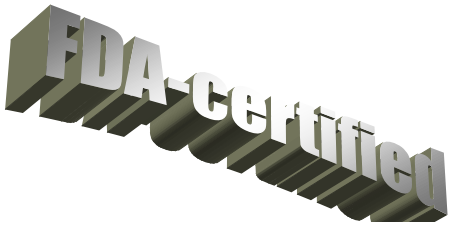

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MindFull

The Neuroscience of Consciousness

David Kaiser, PhD

Until investigators of consciousness give up the belief in conscious singularity they will be looking at the structure of their own preconceptions, rather than the physiological activities subserving consciousness. — Joe Bogen

Studying the evolution of human consciousness has its own set of traps. One must be careful with the term *consciousness*, which many scientists avoid, as consciousness can be defined in so many ways. As reactivity to stimuli, bacteria are conscious entities. As a capacity for self-direction, dolphins, chimpanzees, and most mammals are conscious. The late Joseph Bogen MD taught a course called "Consciousness" at UCLA for psychology and neuroscience students as part of the neurobiology curriculum and if you knew Joe, the course was like him, confrontational in nature and erudite. I used to say that Joe was George C Scott on angel dust. He certainly could fill a room.

Joe made us read ancient papers, some of which I swear were in Egyptian hieroglyphics, and the primary argument was that the thalamus is all we need to generate consciousness. He described thalamic functions as they lined up with properties of consciousness. Many of the papers are relevant to our understanding of EEG rhythms, although what follows is a fast-forward through the vast seas of neuroscientific research, a jumpy stream that resembles consciousness itself.

Horace Magoun and his colleagues identified an arousal or alerting system in the brain in the 1950s, a specific segment of brainstem (in cats) which aroused the animal from sleep whenever stimulated. Lesioning areas near this segment but not part of it, such as lesioning the afferent pathways, left a cat behaviorally attentive, but lesions of the ascending reticular activation system (ARAS), the name first given this segment, resulted in unconsciousness or deep sleep. Magoun concluded that the ARAS was the first point in the encephalon to maintain "central alertness" of the waking state, what others would call the first point of consciousness.

Joe followed Magoun with visual and attentional papers, one more technical than the next. Skinner & Yingling (1977) summarized the three primary measurements of neuroelectricity related to attention: (1) EEG desynchronization of 8-12 Hz, (2) large amplitude EPs, and (3) negative slow potential shifts in prefrontal cortex. The mesoencephalic reticular formation (MRF), as the ARAS came to be called, along with the mediothalamic frontal cortex system (MTFCS) and thalamic reticular nucleus (RTN) were all implicated in attentional operations according to neuroelectrical literature. The MRF was determined to be excitatory, phylogenetically older, not specific to any sensory stream (modality nonspecific), and diffuse in action. It is partnered up with its functional complement, the MTFCS, which is inhibitory, modality specific (visual only, or auditory only, etc), and with short effect. The MTFCS inhibits ascent of irrelevant sensory stimulation in order to keep orienting reactions under control. Together with the regulatory capacity of RTN they act as a highly selective gating mechanism. By regulating MRF and MTFCS inputs, the RTN controls passage of information to the cortex, delegating which sensory events are to be attended and which are to be ignored.

Livingstone & Hubel (1988) identified two independent and distinct systems of visual perception, parvo and magno. The parvo system is sensitive to color, unable to exploit contrast, slow in temporal resolution, but with high spatial resolution, whereas the magno system does not detect color , has low spatial resolution but can exploit contrast and has fast temporal resolution. Again a complementary partnering exists in this system higher up the sensory processing pathway. The parvo-temporal lobe system identifies visual objects and the magno-parietal lobe system locates objects in absolute and relative space.

Hubel (1988) describes how visual centers evolved to detect and decipher moving objects. This means that for examination of stationary objects, the system has to resupply the movement, which is done by a series of saccades and microsaccades (movements of the eyes). Apparently everything was on the move in the phylogenetically distant past, much like in the early universe.

Much of what we do is focus a person on his or her intentions, changing brain rhythms without leaving the chair.

As neurotherapists, we impact the EEG rhythms of attention and motor response and the flow of consciousness. Much of what we do is focus a person on his or her intentions, changing brain rhythms without leaving the chair. The complexity and organization of intentionality is served by the complexity and organization of neurophysiology. Although the level of neuroscientific knowledge summarized above may seem sporadic, as was the course on consciousness, we can take from it at least one principle, the principle of complementarism, a yin/yang, a male/female of brain organization serving consciousness.

We should keep this in mind when we challenge the brain with operant conditioning. When we push the brain in one direction, it has the tendency and an evolutionary history to push back. For every action there is an equal and opposite reaction. Newton's third law of motion exists at every level of

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Mindfull

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neurophysiology, at least those systems that serve consciousness. This understanding leads us to the understanding that nonconscious systems have to follow the complement of complementarism, which is pluralism, number without purpose, existence for existence's sake. And when such systems eventually butt head with conscious systems, consciousness readjusts and reacts to their actions, as consciousness is always a product of balance.

We left neuroscience for philosophy on our final day of the class. Turing (1950) posed the question "Can machines think?" as computers were just becoming digital. Our brain is biochemical, a carbon-based process, part analog, part digital due to refractory periods and threshold potentials in neural firing, but Turing wondered aloud whether a mind could also emerge in transistors, in the geochemistry of computing (silicon, gallium, germanium). Objections against such a possibility exist -- theological, mathematical, and psychological. While it's true that a machine can have a syntactical relationship with the world in that it is able to follow formal rules of symbol manipulation, no one has proven it can have a semantic relationship with us, that a computer knows what each symbol refers to in the real world. Computers are binary creatures, one and zeroes, the movement of electrons without regard to higher representations of information. We have a conscious mind because it serves our survival, it helps us attain metabolic goals. But if a computer has all its needs met, if the electrons flow without purpose, it can never develop actual semantic relationships. Semantics runs to our core -- what to eat, fight, flee, or mate with, and all shades in between. Only when a computer has metabolic requirements and must select which objects from the world to consume and which objects to avoid, only when the world impacts its certainty of existence, might the twinkling of transistor-based consciousness emerge.

We have a conscious mind because it serves our survival, it helps us attain metabolic goals.

Here is Joe's website, a plethora of scientific investigation into consciousness, split-brain research, and creativity, a website I once labored over, http://www.its.caltech.edu/~jbogen/

Underlying Treatment Issues in Neurofeedback as Exemplified by Treatment of Seizure Disorders

Part 1

Len Ochs, Ph.D.

It is my observation that while epilepsy, the recurrent abnormal brain electrical events still take center stage for medical treatment (drugs and at times surgery), there is a wide range of brain electrical phenomena that influence, if not govern, behavior. Not only is neurofeedback becoming more prominent as a tool that can ameliorate many types of epilepsy – Barry Sterman's work is certainly still influential here – but neurofeedback can make its presence felt over the spectrum of disorders. Much of what I present here will be from my own experience of nearly 35 years of using neurofeedback – both standard and the Low Energy Neurofeedback I've nurtured over the past 20 years. (And during that time the LENS has changed considerably. While preparing this article, I had the opportunity to review many of my partial and complete treatment failures over the past 10 years of using the LENS, and was not at all surprised to see how much of role brain irritability—the other end of the continuum from periodic convulsions—played in my treatment failures. It's only been over the past year that we've seen the development of what may be much better ways of approaching the identification, evaluation and treatment of seizure activity.)

Some instances of epilepsy, when thought of as recurrent episodes of convulsions of varying degrees of involvement, may only be treated by medication and surgery at this time. And these seizure problems may only be fully evaluated by physicians and medical tools. There are inherent limitations of the medical approaches, however. These limitations generally concern themselves with the narrowness of the way brain irritability has been viewed, as exemplified by the following story. A patient, in this case a very bright, profoundly deaf computer programmer, is subject to recurrent tonic clonic seizures on nearly a nightly basis. Late in my career as a psychologist, I was referred this patient when he was an inpatient at a local psychiatric locked unit. He was there as a result of his latest suicide attempt. It seems that before his nightly seizure he would be awakened from his sleep by what he describes as a sound like a jet engine near his head. He knew from this auditory aura that he would soon lose consciousness, fall from the bed, and start on a too-frequent trip into days of exhaustion, and loss of work and family time. He was, to say the least, depressed. He was admitted to Stanford University Hospital for a sleep study and had his usual seizure. It was recorded on video tape. He shook, and his lips turned blue. He was a mess the next day. He was embarrassed when the neurologist looking at the raw EEG record from the previous night, not seeing any of the signature spike and wave activity pronouncing the seizure a "pseudo seizure," also known as psychogenic or nonepileptic seizures (Rowan, A.J. & Gates, J. R., 1993), which the patient interpreted incorrectly as something that made up and not founded in fact.

Here's a story from the world of neurofeedback assessment and treatment that will later bring us back to better understand what happened in the hospital to the above patient. In 1976, when I was first trained in the use of brain mapping and EEG in the clinical treatment of problems such as head injury and ADD, high amplitude EEG was considered a sign of pathology. If we saw high amplitude frontal delta it was a sign of brain damage. Thus, when amplitudes decreased it was a sign that people were getting better. For years this is what I saw; and this is what I taught my students, and we all rejoiced when the amplitudes dropped. Several years ago, however, I began to notice some EEG amplitudes rising in the session data of people in treatment with me. While some site amplitudes were initially low at the start of treatment, their amplitudes and Standard Deviations rose as treatment progressed. In contrast to what I expected, however, I also noticed that the client's functioning increased as much from amplitudes rising as it did from amplitudes dropping. And if this sounds a little like what Val Brown has been saying I think he's correct in this respect. In addition, I thought that the newly-increased amplitudes and variabilities would eventually drop, once more restoring the order the universe formerly had. But this frequently did not happen. These amplitudes and standard deviations remained high, as did the clients' high functioning. But frequently the amplitudes that rose with treatment did not return to their former level.

Then came the fearful cries from the practitioners using the LENS system. Topographic brain maps are done. The users of the LENS approach encountered the same problem of rising amplitudes in their maps. They had no context for evaluating the significance of these phenomena. They thought that the maps were indicating that the treatment was failing, and that they were doing something wrong.

This is the first observation: formerly low amplitude EEG could rise and become more variable and function would improve with it.

Whenever they complained that their treatment was going amiss because of the maps showing rising amplitudes and standard deviations, I asked them "How is the patient doing?" By far the large majority of the time they indicated that the patients' functioning was improving markedly. This is the first observation: formerly low amplitude EEG could rise and become more variable and function would improve with it. The second observation is that EEG sig-

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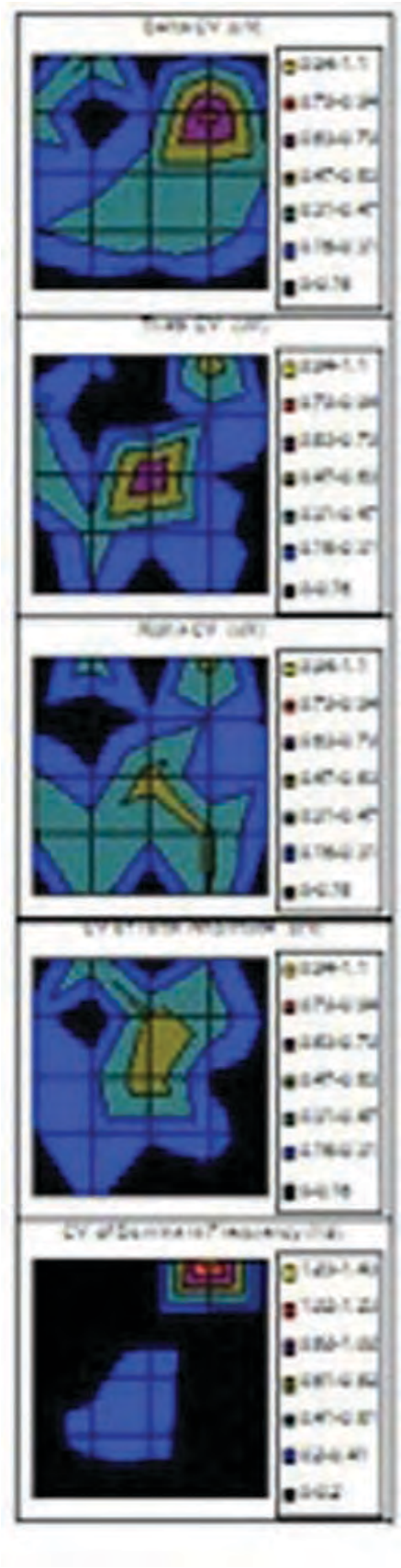

Figure 1.

Seizure Disorders

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nal amplitudes could drop throughout the LENS treatment and EEG activity could become less variable as a sign of increasing pathology. This smoothing process could make both the raw and measured EEG signals lower in amplitude and variability. I searched the EEG and neurology literature for an explanation of this process and found it to be sparse. There is a phenomenon in the literature called EEG "suppression." It refers to the lowering of amplitudes, but I found nothing except in Val Brown's talks about decreased EEG variability.

Let's go back to the sleep study I was talking about before. Remember, the neurologist saw no evidence of spike and wave, and therefore pronounced the problem one of pseudo seizures. It seems reasonable to me to look at such phenomena as a reasonable smooth raw EEG waveform as suppressed when it occurs in the presence of convulsions. In Fig. 1. (Fisch, B. J., 1991) Elements from waveforms 1, 2, or 3 may be seen after a convulsion, instead of any of the others, which might be expected if a seizure was involved epileptiform activity.

I hypothesize those waveforms 1 – 3 can be suppressed forms of any of the others. Waveforms 4 – 10 can be neurochemically integrated or smoothed, resulting in Waveforms similar to numbers 1 though 3. This means that the amplitudes will be lower and their variability, or spikiness, smoothed.

There are two criteria for the hypothesis of suppression: First, the waveforms of those who have convulsions of various types show more variability with both maturation and exposure to the LENS approach. Second, there are cognitive, mood and motor dysfunctions that are present in seizure patients when the waveforms are smoothed (examples 1 – 3 above) and are remediated when the waveform shows greater spikiness with the removal of suppression.

In suppression, the real spike and wave would have been minimized, smoothed, and integrated by the action of the same inhibitory neurotransmitters that are the defenses against seizures. If the implications of what I'm saying have any merit, the raw EEG may have to lose its status as the gold standard of the meaning and implications of the EEG behavior under these conditions. In fact, the raw EEG, when suppressed, may present an inaccurate and deceptive picture of what's really going on: lingering seizure activity, on the one hand, or just plain irritability and reactivity of the brain, on the other hand.

It seems reasonable to me that these inhibitory neurotransmissions, the brain's attempt to block the spread of seizures, would also inhibit and impede the brain's electrical activity that provides our higher functioning. There are two consequences of these neurochemical blockades. First is the requirement of higher energy expenditure to overcome these neurochemical blockades, and the second, a frequent accompaniment to seizures, would be functional impairments that remain calling the brain to protect itself against the seizures.

Functional deficits are often tied to neuropsychological nuclei or brain locations that are thought to be important to activate the missing functions. A brain site might be less than functional internally to that location, or less than functional in its communication with other sites in a network. An example might be a site important to some aspect of speech, or to auditory processing. While this may be completely true, my own observations of patient *fatigue,* and functions such as initiation or completion, may be more related to how hard the patients need to work to overcome their own neurochemical barriers to the connectivity within the brain, which are needed to achieve high functioning. The more the brain is defensively blocked in its communication with itself, the greater the patient's experience of fatigue due to trying to overcome these blocks to functioning. In other words, instead of looking at ADD/ADHD as representative of dysfunctional physiology, it may be more useful to look at fatigue issues being caused by blocks to communication and connectivity in the brain. These blocks make it difficult to initiate, complete, think, organize, attend, sequence, remember, and so on. When these functions are reduced, the patient looks and sounds less motivated, when in fact their fatigue comes from trying so hard that they wear themselves out. That there is a chemical basis for this function is echoed in the productivity of a hypomanic's behavior, or the reduced neurocognitive functioning in older-aged males, or in those who

have illnesses that interfere with the levels of serum testosterone. The volume of the inhibitory blockade or the volume of the excitatory communication may determine the ease with which communication takes place in the brain, and consequently which functions present as impaired or adequate. It is saddening to me that the processes that impair the functioning of individuals also impair their ability to relate to the health-care system – or even look interested in helping themselves. It then becomes much easier to blame the victim because they appear so unmotivated. It then also demands more sophistication in our estimates of who is malingering or committing fraud in looking for health benefits. Let's now move from the issues of energy to working EEG suppression.

Now here's where working with the brain's defenses becomes interesting. We often thought that it would be risky to release the suppression - that which the brain has put into place to provide protection from seizures. I certainly was on the lookout for seizures that arise from the lifting of suppression. The surprise was that to date we have not seen any seizures occurring as the amplitudes and standard deviations rose. Instead, only functionality increased, in nearly all seizure patients, before the seizures themselves started to reduce in duration and leave the patient with a shorter period of post-seizure fatigue.

I imagine the reason we have not seen any seizures is that evolution never expected us to live as long as we do. This is a recent phenomenon. Because we live increasingly long lives, the mechanisms that remove the neurochemical inhibitors have not had a chance to evolve. Thus, the clients' ability to refresh their own neurochemistry is impaired, compared to the better ability of the people who do not have lingering problems, and who have evolved to refresh their own neurochemistry.

There were two instances in which untoward problems did occur from LENS treatment. First, some patients have had childhood or adolescent seizures, tics, explosions, or migraines. Those patients who had resolutions of these problems early in their lives did have a relatively brief reoccurrence of them if, later in their lives, they sought treatment for residual functioning problems. We soon learned that the functioning problems of people who had previous severe neurological issues were clues that the resolutions of their seizure-type problems were incomplete. The existence of functional problems began to look as if they were caused by persistent neurochemical inhibition still in place because their neurological problems had not fully resolved themselves.

The second instance is when people had sought the help of neurofeedback to make their seizures, tics, and migraines go away, yet they still had functional problems (while the organic problems for which they sought treatment extinguished). These people did have short (1-week, on average) recurrences of seizures. We learned to inform them of this probability so they could be better prepared for temporary reoccurrences of these problems.

Finally, I estimate how much irritability resides in the physiology of the brain by how much variability there is in the EEG

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Seizure Disorders

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signal. While not convulsant in quality, or epileptiform (spike and wave), there is, nevertheless the kind of energy that rattles the EEG signal and makes it vary, as measured by an EEG signal with a high standard deviation. As a preliminary experienced-based estimate, I propose that an EEG standard deviation of less than 25% of the amplitude at the site from which it is being measured is an EEG that is suppressed, based on our experience with the LENS Suppression mapping. Beyond that, I estimate on the same basis that an EEG standard deviation that is greater than 35% of its measured amplitude at any site has too much energy, which would allow it us to see less change from neurofeedback than we would hope and expect to see. Of course this is an over simplification; and there is much to be learned about the phenomena of seizures, irritability, and suppression. But this is a starting point from which we can begin to explore this new paradigm. Here are some examples of EEG amplitudes that rise with improvements in functioning.

CASE 1: A five year-old girl, born with cerebral palsy, seizures, with unknown intellectual capacity.

On the left in each of these examples is the LENS Suppression Map; on the right is the standard LENS map.

The suppression maps plot the coefficient of variation, the ratio of the standard deviation of the variable to the amplitude of the variable. The squares on the left in the suppression map are top-down view

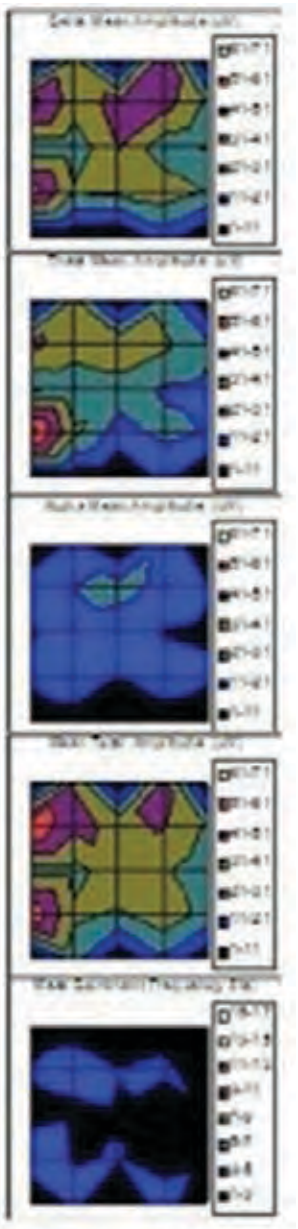


Figure 2.

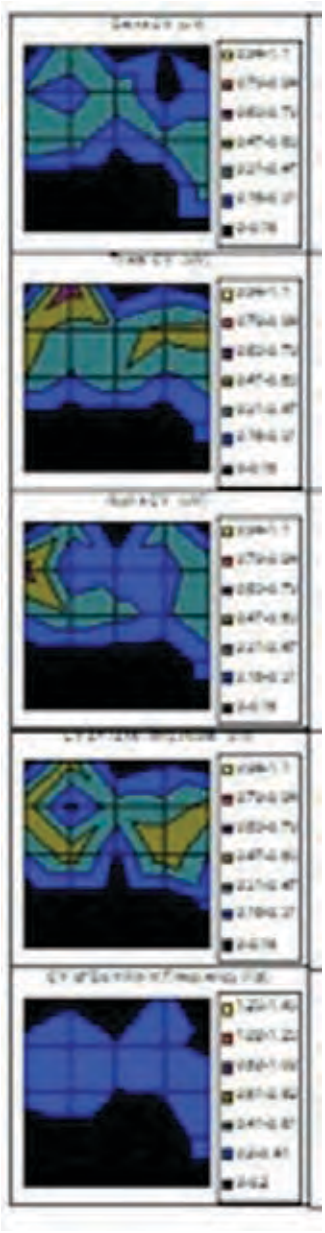


Figure 3.

INITIAL MAP

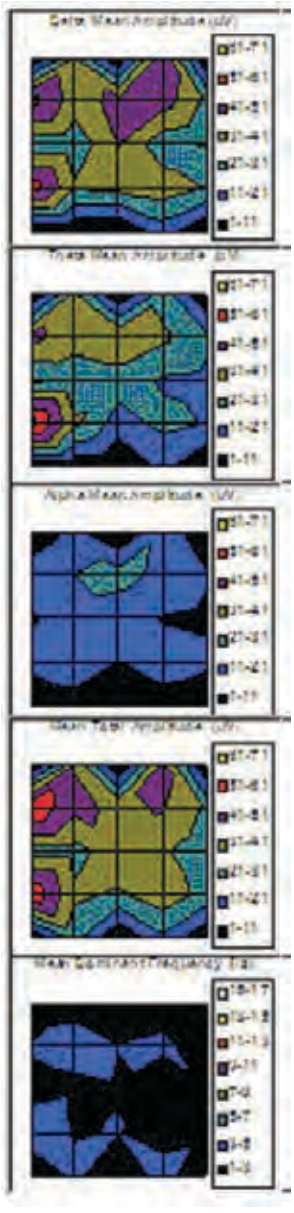


Figure 4.

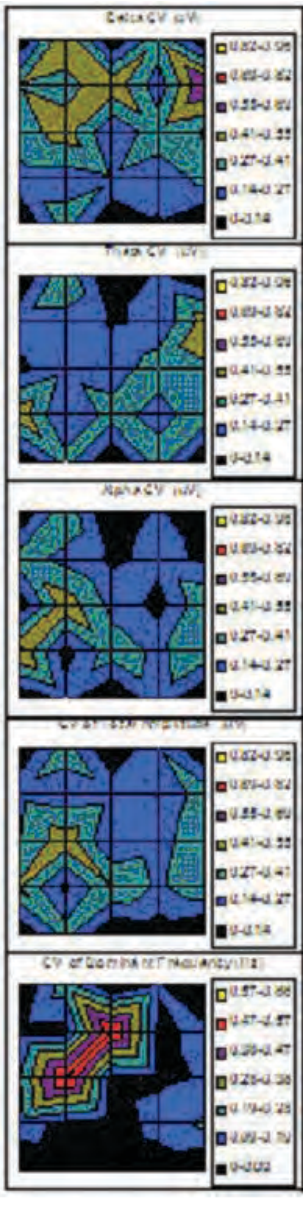


Figure 5.

FINAL MAP

Case 2: a 45-year old female with multiple personality disorder and psycho-motor seizures. Twelve years of psychotherapy.

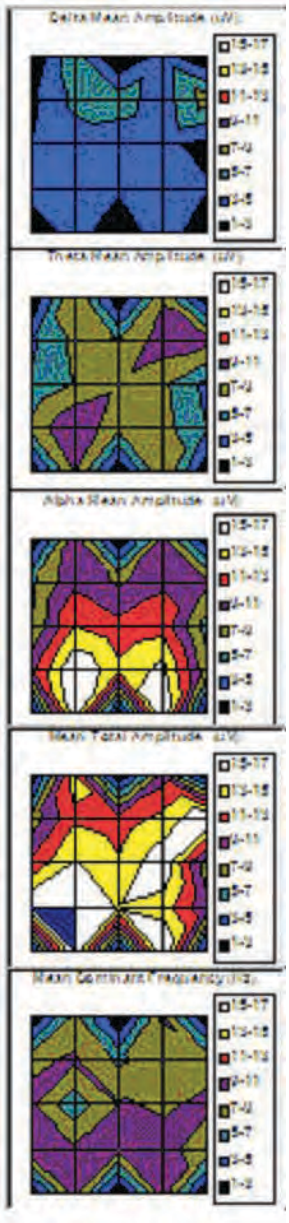


Figure 6.

Figure 7.

INITIAL MAP

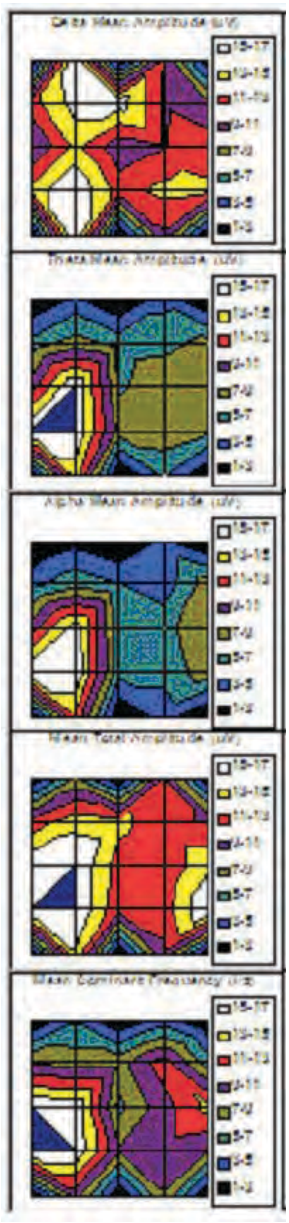


Figure 8.

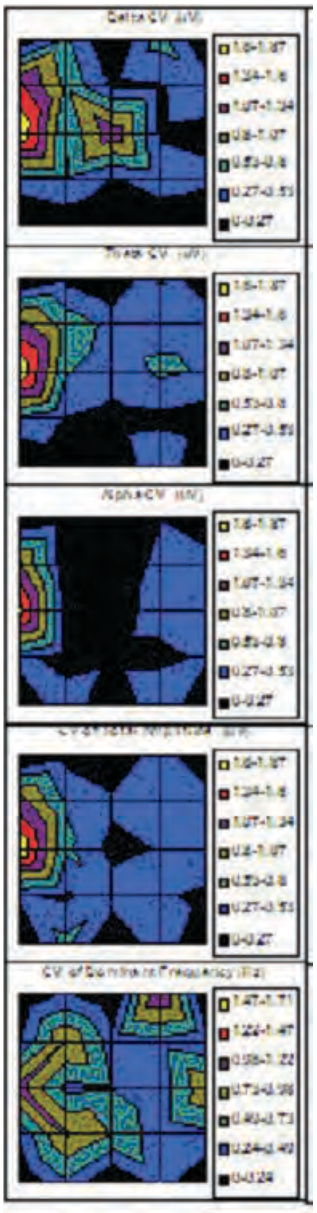


Figure 9.

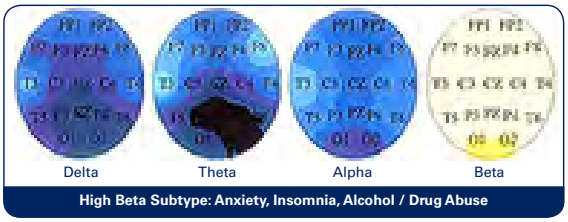
FINAL MAP

Underlying Treatment Issues in Neurofeedback as Exemplified by Treatment of Seizure Disorders Part 2 will be printed in the summer 2010 issue of *NeuroConnections*

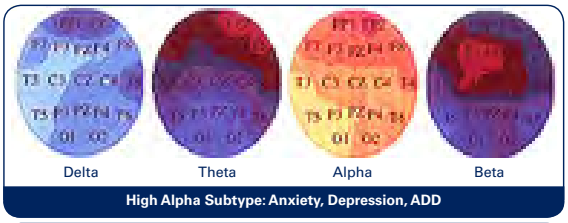
SCOTTSDALE NEUROFEEDBACK INSTITUTE

QEEG mapping service

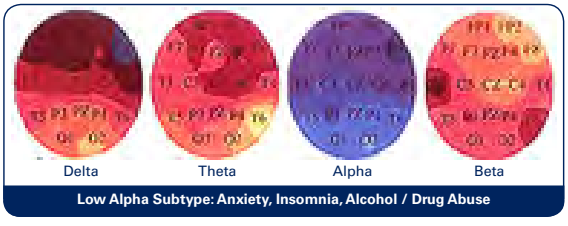
QEEG / TOPOGRAPHIC BRAIN MAPS:
Generalized Anxiety Disorder Subtypes



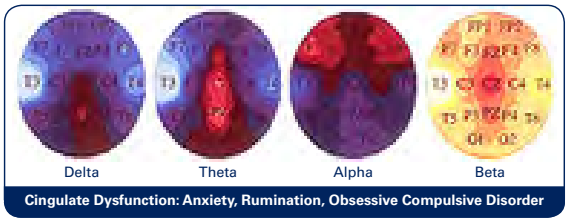
High Beta Subtype: Anxiety, Insomnia, Alcohol / Drug Abuse



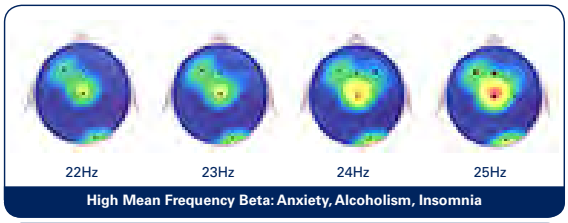
High Alpha Subtype: Anxiety, Depression, ADD



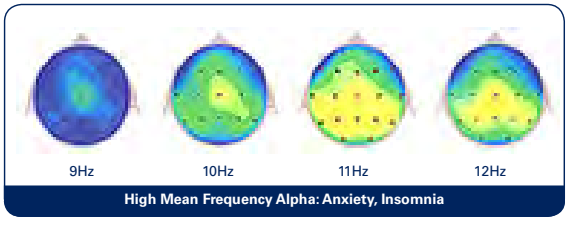
Low Alpha Subtype: Anxiety, Insomnia, Alcohol / Drug Abuse



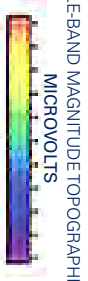
Cingulate Dysfunction: Anxiety, Rumination, Obsessive Compulsive Disorder



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**
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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
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Established 1982

Dear ISNR Members—

The Public Relations Committee is in need of your assistance to vastly extend the range of their efforts in two simple ways:

1. Providing suggestions of media targets for the PR Committee and Board of Directors to address with letters and other types of contact. The working definition of "media target" would be

- national television programs,
- radio programs,
- professional organizations,
- patient or organizations concerned with neurofeedback potential disorders
- continuing education organizations for various professional fields that should be knowledgeable about or referring for neurofeedback,
- prominent individuals who have either written about a disorder that neurofeedback improves or
- celebrities who themselves have gone public with a disorder or bothersome symptom that neurofeedback likely would successfully address
- science or other journalists that might be interested in neurofeedback

2. Taking the initiative and a few minutes to send individual faxes, e-mails, or hard copy letters to media targets that are locally based or of particular interest to you as individual providers. As an assist, the Committee has written a form letter that you can tailor to your own style to fit specific situations of which you become aware. Contact Grayce Stratton at DrGrayceStratton@aol.com for further assistance.

The Committee will compile a listing to be made available to the entire membership when complete.

The Public Relations Committee:

Sarah Prinsloo (Committee Chair)
Grayce Stratton
Cindy Perlin
Dianne Roberts Stoler
Kathy Abbott
Tom Collura and Cynthia Kerson [ad hoc as President and Executive Director (respectively)]

ISNR 18th Annual Conference

near Denver, Colorado

September 29-October 3, 2010
Preconference Workshops Sept 27-29

Confirmed Keynote Speakers to Date

Norman Doidge, MD

Norman Doidge, M.D., is a psychiatrist, psychoanalyst, researcher, author, essayist and poet. He is on the Research Faculty at Columbia University's Center for Psychoanalytic Training and Research, in New York, and the University of Toronto's Department of Psychiatry.

Dr. Doidge served as Head of the Psychotherapy Centre and the Assessment Clinic at the Clarke Institute of Psychiatry, and taught in the departments of Philosophy, Political Science, Law and Psychiatry at the University of Toronto. He has published on trauma, problems in love, psychiatric diagnoses and intensive psychotherapies, and is the author of standards and guidelines for the practice of intensive psychotherapy that are widely used in Canada. In 1993 he presented his early research at the White House in Washington, D.C., and is credited with helping preserve these treatments as part of the Canadian and Australian health care systems. He is a Training Analyst (a trainer of psychoanalysts) in the Canadian Institute of Psychoanalysis. Dr. Doidge has won a number of scientific awards, including the U.S. National Psychiatric Endowment Award in Psychiatry; the American Psychoanalytic Association's CORST Prize in Psychoanalysis and Culture; the Canadian Psychoanalytic Association's M. Prados Prize; and election to the American College of Psychoanalysts for "many outstanding achievements in psychiatry and psychoanalysis... and national leadership in psychiatry." He was recently awarded the Mary S. Sigourney Prize, the highest award in international psychoanalysis, and the National Association of Mental Illness Ken Book Award. He is a reviewer for the Harvard Review of Psychiatry.

Jonathan Marks, MA, BCL

Currently a fellow at Harvard Uninversity's Edmond J. Safra Foundation Center for Ethics, Jonathan Marks is associate professor of bioethics, humanities and law at Penn State University, and director of the Bioethics and Medical Humanities Program at the main campus, University Park. His mission is to develop the bioethics curriculum and strengthen interdisciplinary and collaborative scholarship in the field, bringing together dynamic scholars from liberal arts, medicine, life sciences and law, within his own institution and in the academic community at large.

Much of the literature in bioethics is concerned with micro-bioethics questions, often involving discrete issues of patient care. Although these questions are of considerable importance, Jonathan is particularly interested in exploring macro-bioethics issues involving, for example, the impact of industry and national security funding on biomedical research, access to health care, and the intersections between environment, public health and human rights. These issues are just as important but often neglected, particularly in mainstream media.

Alvaro Pascual-Leone, MD, PhD

Dr. Pascual-Leone is the Director of the Center for Non-Invasive Brain Stimulation and Professor of Neurology at Harvard Medical School and the Beth Israel Deaconess Medical Center in Boston.

Dr. Pascual-Leone is Board Certified in Neurology and Neurophysiology by the American Board of Psychiatry and Neurology. His focus is to understand neural plasticity at system's level. He seeks to identify rules that are invariant across neural systems and domains. He believe that plasticity is the normally ongoing state of the nervous system and that a coherent account of any neurocognitive theory and neural system has to contemplate plasticity as an integral property of the nervous system and the obligatory consequence of each sensory input, motor act, association, reward signal, action plan, or awareness. In this framework, notions such as psychological processes as distinct from organic-based functions or dysfunctions, or of "good" and "bad" plasticity, cease to be informative. Plasticity is the reason for development and learning, the cause of disease, and a mechanism of functional recovery. The challenge is to learn enough about the mechanisms of plasticity in order to manipulate them, suppressing some changes and enhancing others, to gain a clinical benefit and behavioral advantage for a given individual.

In the laboratory Dr. Pascual-Leone combines various brain imaging and neurophysiologic methodologies to establish a causal relationship and a precise chronometry between regional brain activation and behavior. PET or fMRI identify information about brain areas associated with behavior. TMS can transiently deactivate a region of the brain, thus creating a "virtual patient" and explore causal relations. EEG, MEG and ERPs can provide further chronometric information. Repetitive TMS and tDCS allow the non-invasive modulation of activity in a specified cortical target area and its functionally connected cortico-subcortical neural network. MRI and EEG can guide such applications of neuromodulation. Such non-invasive approaches can lead to clinically relevant therapeutic effects in neuropsychiatry and neurorehabilitation, and serve as proof-of-principle prior to more invasive neuromodulatory interventions.

Confirmed Invited Speakers to Date

Donald Cooper, PhD

Donald Cooper received his PhD in Neuroscience from the Chicago Medical School in 2000. Dr. Cooper is the recipient of an NIH career award to investigate gene expression in cocaine addiction. His laboratory is funded by the NIH to study Ecstasy and cocaine in the brain memory and reward system.

The long-term goals of Dr. Cooper's laboratory are to understand information processing in the brain motivation/reward memory circuitry and characterize the adaptations and impaired neural memory mechanisms associated with depression, addiction and schizophrenia.

Dr. Cooper's neurophysiology laboratory combines behavioral, molecular genetic and detailed electrophysiological analysis to understand how psychostimulant drugs alter neuronal impulse activity leading to short and long-term changes in communication within mesolimbic dopamine system. Their approach to this problem utilizes state-of-the-art technology (e.g. DNA microarrays, viral gene transduction, infrared and fluorescence visualized patch-clamp physiology and intravenous drug self-administration) and complementary levels of analysis (e.g. drug self-administration, in vivo and in vitro physiology, molecular techniques and computer simulation) in order to gain insight into how this system functions under normal and pathological conditions.

Dirk DeRidder, MD

Professor Dirk De Ridder is a neurosurgeon, working in Antwerp, Belgium, whose research is focused on the pathophysiology and treatment of phantom perceptions. He developed the technique of electrical auditory cortex stimulation for tinnitus and somatosensory cortex stimulation for pain and recently for auditory hallucinations as well. His expertise extends beyond tinnitus and he is additionally investigating a diversity of clinical populations with a variety of neuroimaging techniques and neuromodulatory interventions.

Hartmut Heinrich, PhD

Hartmut Heinrich received his PhD from the University of Heidelberg. His thesis was "Wavelet analysis methods in a study on attention deficit / hyperactivity disorder". Since 2003 he has been the head of the working group "Neurophysiology in child & adolescent psychiatry" at the Dept. of Child & Adolescent Psychiatry, University of Erlangen and Heckscher-Klinik in Munich, Germany.

Jason Soss, MD

Jason R. Soss is a staff neurologist and Assistant Professor of Neurology at UCLA School of Medicine. He is trained as an epileptologist specializing in dense array monitoring and intracranial recording, and is engaged in a variety of EEG research studies.

Dr. Soss completed a fellowship in Neurology at UCLA School of Medicine in 2002, a residency in Neurology at the University of Michigan Health System, and earned his medical degree from Thomas Jefferson University in Philadelphia. He is a Diplomate of the National Board of Medical Examiners, and Board certified in Clinical Neurophysiology and in Neurology. His research includes localization of seizure onset through nonlinear analysis of scalp EEG and physiologic mechanisms of epilepsy. Past research was funded by the Epilepsy Foundation of America and the National Epifellows Foundation. He is a graduate of Yale University.Clinical Neurophysiology.

Ute Strehl, PhD

Dr. Ute Strehl is an assistant professor and award winning researcher with the renowned neurofeedback research group headed by Niels Birbaumer, Ph.D. at University of Tübingen, Germany. Over the past decade, Dr. Strehl has contributed a series of landmark studies establishing the scientific basis for Slow Cortical Potential (SCP) neurofeedback training in the treatment of epilepsy and attention deficit disorder symptoms.

Dr. Ute Strehl is one of the scientists active in the ongoing research and development of neurobehavioral treatments for seizure disorders in Tübingen. An Assistant Professor on the medical faculty there, she teaches psychology for medical students. In addition to teaching and research, Dr. Strehl is also one of the institute's administrators.

Dr. Strehl brings to her work a background in Behavioral Therapy, Rational Emotive Therapy, and Client Centered Psychology. In 1994 she received the Neuropharmaka Award for Behavioral Research in Parkinson's disease. Her area of research is in developing behavioral treatments for neurological disorders such as Parkinson's and seizures.

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upcoming workshop events*

Jan 21–24 Aug 12-15	**QEEG using Discovery 24E** (19 channels) **, Neuroguide, Live Z-Score** w/Collura, Thatcher, Smith, Mrklas	
Feb 10-14 Sep 9-12	**Getting Started with NeuroFeedback and Database Training Methods** w/John Demos	
Mar 5–7	**Get Started with MINI-Q and Database Guided Training** w/ Richard Soutar	
Apr 15–18 Oct 21-24	**Live Z-Score EEG Training Method** (4-19 channels and above) **and Introducing NeuroGuide** w/Thatcher, Collura, Smith, Mrklas	
May 19–23 Nov 11-15	**Neurofeedback using Quantitative QEEG and Bmans** w/Thomas and Linda Brownback	
Jun 11–13 Dec 3–5	**Using Live Z-Score Method with Neurofeedback (Clinical Perspective-Hands on)** w/Mark Smith/Collura	
workshop events to be scheduled	**Assessing and Training Brodmann Area Functions with SKIL and BrainMaster** w/David A. Kaiser, Ph.D.	
	Slow Cortical and Phenotypes w/Jay Gunkleman	

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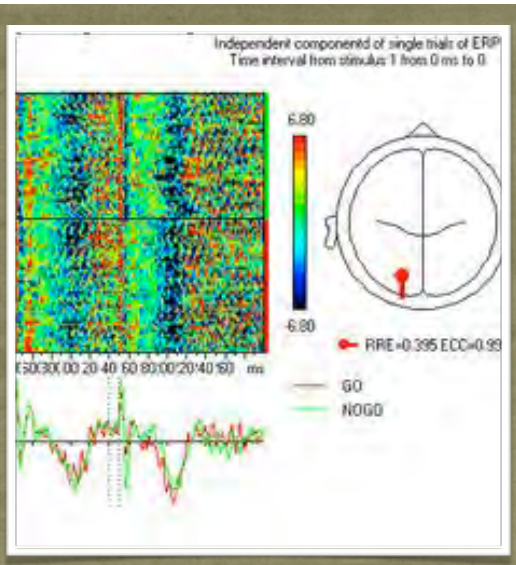

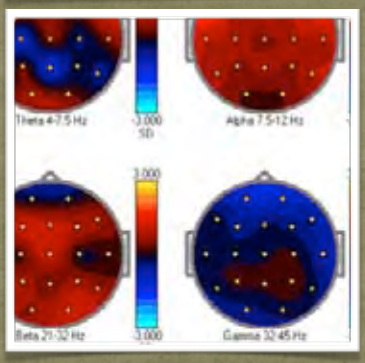

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