NEURO CONNECTIONS Newsletter

Summer 2010

A joint newsletter from the ISNR
& the aapb Neurofeedback Division

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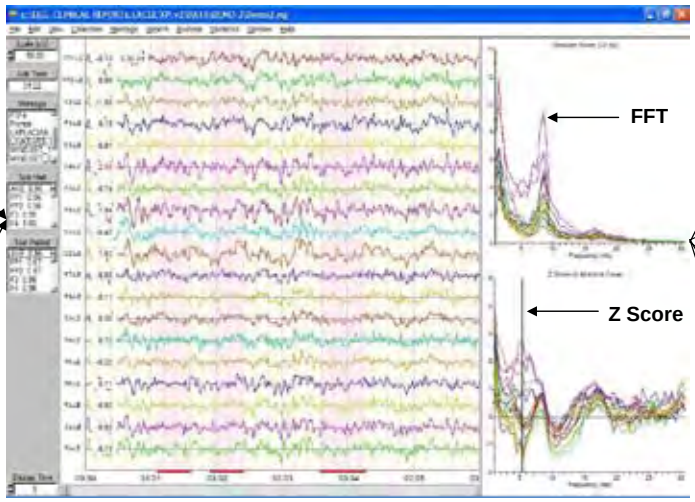

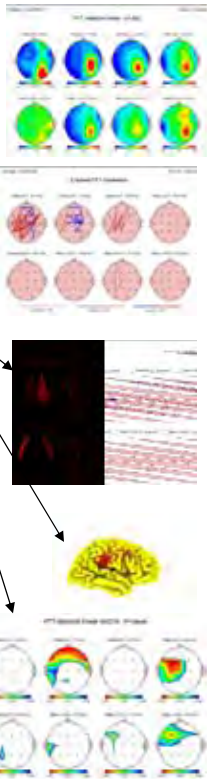

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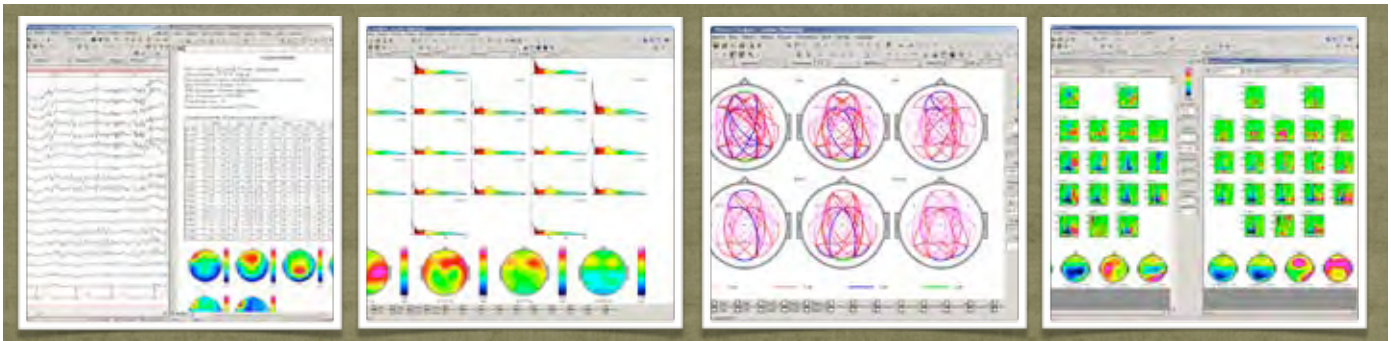

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NeuroConnections
ISNR Co-editor: Merlyn Hurd, PhD
merlynh@aol.com
AAPB Neurofeedback Division Co-editor: Roger H. Riss, PsyD
rriss@madonna.org
Managing Editor: Cynthia Kerson, PhD
office@isnr.org
Journalist for MindFull: David Kaiser, PhD
davidkaiser@yahoo.com
Student Editor: Kimberly Weeks, MS
breetheasy@earthlink.net
Publisher: International Society for Neurofeedback and Research
office@isnr.org
Design: Rosalie Blazej
rblazej@pacbell.net

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

Direct all correspondence and inquiries, including commercial advertising information and classified ads to:

ISNR
1925 Francisco Blvd. E. #12
San Rafael, CA 94901
Phone: (800) 488-3867 Fax: (415) 485-1348
Email: *office@isnr.org*

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

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Letter from ISNR President

Tom Collura, PhD

It has been almost 50 years since it was discovered that EEG states could be internally perceived, and that EEG rhythms could be operantly conditioned. Joe Kamiya made his first report in 1962, and published on the conscious control of brainwaves in 1969 (Kamiya, 1962, 1969). At about the same time, Barry Sterman discovered that operant conditioning of the EEG was effective in suppressing seizures in cats (Sterman and Friar, 1972). Shortly thereafter, work was continued by Lubar and others on humans (Lubar and Shouse, 1977; Lubar, Shabsin, & Natelson, 1981). Sterman's cats were exposed to potentially lethal doses of hydrazine fuel, yet the operantly trained cats withstood and survived these doses. Their survival mechanism demonstrably involved changes in intrinsic regulatory mechanisms, and possibly their immune systems, as well. To me, this are Nobel-worthy discoveries, and it is a mystery to me why this procedure is not now commonplace in the military, clinics, schools, and in homes.

Since these initial discoveries, there has been a surprising dearth of continued rigorous, clinically relevant research on this topic. A search of pubMed in the U.S. reveals no well-funded, randomized, double-blind, placebo-controlled studies of neurofeedback in epilepsy in the past 3 decades. The majority of publications that do exist arise from Eastern Europe, with some exceptions (e.g. Walker, 2010). The most

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Letter from AAPB NFB DIV President

Thoughts are the most real things in the Galaxy.
—Robert Heinlein

When we evaluate EEG activity, we take the wonderful attributes of Psychology, such as attachment, attention, and intelligence, and overlay the immutable laws of Physics (energy, force, statistics) to try to make sense of this information and predict change and states. When brain function is conceived as energy or energy regulation, we can leap to any scale of investigation using the same principles to gain a better grasp of what is going on inside a person's head. Consider the leap I often make, comparing people to galaxies.

Both people and galaxies evolve and mature in predictable ways based on age, size, structure, and distance to others of their kind. Both are open systems and attraction, entropy, and circulation explain about 90% of the behavior of each. Galaxies are mostly gas, dust, and empty space, with the occasional star being formed in their interior -- that sounds about right for people, too. Most of a galaxy's mass cannot ignite and is unseen; likewise much of what steers a person may be felt but not seen, an individual's dark matter. Galaxies exhibit a wide range of shapes, though most are either elliptical or the more familiar and beautiful spiral forms such as our own Milky Way. Elliptical galaxies are smears of light with uncertain regularities, smooth and featureless, whereas spiral galaxies are less modest in exhibiting their distribution of stars. Spi-

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Letter from ISNR Co-Editor

Merlyn Hurd, PhD

Dear All,

Summer has arrived both in reality and in this edition! The theme for this summer edition is Epilepsy and has Sterman, Ochs, and interview of Sterman by Gismondi as the main articles.

The treatment of Epilepsy, as we hopefully all know, gave us the techniques for neurofeedback. Epilepsy is important to me for a very simple reason. I was diagnosed with it in my thirties and the fact that the diagnosis was only provided after one scary day points to the difficulty surrounding this disorder. The scary day started off with not feeling "right" and progressed to "I need to get to a doctor." My husband had admonished me to just rest as he went to a rehearsal of a play he was in. Sometime after lunch I got myself down 4 flights of stairs, into a taxi and went to we women's major doctor, a gynecologist. Because I was going in and out of consciousness the cab driver held my hand all the way to the doctor's office and even carried me into his office. My gyn promptly and correctly said he could not treat me and called his major doctor, Dr. William Hitzig (of Hiroshima Maidens fame), who sent his Rolls Royce to pick me up and take me to his office (small hospital really). I was clearly in status epilepticus. After eight hours of trying to stabilize me, I was transferred to Mt Sinai Hospital and for four days underwent every type of test, including hypnosis to find out what was causing the seizures. Please believe me hot isotopes injected into your neck do make you think your brain is being cooked.

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.

Finally Dr. Bender (of brain surgery textbook fame) came in, said stick out your left foot and rolled a tiny disk up it. The Babinski response was immediate. He pointed to that response and said, "This is the only fact, after all the tests, that tells me you have a brain injury and that it is probably the seat of the seizures." Why tell this story? Because it underlines the difficulty of diagnosing seizures and the consequences when the diagnosis has not been made. Had there been incidents before this? Yes, but not recognized. After all, girls do faint, we all know, in various situations. After dental work; in a too hot classroom; at an exciting football game; after minor surgery; taking care of a newborn and two toddlers (just overtired, right?) and on and on.

The estimate is that there are 45 million people worldwide who experience epilepsy and in only 30% will the causal factors be found. Thus 70% will be treated and the causes of the seizures will not be known even with tests, keeping logs and/or monitoring by spouse, family members and doctors. The medical community has a rule of thumb; if a seizure happens only once, no treatment is recommended and the incident is not considered a seizure. For there to be a diagnosis of epilepsy, there has to have been more than one incident and certain criteria for the particular type of seizure must be met. On the one hand these rules of thumb prevent overmedication. On the other hand, since there is such a large percentage for which the cause cannot be found, over-

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Letter from AAPB Co-Editor

Roger Riss, PhD

Welcome to the summer 2010 issue of NeuroConnections. This issue focuses on the role of neurotherapy in the treatment of epilepsy. Since publication four decades ago of Sterman's first seminal studies in this area, additional millions of dollars have been expended by "big pharma" on the development and marketing of new antiepileptic drugs (AEDs). In spite of these efforts, an estimated 25% of children continue to receive inadequate seizure control with conventional pharmacological treatments, an outcome which can have profound impact on their subsequent academic development. Moreover, among "medication responders," parents are understandably concerned that seizure control may come at the expense of unwanted medication side effects, impacting mood or cognitive alertness. Interest in evidence-based non-pharmacological treatments for seizure management may be particularly high for parents whose children have developed toxicity on AED medications.

Non-pharmacological treatments which have received initial research support include avoidance of seizure triggers, relaxation techniques, and educational interventions focusing on lifestyle management such as diet, sleep and recognition of seizure "warning signs." In contrast to the limited number of studies devoted to these topics, research support for neurofeedback-based interventions now spans four decades and several continents. A Medline search of the terms *neurofeedback* and *epilepsy* currently yields 135 peer-refereed citations. Moreover, clinicians have a choice of at least three distinct approaches supported by multiple studies and clear theoretical models: 1) Sterman's classical SMR protocol focusing on inhibitory training of thalamocortical loops over motor cortex. 2) slow-cortical potential training, as investigated by several prominent European research groups and 3) individualized QEEG-based protocol development, as exemplified by Walker's recent papers.

An emerging area of science such as neurofeedback necessarily involves a dynamic interplay between evidence-based practice and active stretching of the boundaries of established medicine. A recent Cochrane Review of evidence-based practice in epilepsy treatment favorably noted the current body of neurofeedback interventional research, but nonetheless called for additional large-N randomized controlled studies - perhaps this is a task that will be taken up in the next decade, and by the next generation of neurofeedback researchers?

Roger Riss, PhD
AAPB Co-Editor

Letter from ISNR ED

Cynthia Kerson, PhD

This spring promises. It has been a very productive year to date for ISNR and the Research Foundation. I hope you read your monthly emails as they offer up our latest goings-on. Please feel free to contact me with any interest or questions you have at office@isnr.org.

These past few months I embarked on my own personal journey which included neurofeedback training, as guided by my dear friend and ISNR Board member (who asked to be anonymous). I did 12 sessions of neurofeedback, augmented it with AVE (utilizing the devices by two awesome ISNR members) and meditation (so inspired by yet another remarkable ISNR member) and have found really great results. One should never believe that the journey is complete. Clinicians owe it to themselves and their clients/patients to take care of themselves. We have a responsibility to be proactive in managing our and their emotional and physiological wellbeing.

When the stewardess recites her spiel she suggests that should there be an emergency the adult take action and then assist the child. I think this is good advice; clinicians should take care of themselves and then respond to those who seek their help. This may be especially true in our field for which there is such a critical eye.

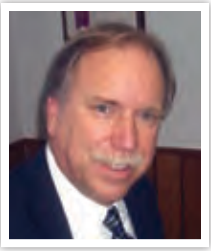
I contribute this not because I need your sympathy or interest in my personal journey, but because I want to share that what clinicians do and how they present themselves has impact. Clinicians should change the lives of those who seek their services. Even those who are functioning well (OK, so some of you may argue that in this case) may at times need to peel back yet another onion layer to be of best service.

Thank you to my ISNR friends,

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

Letter from AAPB ED

A Changing of the Guard



David L. Stumph

Each year, new representatives are elected to the AAPB Board of Directors. And each year, there is a new set of priorities, directions, and fresh ideas to address member needs and offer exciting new approaches to achieve growth. This year is no exception. New AAPB President Carmen Russoniello has set a series of objectives for this year that include:

- A focus on membership growth
- Membership growth with an emphasis on student members and those new to the field
- Reaching out to associations representing different fields such as medicine, physical therapy, and others who would benefit from knowing more about biofeedback and neurofeedback

AAPB's Membership Committee has been charged with developing a comprehensive membership campaign that includes a challenge to each member to recruit at least one new member. In addition, a special focus of the campaign will target universities for both faculty and student members addressing the goal related to increasing the need to attract a younger group into our ranks.

In addressing the need to reach out of non-biofeedback related organizations, each member of the Board has been challenged to find a way to get onto educational programs directed to professionals from such organizations. This is something that each AAPB, ISNR, and BCIA member/certificant can do as well. Collectively, we all need to be thinking of ways to get the message out about the benefits offered by biofeedback and neurofeedback in healthy living and peak performance.

We tend to "preach to the choir." And while it is important to keep our community up to the moment on new advances, we also need to be on the lookout for ways to spread the word beyond our own small community. As a famous Chicago architect, Daniel Burnham, once said, "make no little plans: they have no magic to stir men's blood. Make big plans… aim high in hope and work." Biofeedback has made great progress in recent years. But in the scheme of things, it is still small potatoes. Since Mr. Burnham made his remark back in the late 1800s as Chicago was coming into its own, the city has grown to become one of the largest and most vital cities in the United States.

So, as our organizations all make changes in leadership and focus from year to year, let's always stay focused on our real challenge at hand—educating others about why biofeedback and neurofeedback need to be considered in everyone's daily lives. While we experience a changing of the guard, we need to also change our collective mindset to one that thinks well beyond our own small community. Let's make big plans and aim high to make biofeedback and neurofeedback household words. To make that happen, we need each of you involved in reaching out and spreading the news.

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

ISNR President

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notable research activity has been in connection with Slow Cortical Potential training, and has been conducted on the other side of the Atlantic (e.g. Fritz, 2010). More recently, the European groups have begun to look at alpha and SMR training, thus coming full circle to the work at UCLA in the 60s and 70s.

Why the hesitance to accept an innovative, relatively harmless, and clearly cost-effective intervention? On a positive note, Barry Sterman was invited to speak at the Cleveland Clinic / Bakken Heart/Brain Summit last year on the topic of EEG biofeedback for epilepsy. However, the Clinic and the rest of the medical world have yet to take this modality seriously, a full 18 years after this author first proposed it to the epilepsy department. This year, at the major high-profile biofeedback conferences (AAPB 2010, BFE 2010, Cleveland Clinic 2010), the emphasis remains on peripheral, heart, and breathing-related biofeedback. While these are without dispute a critical underpinning of the field of self-regulation, stress-reduction, and symptomatic relief, they do not hold the dramatic potential to reduce the use of anticonvulsants and related medications, thereby avoiding negative side-effects and a litany of drug-induced disorders.

Many of us have had recent direct experience with the power of neurofeedback in dramatic circumstances. 18 years ago, we successfully remediated our very first client, a 15-year-old with ADHD using neurofeedback, and he continues to do well without medications to this day (Collura, 1995). Four years ago, a three-year-old boy with chronic seizures was remediated using an innovative form of brain connectivity training (Smith, 2008). This boy has been seizure-free for four years, and is now looking at seriously reducing, and hopefully terminating, his anticonvulsant medications. Neurofeedback and conventional interventions can work together to maximize well-being and personal autonomy, while allowing nature to take its course.

More recently, a prominent local psychologist sent a family member to our small clinic to see if neurofeedback could help him get on track. He had had an unfortunate experience with an SSRI, causing him to have a breakdown of sorts. He had to discontinue his schooling and take time off to recuperate. We performed an EEG and with Jay Gunkelman's help recognized a phenotype (spindling frontal beta) that suggested non-response to SSRI's, with a possibility of side-effects such as those experienced. If an EEG had been taken initially, this episode could have been avoided. Neurofeedback helped him get back on track, and resume his path in life. Despite this success, we do not yet have crowds at the door. It is as if the result is too spooky, too unsettling, to be acceptable to the zeitgeist. At least for the time being.

I am left with the feeling of someone who has seen something that undoubtedly works and holds great promise for the future, if only the rest of the world would sit up and take notice. It is a fact that the Wright brothers flew their flyer around the fields of Ohio for several years before the US War Department recognized that powered flight was possible. Local newspapers contained reports of their increasingly long flights for years, and these chronicle their developments that were underground until the time was ripe for the world to accept their achievements. In the case of neurofeedback, it appears that we will have had to carry on years of clinical and experimental work before this intervention finds its place in the main lines of therapeutic use.

Hsu, Chen, Hsu, and Beggs (2008) propose that epilepsy Is a learned behavior and that it can be unlearned. To those of us in neurofeedback, and especially with clinical experience with seizures, this is a given. Where in the chain of causality does the brain end and the mind begin? We know that the brain has developed and controls its own self-regulatory systems, and that the full range of excitatory and inhibitory activity is subject to autoregulation, hence operant change.

In an imaginary retrospect, I picture what it would be like if children routinely received QEEGs, were assessed for possible disregulation, and treated with a combination of biofeedback and EEG operant training. This would optimize their chances for development and self-actualization, and restore a freedom that is lost in the process of diagnosis and medication. By looking back and asking, "why was it not this way," we can hope to set a path for the future, so that we can look back in another 30 years and say, "We know why it is better now, and we helped make it real."

In recent talks, Joe Kamiya talks about a new age of psychology in which our vocabulary is enriched by correlating internal subjective states with EEG parameters. He imagines a time when human evolution has taken another step, in which inner space becomes as familiar as outer space. He talks about creating coordinate systems that map our internal lives with the precision of a modern GPS, and help to move human consciousness toward further fulfillment and actualization. This vision, which he has been sharing for years, surely points the way toward a more sane, humane, and effective science of the brain, mind, and society.

We have learned that approaching epilepsy with EEG biofeedback can be scientific, rigorous, and effective. Notable among the developments in the last 30 years is the use of QEEG for the assessment and planning of interventions. QEEG provides an objective, meaningful assessment of brain function, and has been found relevant to neurofeedback planning and treatment over many years of work (see Collura, 2009 and entire issue of Appl. Psych. & Biof. V. 35). Most of this work has been driven by clinicians doing research in the course of their practices. If the money that has been spent on one functional MRI machine were to be directed toward funding formal research in QEEG and neurofeedback in epilepsy, we would have a much greater understanding of the true possibilities in this area.

Epilepsy is but one of the manifestations of disregulation, whether its origin is genetic, chemical, environmental, even learned. Due to its severity and life-threatening nature, it stands as a priority for clinical and experimental research. It is a disservice to the community that this work has not proceeded as far as it could have. Those of us who know that neurofeedback works have an implied duty to share its benefits with our fellows, even if they don't know that it is there for them. To those of you who resonate with this principle, consider this a call to arms, to support research and clinical work in self-regulation and epilepsy, and to help set a course for a more self-empowered and seizure-free future. Those with interest in supporting and/or participating research in neurofeedback are invited to contact the ISNR to see how you can make a contribution through our joined efforts.

Tom Collura, PhD
ISNR President

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ISNR CO-Editor

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medication can take place. So what is the definition of epilepsy? The U.S. National Library of Medicine states: **"**Epilepsy is a brain disorder involving repeated, spontaneous seizures of any type. Seizures ("fits," convulsions) are episodes of disturbed brain function that cause changes in attention or behavior. They are caused by abnormally excited electrical signals in the brain."

The number of diagnostic codes in ICD that are listed, so far, are 19 with migraines having several more. When one looks at the range from headaches to full blown epilepsy, there is an understanding that there is a range of electrical disturbances that fall between the diagnosis of headaches to intractable epilepsy. Headaches being the "benign" type.

What do Neurofeedback and QEEG contribute to the identification and treatment of this disorder?

QEEGs help us to ascertain the areas of concern. In the raw EEG, the spike and wave pattern is distinct and the episodic morphology is usually clear. With QEEG, we are in a unique position to not only pinpoint the activity but to then design neurofeedback protocols for the areas of concern.

Other medical tests that are often run are:

Various blood tests and other tests to rule out temporary and reversible causes of seizures, such as:

- Blood chemistry
- Blood sugar
- CBC (complete blood count)
- CSF (cerebrospinal fluid) analysis
- Kidney function tests
- Liver function tests
- Tests for infectious diseases

Tests for the cause and location of the problem may include:

- Medical EEG
- Head CT or MRI scan
- Lumbar puncture (spinal tap)

Most of my clients have had all or a high percentage of these tests, but not QEEGs. I have had more than one neurologist state they are using my QEEG analysis of a client to understand and

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monitor the progress even with drugs for the client. In my opinion a QEEG needs to be conducted for every client who presents with either confirmed or uncertain epilepsy, Several years ago a client, who was sent to me by a neurologist,stated "I don't know if I have seizures. I just sometimes wake up on the floor of a store and people say I had a seizure." Psychologically we can make a lot of conjectures regarding her statement. At the same time her statement points up to the fear that surrounds the diagnosis of epilepsy. When I was the Director of Health, Nutrition and Mental Health for NYC HeadStart, one of my tasks was to assess children with epilepsy in which the parents and staff were adamant was not true. Why did they fight it and deny treatment to the children? Because it was believed that when this child grew up, he/she would not be marriageable. Fear and shame still lurks around this diagnosis so if you have a client or a child or parent with it, be sure to take a very extensive client and family history to be able to help him or her address it appropriately.

Neurofeedback studies continue to show the reduction, and often elimination, of seizures. This alone is a welcomed relief to patients and neurologists. Still, neurofeedback is not listed as a preferred treatment in any of the medical treatments descriptions. The treatments recommended are medication and surgery. If you have ever had a client who still has seizures, despite surgery, you know the anger and fear that they are experiencing. I have treated clients who have had surgery and also a client who had 40 seizures per hour. In my experience the surgery clients had some relief from neurofeedback while the client with 40 seizures per hour is still seizure free threeyears after neurofeedback treatment.

Regarding elimination of seizures and medication, I can talk from my own experience. Since I had taught myself self-hypnosis due to allergic reactions from pain killers, I used the same techniques to learn to control the seizures. After two years of taking Phenobarbital, I became seizure free and then medication free six months later. One word of caution, seizure activity may still take place if the conditions are "right." Some studies have found that a high percentage of patients are seizure free because they learn the conditions that may induce a seizure and they learn the techniques for controlling them. In my own case, when I get those signals, I know to breathe deeply, stop what I am doing and go into a controlled calm state. You can laugh, but I also talk to my brain and soothe it.

Neurofeedback studies continue to show the reduction, and often elimination, of seizures.

This letter has been a little longer than usual because this disorder is one that neurofeedback and QEEG can be of help to so many people and I hope this letter/article has been informative and useful.

Have a great summer. See you at ISNR in Denver in the fall!

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

AAPB NFB Div President
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rals sometimes possess arms and shoulders (material connecting the center bulge to each arm) and sometimes they do not, and the angle at which we encounter a spiral can affect our ability to recognize it as such, much like the angle by which we meet some people can hide their general nature. The classification of galaxies evolved to include "social" forces, as it was recognized how some galaxies gravitationally influence the structure of others. Tidal forces, as they are called, can form rings in some galaxies, cloud the interior of others, or diminish the halo of stars orbiting the center. People are also the product of tidal forces.

Speaking of tidal forces, are all disciplines becoming evidence-based at once, due to some massive pressure from without? Is poetry now evidence-based? I ask this question because the other day I encountered "evidence-based spirituality," a concept both surreal and sublime (see isseeem. org), and although it does make sense, what makes more sense in my opinion is evidence-based neurotherapy. Evidence-based neurotherapy fuses the practices of mental health, medicine, and applied sciences into an ethical, effective, and hopefully more commercially-viable amalgam than our current aggregation of stars, gas, and dust. Now that Congress has passed the resounding health care bill, whatever it may eventually say, we can assume further centralization of care will favor (and reimburse) certain clinical practices over others, even more than they have in the past, and we need to position our field to be among the winners of this increasingly science-based competition. To achieve this we have to develop our own evidence-based handbook for neurotherapeutic practices, one that assesses the strengths and weaknesses of each approach empirically and for each symptom, and while we are doing this, we need our own "DSM," one based in physiology and neurobehavior, to compete and overcome the limitations of the psychobehavioral classification scheme of the current DSM (Diagnostic and Statistical Manual). "The Assessment and Care of Galaxies" (ACG) might be my title of choice, tongue-in-cheek, but who in Washington would understand such a leap..

David Kaiser, PhD

Neurofeedback in the Treatment of Epilepsy: Research History and Present Clinical Perspective

M. Barry Sterman,

To follow is an excerpt of an article in press that introduces foundational ideas of QEEG and SMR training for Epilepsy. For some of you, this may seem rudimentary, for others a good foundational understanding. Providing this excerpt to NeuroConnections readers allows us to see what is being accepted by the outside communities as an introduction to our field. (CRK, Ed)

Introduction

As a young neuro-behavioral researcher my doctoral thesis project was focused on the brain's mechanism for the voluntary initiation of sleep. Following up on the classical conditioning studies of Ivan Pavlov which had identified a state he termed as "internal inhibition," we were able to document the existence of a forebrain system that, when stimulated electrically, was capable of initiating an EEG and behavioral sleep-onset pattern in cats (Sterman & Clemente, 1962). Pairing this stimulation with a preceding auditory tone resulted in a learned sleep-onset response through classical conditioning (Wyrwicka, et al 1962). Other studies at the time focused on brain mechanisms mediating learned behavioral inhibition, and had revealed a uniquely correlated 12-15 Hz EEG rhythm localized to sensorimotor cortex. We labeled this as the sensorimotor rhythm, or SMR. The similarity of the SMR to the known EEG "spindle" pattern during quiet sleep, both of which were associated with a state of motor quiescence, led to the novel idea of attempting to increase the SMR using EEG operant conditioning, with the goal of determining if this might produce a corresponding increase in sleep spindle activity thus establishing a common underlying mechanism for this state (Sterman, et al, 1970). Results supported this hypothesis but led accidentally to the discovery of an anticonvulsant effect as well (Sterman & LoPresti, 1969). Continuing animal studies identified a physiological pattern of responses correlated with the SMR in primary motor pathways. Additionally, bouts of oscillating cellular

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Epilepsy

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discharge in the afferent somatosensory thalamic nuclei were found to mediate the cortical SMR pattern. All of these findings were indicative of reduced motor excitability. The associated elevation of convulsive thresholds and protection from exposure to a toxic chemical suggested that we examine this method for the treatment of human seizure disorders.

Our first study in this regard involved a 23 year-old female epileptic with 3-6 monthly primary-generalized nocturnal tonic-clonic (gran mal) seizures that were not controlled by anticonvulsant medications. This patient ceased having seizures almost immediately after we began the EEG operant conditioning training, which consisted of 3 hourly sessions per week. After 3 months seizure free we decided to submit a paper to the journal "EEG and Clinical Neurophysiology" describing these results. This paper was rapidly accepted and published within months of submission in 1972 (Sterman & Friar, 1972).

Clinical Methods & Findings

Our initial clinical report was followed by a series of human studies involving both crossover and placebo controlled group designs. Significant seizure reductions in epileptic patients were documented in response to reward for increasing mid central cortex 12-15 Hz sensorimotor EEG rhythmic activity. Two independent meta analyses of the peer-reviewed, published papers in this literature have appeared in the last decade. Sterman (2000) reviewed 24 studies involving 243 mostly partial complex poorly controlled seizure patients provided with central cortical SMR feedback training, and found that 82% of these subjects registered seizure reductions greater than 50%, while Tan et al.(2009) evaluated data from 63 studies and selected 10 for comprehensive evaluation that met stringent criteria for controls, population, and seizure details. They report that 79% of the patients treated with SMR feedback training experienced a statistically significant reduction in seizure frequency, despite a collective history of failed medication therapy. In two studies from my laboratory extended design and a relatively long-term follow-up both showed that EEG and clinical benefits were sustained over time (Figure 1). Further, pre-post neuropsychological testing showed that responding SMR trained subjects also improved significantly in performance of tasks specific to the hemisphere contralateral to their fronto-temporal lesion, indicating a reduced corrosive disturbance from the seizure focus (Lantz, Sterman, 1988; & Sterman & Lantz, 2001).

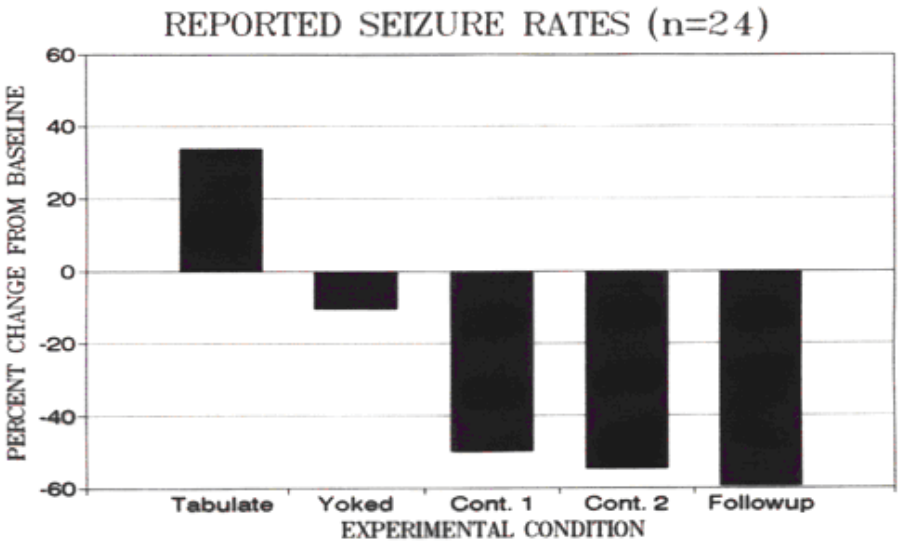

Figure 1. Plot of reported seizure rates in three experimental groups of eight randomly assigned complex-partial epileptic subjects with medication-refractory seizures. The groups received one of the following treatments each for six weeks, including 1) detailed tabulation of seizures, 2) non-contingent SMR ("yoked") training , and 3) contingent SMR training. Following this initial period all 24 subjects were combined into one SMR contingent training group for 6 additional weeks, and then gradually withdrawn from training. A final six week follow-up seizure tabulation period completed this analysis. Data are plotted against group baselines. A significant reduction in seizures was registered after contingent training only, and this effect increased progressively across subsequent conditions. (Derived from 8).

EEG operant conditioning methods for EEG biofeedback training have diversified as differing hardware and software products have emerged, and as individuals with differing backgrounds and credentials have entered the field. A lack of methodological standards and professional regulations has contributed to an undesirable inconsistency in the competence and effectiveness of therapeutic applications. Nevertheless abundant peer-reviewed research conducted by qualified individuals has proven the worth of this method as a viable alternative treatment for seizure disorders. Accordingly, an attempt will be made here to provide some idea of the systematic, evidence-guided approach to treatment used in the author's program.

In our practice patients are subjected to a requisite quantitative multi-channel EEG evaluation (QEEG), using hardware and software complying with both technical and learning theory principles. These principles, which involve hardware specifications, timing, and acquisition criteria, will not be discussed here but are critical to valid and reliable data collection and effective operant conditioning applications. Data obtained from this study are combined with medical reports and information gained in a comprehensive intake interview. The QEEG and background information guide the design of an empirical protocol, often with several training components, that will be used consistently throughout the treatment period, in our case consisting of one or two 60-90 minute treatment sessions per week for at least 12 weeks. Subjects are seated in front of a large monitor screen and are instructed the requirements for reward. Reinforcement consists of visual images and tones, as well as a numeric display of scores achieved and the time remaining in a trial. On rare occasion a committed parent may be seated next to a more challenged patient and provide additional reinforcement in the form of earned treats, such as raisins and small candies.

The display that subjects see can vary within limits but must always be as simple as possible and must provide information exclusively relevant to achieving the desired EEG changes. One such display is shown in Figure 2. It consists of a series of 4 rectangular boxes, each with a segment of band-passed EEG data for selected frequency bands, enclosed by reward threshold limits set to provide normalizing guidelines. If the objective is to increase the amplitude and/

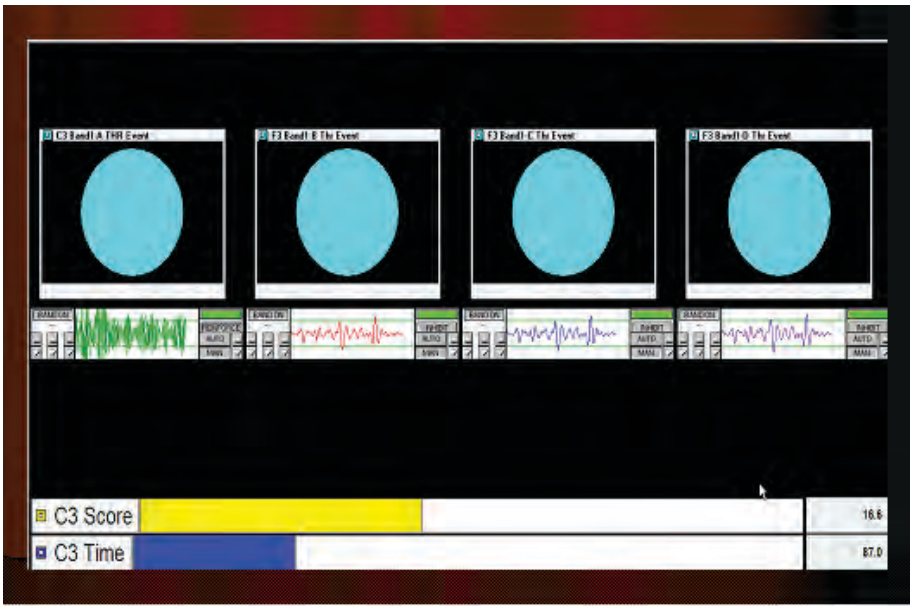

Figure 2. This primary display used in our SMR biofeedback program conforms strictly to operant conditioning principles, while still promoting cognitive engagement in the human subject. Reward here is for 2 different EEG frequencies at 2 different cortical sites. The Far left "green" site shows reinforced 12-15 Hz band-pass activity at C3. Progressively more difficult suppression of abnormal 3-5 Hz slow activity at Fz is addressed here through "successive approximation", and consumes the final 3 display units from left to right. See text for more details.

or incidence of a particular frequency band the band-pass display must exceed the upper threshold guideline. If it is to suppress that frequency band it must drop below the threshold line. The duration of the required response can be adjusted and is typically a quarter to one-half of a second. When the desired response is achieved a small horizontal bar at the upper right of each band-pass display turns from red to green, and a large blue ball appears above the band-pass, together with a chime or other tone. The display is frozen for 2 seconds and then becomes active again, thus providing for discrete trials. A yellow score bar at the bottom of the screen advances one unit. The timing of each performance set (typically 3 min.) is indicated by a moving blue bar at the bottom of the screen.

For facilitation or suppression of a relevant EEG pattern, display boxes can be arranged in a horizontal sequence, with each box monitoring the same electrode site and frequency. Sequential thresholds can then be set to promote or inhibit progressive increase or decrease from left to right, thereby guiding the desired response through a process of "successive approximation." Numerous other configurations are possible. In the case shown in Figure 2 the band-pass at far left is set at 12-15 Hz (SMR) for the C3 electrode site, and the remaining three bands to the right are all set to 3-5 Hz at left medial frontal location Fz, with successively more difficult thresholds in order to guide and promote suppression of this band at this site.

Performance outcome can be measured systematically by tracking scoring rate (sequential number of rewards) per trial across a series of trials, together with corresponding EEG data. Findings from the case described above provide an example. To the top of Figure 3 is a plot of reward rate (number of criterion responses) across 4 successive three-minute EEG feedback trials. The patient had been rewarded for simultaneously increasing 12-15 Hz SMR activity at C3 and reducing 3-5 Hz activity at Fz, as described above. However, the data presented here show a subsequent training session where reward for SMR activity at C3 was combined with a new pattern of sites in frontal cortex where simultaneous suppression of slow 3-5 Hz activity was required. These sites included F7, F3, and the previously trained Fz locations. The smoothed EEG plots associated with this training are shown below the reward-rate curves, starting with the C3 12-15 Hz channel. Activity in this band became increasingly less variable across trials. Data from the 3 frontal recording sites, however, all show a progressive decrease in amplitude across the training period, with the greatest suppression occurring at the previously targeted Fz site. Thus, the stabilization of SMR activity and simultaneous generalized suppression of frontal slow activity result-

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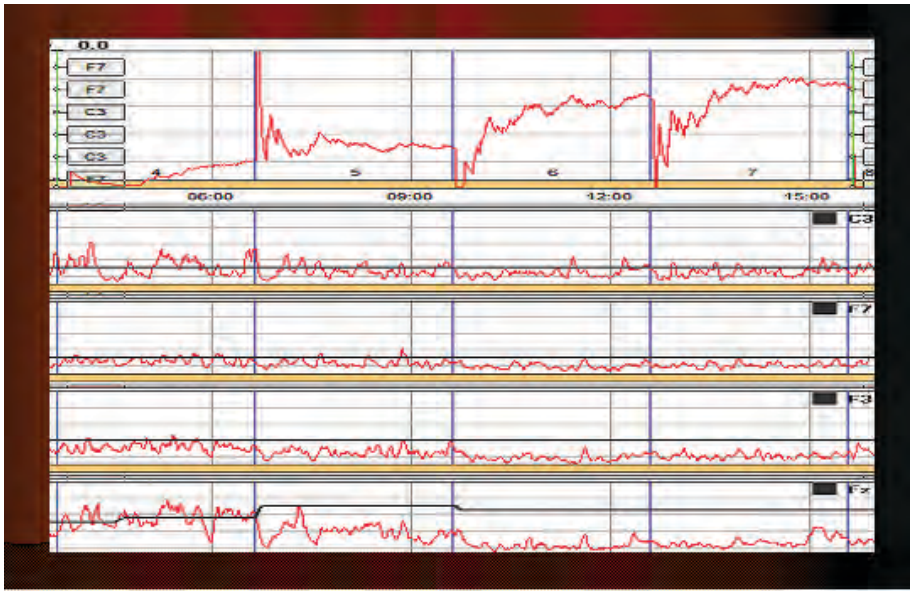

Figure 3. This performance plot from the patient and feedback display shown above registers scoring rate per 3 minute trial at top, and corresponding smoothed amplitude output in the band-passed frequencies set for each electrode placement. In this case the patient was rewarded for increasing 12-15 Hz SMR activity (top EEG trace), and decreasing 3-5 Hz slow activity at Fz (bottom EEG trace) The subject showed response acquisition both within and across 3 min. feedback trials, together with a stabilization of the SMR frequency and a reduction in frontal slow activity.

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ed in a progressive pattern of incrementally increased rewards, both within trials and across the session. Such a pattern is clearly indicative of learning.

We feel that it is important to at least have a rational model that explains the physiological "how" of EEG and behavioral outcomes with neurofeedback training. A pattern of highly relevant physiological changes was documented as a basis for the SMR rhythm in animal research (Sterman, 1996)). While it is difficult to evaluate neurophysiological changes in human subjects similar to what was accomplished with animals, it is likely that the responses observed are common to all higher mammals. New imaging methods allow for assessment of localized metabolic changes in the human brain during and after EEG feedback training. Several recent studies have suggested that the specific pattern of motor inhibition documented as underlying the generation of the SMR is directed by output from the striatum of the basal ganglia. Niels Birbaumer observed increased striatal metabolic activity with fMRI analysis in subjects producing SMR activity (see the article by Wyckoff and Strehl on 13 page, this issue, for more information on Dr. Birbaumer's work with SCP training for Epilepsy). Further, Lévesque and colleagues (2006) studied pre-post-fMRI blood oxygenation level dependent (BOLD) response patterns in learning-disabled children trained to increase SMR activity and found a specific increase in the metabolic activity of the striatum and substantia nigra. The SMR trained subjects showed significant academic improvement as well. If activation of these inhibitory basal ganglia networks can become labeled by the SMR through contingent feedback training, and responsable circuits potentiated by this association, motor inhibitory regulation would be generally facilitated.

Conclusions

Alternative explanations for therapeutic results include such considerations as short-lasting expectation effects, and changes in patient behavior. However, we must once again point out that the prolonged seizure suppression effect documented in our animal studies, and in relation to nocturnal seizures arising out of the uncoscious state of sleep in human subjects, would seem to rule out placebo or non-specific effects. This conclusion is supported further by the finding of improved neurosychological performance after SMR training in tasks mediated by the hemisphere contralateral to disrupting localized epileptogenic lesions. Additionally, an alternative explanation for improved seizure control based on increased medication compliance has been rejected through studies that carefully monitored blood levels of prescribed anticonvulsant drugs before, during, and after training. Finally, it must be pointed out that the epileptic patients who have demonstrated clinical improvement in neurofeedback research studies, and many who have sought this treatment clinically represent unquestionable failures of current anticonvulsant drug therapies. It is particularly noteworthy that positive outcomes have mainly been obtained treating complex-partial seizure disorders, an extremely difficult sub-population of epilepsy patients.

One has only to study epileptic patients over time to appreciate the physiological, cognitive, and social consquences of prolonged anticonvulsant medication use.

Looking back over the years since our surprising positive results with Mary, the young lady who was our first human epileptic subject, I can only wonder how many other epileptic patients like her might have benefited from a chance to try SMR neurofeedback. I will never forget the day she walked into my office, after reducing her medications, with an amazed expression on her face. When I asked what had happened she said she was walking down the hall and realized that she could hear her own footsteps for the first time in years! One has only to study epileptic patients over time to appreciate the physiological, cognitive, and social consquences of prolonged anticonvulsant medication use. Yet despite the fact that most of the patients treated with neurofeedback have been refractory to these medications, virtually all are helped to a greater or lessor extent by neurofeedback. Notwithstanding this reality, neurofeedback is generally not endorsed by medical professionals or epileptic support organizations.

In all fairness, however, there are perhaps some good reasons for this. Certainly, epilepsy is a complex disorder with many differing origins, expressions, and degrees of severity. Neurologists know this, and are put off by individuals using unexplained therapies and making undocumented and unrealistic claims. I share this concern. Despite the encouraging findings and concepts reviewed here it must be remembered that there are significant issues at virtually every step of the thinking about seizure disorders and the practice of neurofeedback. Unfortunately, while there is a standardized medical classification of seizures and approach to the use of medications (effective or not), the practice of neurofeedback has yet to standardize pre-treatment assessment or treatment methods and outcome evaluations. It is for this reason that we have sought to develop consistent and reliable criteria for guidance through quantitative EEG, and a system of standardized protocol applications for treatment based on learning principles. We hope in this way to maximize both treatment transparency and efficacy.

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The Disease Once Sacred: A Brief History of Epilepsy

David A Kaiser MFA, PhD

All who drink of this remedy recover in a short time, except those whom it does not help, who all die. Therefore, it is obvious that it fails only in incurable cases
—Galen (2nd century AD)

When King Charles II of England (1630-1684/85) suffered convulsions during his final days, he underwent the best treatments Europe had to offer a monarch: the letting of 24 oz of blood from his arm and shoulder, purgatives to induce vomiting, blistering of the scalp, sneezing powder to relieve the pressure of humors on his brain, cowslip flower powders to strengthen the brain, pigeon dung plastered to the soles of his feet to calm him, an enema made of rock salt and flowers, drinks of barley water, white wine, and absinthe—finally something helpful… along with various medicaments like melon seeds, dissolved pearls, nutmeg, cloves, human skull extracts, powdered stone, and cannonball sweat. Okay, the last was added by me, but this list amounted to the best treatments 17th century medicine had available at its disposal. That the man who restored the English monarchy in his youth was medically tortured the last few days of his life did not escape the notice of dignitaries present during the procedure, including Sir Charles Scarborough, one of a dozen physicians attending the dying king who recorded this list of "treatments" for us. (Crawford, 1909).

Treatments for epilepsy have improved since the days of Charles II and ironically he may have actually suffered from a kidney ailment, as some scholars believe, and it was this ailment that caused seizures. If so, drinking flowers and bone probably didn't help his condition. In other words, if people suffered a serious ailment in the 17th century, they could only hope that no doctors were available for their care.

There are few things associated with EEG as much as epilepsy, a term which encompasses a range of brain breakdowns that manifest by loss or change of consciousness with or without convulsions. We've known about epilepsy since we've known about ourselves; seizures are described in the earliest medical writings we have. In fact few conditions have attracted so much attention and generated so much controversy as epilepsy. The maladies of epilepsy and aphasia were the dysfunctional engine that propelled behavioral and clinical sciences into the modern era.

People have written about seizure disorder and its mysteries for nearly 4,000 years, starting with Assyrian, Akkadian, and Babylonian accounts (Stol, 1993; Eadie, 1995; Magiorkinis et al., 2010). The Code of Hammurabi of the 2nd millenia BC even mentions epilepsy in slave sale contracts, a buyer-beware notice (Stol, 1993). The Babylonians believed all diseases had supernatural causes, as did the Sumerians, whose name for epilepsy was "what has fallen from heaven," a word similar to their word for meteorite. They also called it the "Hand of God" with the god being given an angry adjective such as debilitating, fierce, devouring, or raging. An epilepsy sufferer was said to be struck by the "Hand of the Fierce God," for instance. Plato added his two cents on the etiology of seizures in *Timaeus*, considering its cause to be a mixture of phlegm and black bile which confused the divine circles of the head—not bad for the 4th century BC. If he had predicted distortion of divine circles of electromagnetism, he could be describing modern-day identification of nonconvulsant seizures with EEG. Aristotle, on the other hand, believed that food in the stomach produced vapors that rose to the head in the veins to cause seizures, relating the body to brain as he tended to do (Eadie & Bladin, 2001).

I am about to discuss the disease called "sacred." It is not, in my opinion, any more divine or more sacred than any other diseases, but has a natural cause Its origin, like that of other diseases, lies in heredity The fact is that the cause of this affection is the brain. My own view is that those who first attributed a sacred character to this malady were like the magicians, purifiers, charlatans, and quacks of our own day. Being at a loss, and having no treatment which would help, they concealed and sheltered themselves behind superstition, and called the disease sacred, in order that their utter ignorance might not be manifest.—Hippocrates 430 BC

Epilepsy was regarded in ancient times with awe and superstition until Hippocrates revolutionized our understanding and care of diseases with his careful system of observation and treatment. The Hippocratic School separated rational methods from magical and religious practices in a number of texts; nevertheless, the association with demons and spirits remained for hundreds of years (Daras et al., 1994; Riggs & Riggs, 2005). In ancient Rome people suffering from epilepsy were avoided or spat at for fear of contagion (Todman, 2008). Saint Paul of the 1st century writes of this habit, in regard to himself (Landsborough, 1987), and three of the Gospels describe a clear case of seizure considered demonic possession. "Teacher, I brought you my son, who is possessed by a spirit that has robbed him of speech. Whenever it seizes him, it throws him to the ground. He foams at the mouth, gnashes his teeth, and becomes rigid. I asked your disciples to drive the spirit out, but they could not." (Mark 9:14-29, NIV). Jesus resolved the problem but not Paul's. In fact the great Christian missionary of the first century may have himself suffered from temporal lobe epilepsy (TLE).

Were Paul's ecstatic visions, his conversion on the road to Damascus, the "light from heaven" which went off in his head, a product of TLE and temporal lobe disinhibition? It is always difficult to diagnose a patient, especially one who died prior to the invention of the MMPI or MRI, but Paul alludes to his illness in his writings and from this sparse evidence scholars have presumed something about his life and his condition. Paul describes an estatic personal experience in his letter to the Church in Corinth in which he felt "caught up to paradise." He was "caught up to the third heaven. In the body or out of the body? That I do not know... and (here he) heard sacred secrets which no human lips can repeat." A

sense of unreality in relation to one's body in space and a dreamy state of auditory hallucinations reminded Landsborough (1987) of his patients' experiences with TLE.

You are all healthy people, but you have no idea what joy that joy is which we epileptics experience the second before a seizure... I do not know whether this joy lasts for seconds or hours or months, but believe me, I would not exchange it for all the delights of this world."—Fyodor Dostoyevsky

The great Russian novelist of the 19th century, Fyodor Dostoyevsky, likely inherited his condition as both his father and his son had seizures, but he was fortunate in having ecstatic auras which are not typical of most individuals with epilepsy. He incorporated such experiences into his novels, and many of his major characters were epileptic such as Prince Myshkin of *The Idiot*. Like Saint Paul, Fyodor spent much time working on theological issues, as his books can attest to. Many sufferers of TLE are hyperreligious and religious conversion following ecstatic auras is not uncommon (Dewhurst & Beard, 1970; LaPlante, 2000; Trimble & Freeman, 2006; Devinsky & Lai, 2004). Much of Western religious and mystical imagery may be explained as limbic contents temporarily brought to verbal awareness, from William Blake and Dante Alighieri to the Prophets of Judaism. We may never know what exactly transpired on that road to Damascus in the case of Saint Paul, but we do know that it changed the course of human history, transforming a persecuted and poverty-stricken Galilean sect of Judaism into the most dominant force of history. But what interests the applied neuroscientist part of me is that his ecstatic experience clearly would have neurological correlates, which given the right circumstances might be replicated. (Which is not to say that on that dusty road Paul experienced something science can explain, only that we may eventually be able to replicate some of the brain state.)

Of course not all religious figures were epileptic (Paul may not have been) and not all individuals with epilepsy are religious, although TLE patients tend to exhibit hyperreligiosity and hypergraphia (excessive writing) and other attributes helpful in starting followings including a belief in a "greater awareness" they have been exposed to. Joseph Smith, Jr (1805–1844), founder of the Latter Day Saint movement, exhibited signs of seizure (or spiritual rapture) in a famous incident he related to others from the spring of 1820. In the woods of western New York he was seized with some strange power that rendered him speechless and then he saw a light as bright as the sun (Dewhurst & Beard, 1970). He went on to found a religion with millions of adherents. Other well-known religious figures who may have suffered seizures include the Judaic prophet Ezekiel (6th century BC; cf. Altschuler, 2002) who wrote of his fainting spells, speechlessness, and compulsive writing, Emanuel Swedenborg (1688–1772; cf. Foote-Smith & Smith, 1996), numerous saints including Saint Teresa of Ávila and Saint Joan, and possibly even Muhammad, founder of Islam. It may be that TLE, particularly of the right hemisphere, enables an individual to break free of conventional organization of experience and allow individuals to experience the earliest moments of Creation, what Gnostics call the Plemora. Joan D'Arc (1412–1431) often felt that the secrets of the universe were about to be revealed to her, an experience shared by many of the physicists working at the Large Hadron Collider outside of Geneva, where the early universe is being simulated with powerful magnets. Joan said her visions were triggered by ringing church bells, i.e., musicogenic, and like Dosteovsky she had an ecstatic aura. Musicogenic epilepsy is often triggered by music with emotional significance, and my own mother had a similar experience of visions in response to church music after alpha-theta training years ago. Joan's voices propelled this teenager onto the world's stage and led the charge in ejecting English rule from French soil. But like many female TLE sufferers of her day, she was tried as a witch, as well as a heretic, and burned at the stake.

Individuals with epilepsy were often considered witches in Medieval Europe. One of the most popular publications of this time was the Malleus Maleficarum, published in Germany in 1487, which instructed magistrates on how to identify, interrogate and convict witches, and it included convulsions as a common feature of witches. The de-facto handbook for witch-hunting was published 13 times between 1487 and 1520, and many more times as the Renaissance dawned. Some scholars estimate that more than 200,000 women were murdered in large part because they exhibited the symptoms of epilepsy (Temkin, 1945). The treatment of King Charles' convulsions 200 years later seems tame in comparison

In the Middle Ages, epilepsy was called the "falling sickness" and people looked to saints and relics for cures (Diamantis et al., 2009). Some interpreted epileptic seizures as a transient form of death followed by a resurrection, a Christ-like experience for the lucky or unlucky (Mann, 2009). Religious and magical remedies were the norm for epilepsy, despite the Hippocratic legacy. Theodorus Priscianus (c.380 AD) recommended occult remedies such as amulets alongside more rational remedies including a healthy diet, massage, daily exercise, and bathing. Alexandros of Tralleis (525–605 BC) recommended "the plant peony plucked during the waning of the moon" or to "wash the head of the patient and burn a ram's horn under his nose and he will fall down" or to wear a ring of jasper (Stol, 1993; Eadie & Bladin, 2001). A thousand years and the same remedies were still being suggested by medical authorities. The step-daughter of George Washington, "Patsy" Custis, wore an iron ring to treat her seizures, and she subsequently died from a brief seizure in 1773 (Doherty, 2004). The famed Roman physician Galen thought that the moon governed periods of epileptic fits. He also divided epilepsy into three forms (incorrectly), assuming that seizures could originate in the body and travel up to the brain.

The chief factor in the cure of epilepsy in the young is change, especially that due to growing up, but seasonal change of climate, or change of place or mode of life, are also important.—Hippocrates 430 BC

Galen's medicinal ideas held sway over most of Europe for a millenia. According to Galen epileptogenesis occurred when substances (humors) blocked the lateral ventricles. Caelius Aurelianus, a 5th century physician, added to this the following factors: drinking too much wine, indigestion, compression of the brain, and sudden frights, and he believed it could be transmitted to nursing infants through mother's milk. Arnold of Villanova (14th century) believed that different humors resulted in seizures at difference phases of the moon and the etiology of seizure included "bites of mad dogs or reptiles, or from poisoned, corrupt, and pestiferous air" which filled up of the chief ventricles of the brain. John of Gaddesden (1492) added wind as a moderating influence "such as the South Wind, or the East Wind, and the North Wind when they follow a South Wind. Also, everything that comes suddenly and quickly heats the

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head, such as a long stay in the south, or bath, or close to a fire..."

The Renaissance ushered in new ideas about the world and humanity and this included new conceptions of epilepsy. Paracelsus, a pioneer in alchemy/medicinal chemistry, challenged Galenism in the 16th century and he believed that epilepsy was present in all of nature including plants: "Earthquakes and falling sickness have the same causes," he was known to say. English anatomist Thomas Willis (1667) believed that "(e)pilepsy is caused by contractions of the membrances around the brain compressing and constricting its substance and preventing the proper and equable expansion of the (animal) spirits...".—an explosion of 'animal spirits' in the brain that resembles the internal combustion engine. As the concept of animal spirits died in neuroscience, the role of electricity in epileptogenesis grew, especially after Galvani (1780) demonstrated electrical activation in his frog preparations, until we reach the semblance of modern-day thinking on this topic.

Epileptic convulsions can be understood, as "the result of experiments made by disease on the brain of man." (John Hughlings Jackson, 1875).

By the late 1870s the modern era of epilepsy research and treatment had begun with the work of three English neurologists, John Hughlings Jackson, Russell Reynolds, and Sir William Richard Gowers. In 1904 the term "epileptologist" was coined by American William Spratling, a neurologist who specialized in epilepsy (Dasheiff, 1994). However we were not yet free of the stigma of epilepsy despite our modern sensibilities. An ugly chapter of eugenics followed, and 33 states enacted laws that allowed for the sterilization of epileptics, notably those under the care of state institutions (e.g., Virginia Sterilization Act of 1925). Many states also enacted laws that forbade people with epilepsy to marry. Only when Congress passed the Americans with Disabilities Act of 1990 were these laws overturned in toto, but not before 60,000 involuntary sterilizations of American citizens had taken place.

Unfortunately there remains misinformation and misunderstanding about epilepsy and seizure disorders to this day, including the notion of an epileptogenic focus. A third of all patients who undergo surgery for intractable epilepsy continue to have seizures postoperatively (e.g., Kim et al., 2008). Brain areas are removed and the patient is no better off. That the supposed starting point for a seizure is removed and convulsions continue reflects how little we actually do know about epilepsy. It may be that the entire brain runs fast in such cases, making training the best option. As many as 50,000 Americans die each year from seizures and related causes (e.g., drownings, car accidents) and 1 in 10 people will suffer a seizure during their lifetime. The mortality rate is 2 to 3 times higher and the risk of sudden death is 24 times greater than that of the general population, yet research funding lags far behind many other neurological afflictions, with $35 a patient for epilepsy compared to $129 for Alzheimer's and $280 for multiple sclerosis (Meacham, 2009).

Epilepsy reflects a hyperexcitability to brain state changes or transient sensory stimuli due to an inhibitory deficiency in neural regulation. Common seizure triggers include sleep deprivation, stress, drugs or alcohol, menstruation, nutritional deficiencies such as low blood sugar, medications, hyperventilation, photic stimulation (flashing lights or sounds), and sleep transitions (e.g., waking, falling to sleep). We have had descriptions of seizure triggers and symptoms for centuries, in ancient records in clay cuneiform tablets and Egyptian papyrus. Epilepsy was called *bennu* in ancient Babylonia and "*apasmara*" (loss of consciousness) or "*rupa*" in Ayurvedic (Indian) texts (Jain & Tandon, 2004). An ancient medical treatise from China, the The Yellow Emperor's Inner Canon (or Classic of Internal Medicine), included epileptic symptomalogy (Scott, 1993). A 4th century account of a generalized tonic-clonic seizure belongs in a modern-day textbook: "After various premonitory signs the patient falls down, stretched out or twisted, and in this condition he remains for some time. After these tonic convulsions he passes into the stage of clonic convulsions and a condition where he appears to be sleeping. The attack is followed by complete amnesia." (Temkin, 1945).

Epilepsy advanced the field of neurology and in no small part produced Western civilization as we know it. Julius Caesar, Socrates, Napoleon, and Saint Paul all suffered seizures from what we can tell. As Tennyson put it, "*All at once, out of the intensity of the consciousness of individuality, the individuality itself seemed to dissolve and fade away into boundless being; and this not a confused state, but the clearest of the clearest, the surest of the surest, the weirdest of the weirdest, utterly beyond words.*" From such moments of clarity may come lifelong motivation to change the world.

The story of the treatment of epilepsy with neurofeedback begins in the 1960s, when Mercury astronauts claimed they saw Polynesian natives waving at them as they flew over the Pacific. In 1967 Gordon Allies, inventor of amphetamine, was contracted to test toxicity of the Mercury capsule rocket fuel with David Fairchild to determine if it caused what were clearly hallucinations on the part of the astronauts, but Gordon died before the contract was over and Fairchild asked his friend M Barry Sterman to help finish the work. At the time Sterman was studying EEG activity in cats and using operant conditioning to see whether brain behavior (i.e., EEG) behaved like motor behavior in terms of acquisition, consolidation, and extinction. The rest of the story is well known in our field: Sterman trained 10 cats to produce SMR (sensorimotor activity) over the motor strip for chicken broth & milk, then Fairchild came knocking at his door and they needed cats to test rocket fuel on. Fifty cats were injected with NASA's rocket fuel monomethylhydrazine (MMH), which resembles gamma-aminobutyric acid (GABA) molecularly and thereby blocks its synthesis in the central nervous system. Without the brain's primary inhibitory neurotransmitter, GABA, the cats' brains were unable to diminish response to stimulation, even mild stimulation such as blowing in the face, so that any stimulation would lead to convulsions (Sterman, personal communication). Of the 50 cats injected with MMH at 100 mg/kg, 10 had undergone SMR training and it happened that these culprits blew the expected toxicity curve. Typically, cats went into a grand mal seizure within one hour after injection of MMH but those trained to increase SMR in the past showed a different pattern: Seven took significantly longer to seize and 3 did not convulse at all, the seizure thresholds changed in response to EEG training. I suspect that SMR training increased GABA-ergic receptor density in the motor or afferent pathways, thereby dampening sensorimotor excitability and blocking any runaway activity or seizure. These results cannot be explained by placebo ("I shall please" in Latin) as cats didn't know what to expect nor did the experimenter.

Neurofeedback now has a 40-year history for treating seizures (Sterman et al., 1969; Tan et al., 2009) and this technology and approach (operant conditioning) has since been adapted to treat an astounding

array of human frailties including mood and sleep problems, attention and learning difficulties, as well as brain injury and drug abuse. The mysteries of brain function still remain with us 4,000 years into our investigation, but with neurofeedback we've added an extremely powerful tool to resolving its disorders.

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Jonathan E. Walker, M.D.

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

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Making Sense of Infra-low Frequency Neurofeedback

Lincoln Stoller, PhD, Tenger Research, LLC

Abstract

Infra-low Frequency (ILF) neurofeedback training is loosely defined as training EEG signals below 0.5 Hz. This is generally well understood in the context of slow cortical potentials (SCP), but poorly understood in cases where trainees' claim to discriminate between harmonics differing by hundredths of a Hertz in the 0.1 Hz EEG range (Othmer, 2010). We analyze signals of this frequency to explain what is happening when we train these EEG components. This training can affect both the SCP and long wavelength, rhythmic cortical activity.

Background

Real harmonic functions of time take the form A•sin(X), where sin( ) is the sine function of an argument given as X, and A is any real number that does not depend on the argument. To display its important structure, the argument X is written as (wt+h) where w is the frequency, t the time, and h the phase.

Harmonic functions form a complete set. This means that any function of time that meets certain conditions can be expressed as a sum of a potentially infinite number of harmonic functions each with a different phase and frequency. For this to be the case a function must be continuous and all of its rates of change must be finite at every moment. Continuity requires that the function model a phenomena that is continuous in time, and "finite rates of change" mean that the phenomena varies smoothly, even if that smooth variation is apparent only at the highest magnification. In particular, even if a phenomenon appears to have an inflection point where it changes direction instantaneously, as discerned by the naked eye, as long as this change either is not literally instantaneous, or it can be modeled as something that is less than instantaneous, then it is completely amenable to being expressed as a sum of harmonic functions.

When a function of time is expressed as a sum of harmonic components it is said to be "decomposed into harmonics," resolved into its "frequency spectrum," or shown as a "spectral decomposition." These conditions are met by most measured quantities of natural systems, such as brain waves, and for that reason we can use the technique of spectral decomposition to describe the EEG.

The fact that such a decomposition can be done says little about the function being decomposed. Measuring some function of time and decomposing it into harmonics adds no information to the observation, but it might make some properties of the function more evident. In particular, decomposing the EEG signal makes various important structures of the signal more evident, and we are able to describe the signal more simply. And for the purposes of feedback, the decomposition enables a person to interact with specific aspects of the EEG signal over a period of time, rather than with the signal at an instant.

Harmonic decomposition is straightforward in theory, but the description given above contains assumptions about how natural phenomena can be modeled mathematically. The two major assumptions are that the phenomena being modeled is not truncated, which means that it has no start or end, and that its values are known at all times. In practice our observations do begin and end, and are intermittent. These practicalities violate the basic requirements of harmonic analysis but, because the underlying phenomena do satisfy the requirements, we can adopt looser rules that apply to our finite and intermittently observed EEG signal. As a result we get some "fine print" that goes under the name of Signal Analysis.

Neurofeedback practitioners learn the rudiments of harmonic analysis, understanding that the erratic EEG signal is composed of harmonic elements. Practitioners are unfamiliar with signal analysis and are unaware of the ambiguities that signal analysis introduces into the EEG spectrum. In particular, infra-low frequency neurofeedback is misunderstood.

ILF Feedback

The longest spectral wavelength that can be extracted from a series of EEG measurements is equal to the time period over which the EEG signal is sampled. This means that to resolve a frequency of 1/8 Hz, or a wave of 8 seconds duration, requires analyzing at least 8 seconds of EEG data. An observation over this time period, or epoch that samples at a typical rate of 256 values per second enables us to discriminate between spectral components roughly 0.1 Hz apart.

Let's suppose the feedback shown to the trainee is updated 16 times per second. With an 8 second epoch, and with each feedback event separated from the next by 1/16 of a second, each subsequent feedback event is highly correlated. If the amplifier is recording 256 EEG values per second, an 8 second window accumulates 2,048 EEG values. The 8-second windows for two feedback events that are 1/16 of a second apart share 2,032 of the same EEG measurements. That is to say only 16 of 2,048 EEG measurements contribute new information that is fed back to the trainee. As a result, the feedback is about 1/20th as responsive to changes in the trainee's EEG as it would be if an epoch of 1/2 second were used, which would only collect 128 EEG values.

Due to this sluggish feedback response we might expect the trainee to be unable to modify their EEG when training a frequency as low as 0.1 Hz. To better understand this we should ask how the trainee's short-term responses change the infra-low frequency components.

What is ILF Training Doing to the EEG?

The EEG signal has its greatest effect on a specific spectral component when it changes in synch with that component. This says nothing more than to satisfy feedback criteria that reward alpha waves, for example, the trainee generates alpha waves. But over

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Infra-low Frequency Neurofeedback

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a short interval of time, an interval much shorter than the frequency of the ILF spectral component being trained, the trainee can only generate a small portion of ILF cycle. What sort of change in their EEG must the trainee accomplish in the short term in order to satisfy a feedback reward criteria based on an EEG component whose full period extends over a much longer time?

The change of a sine wave can be represented by a polynomial that is an infinite sum of terms each of which contains higher powers of the argument. This is called the Taylor Series expansion and for the sine wave it begins with a term that's linear, followed by a term that's proportional to the cube of the argument, and then the argument to the 5th power, and so on. It's important to note that these contributions alternate in sign, which means that when the value of the argument is less than 1, the contributions from these terms both get smaller and partially cancel each other out. When the argument is much less than 1 the higher order terms contribute proportionally little to the infinite sum and can be ignored at the expense of incurring a small error. The smaller the argument, the better the approximation one gets when ignoring the higher order terms.

The long wavelength components of the EEG have the smallest arguments, and when considered over a time interval short compared to their wavelength it's sufficient to compare them by comparing the effects of their leading terms in this power series. In the limit of extremely small arguments one can consider the sine function to look like a segment of a straight line. For example, if you looked at just a few inches of a long, smooth ocean wave, then in spite of the fact that its surface is described by a curved line, the few inches of profile that you could see would look like a straight line that was tilting back and forth like a seesaw. If you were to consider waves of increasingly shorter wavelength, then at some point you would start to see some curvature in this short line; the curvature would appear in phase with this seesaw motion curving up, going flat, and then curving down.

It's important to note that the contribution from a small portion of the full wave component, say 1/2-second of an 8-second waveform, is not symmetric in its amplitude about zero. That is to say that if you were looking at a short straw floating on the surface of a long wave, then not only would the straw rock back and forth like a seesaw, but it would also rise and fall at the same time. What this means for feedback is that the trainee is rewarded for adding a component to their EEG signal that varies between being electrically net positive to net negative over the longer term. This is true whenever the feedback is being generated over a time period much shorter than the epoch over which the spectral components are measured. The difference in the trainee's response to rewards at different long wavelength components is that the longest components record the greatest contribution from the smoothest change in trainees EEG, which is to say a more linear change in the electrical signal over time, while positive contributions to the shorter wavelength reward components (though still much longer than the feedback interval) are generated by incrementally more rapid changes that continue to have a net positive or net negative contribution.

Is ILF Feedback Training the SCP?

Slow cortical potentials (SCP) are discussed by Ute Strehl (2009) who shows them to have positive and negative electrical components. SCP changes persist over periods longer than 6 seconds, show a sharp cusp, a slow relaxation period, and are not periodic. Nevertheless, if 8 seconds of a SCP signal is decomposed into harmonics, then it will include long wavelength components. Its largest signature, however, will be at zero frequency as typical of a DC excursion away from electrical neutrality, but this component is automatically removed from the EEG spectra on that supposition that it represents an artifact in the phenomena of interest, which are presumed to be net neutral, periodic signals. The SCP long wavelength components might resemble the long wavelength components of sinusoidal cortical activity over the short time intervals to which the trainee is attending. If there is no way to discriminate the SCP from ILF sinusoidal activity, then there is no way to distinguish whether it's the SCP or ILF sinusoidal activity that is being rewarded.

Because the electrical signature of the SCP is neither smooth nor periodic it will have a complicated set of harmonics that we cannot assume to be dominated by low frequency components. Using the harmonics of the EEG to train SCP would require rewarding more than the frequencies in a narrow band, and would instead require rewarding a set of frequencies that, when added together, reproduce that aspect of the SCP that the trainee is to develop.

Tom Collura (January, 2009) contrasts the electrical behavior of low frequency harmonics with that of slow cortical potentials in his discussion of BrainMaster's Atlantis EEG amplifier. He points out three differences:

- Long wavelength periodic EEG signals display much weaker voltages than changes due to the SCP.
- Differences in the nature of the training, which partly reflects the different nature of the signals, and which leads to different subjective experiences for the trainee.
- The SCP is episodic by nature and so is less amenable to continuous forms of training, such as entrainment and threshold-based rewards.

Meaningful Signals?

A greater potential difference is that whereas the SCP is thought to originate in the action of the glial cells, the long wavelength EEG components may be nothing more than artifacts generated by transient shifts in the DC potential. That is to say they may be unimportant. This point of view seems to be held by some leaders in the field of neurofeedback, but since it has not been elucidated we're left to guess possible reasons behind it. Here are three possible arguments.

Objection: "EEG wavelengths longer than the EEG epoch don't exist."

Response: I'm unaware of anyone claiming to train such wavelengths.

Objection: "Long duration phenomena cannot effect, or be affected by short timescale training."

Response: While it's true that 1/2-second changes in the EEG are an order of magnitude less powerful than the long wavelength signal itself, we commonly observe changes of this magnitude in signals as a response to feedback training. It's plausible that similar differences may be significant here.

Objection: "Long duration phenomena are unaffected by training in which the trainee's response occurs over a small fraction of such long duration phenomena."

Response: This objection may be based either on the idea that a slow, neural phenomenon cannot respond rapidly, or on

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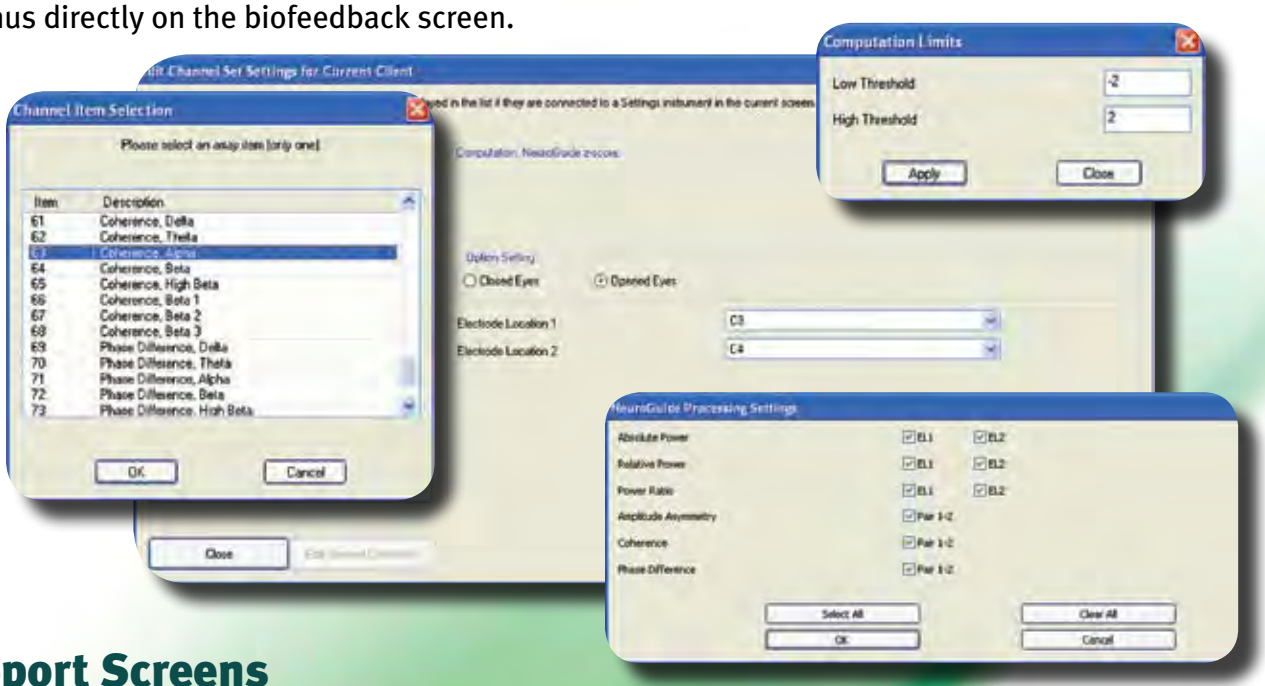

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the assumption that slow and fast neural phenomena are not linked. While we lack knowledge of the generators of the EEG, it is common for nonlinear systems to couple at different frequencies, the slow rocking of a house fan due to an imbalance of the rapidly spinning blades being one example. In this case adjusting the motor's rheostat to make a small change in the speed of fan blades can immediately stop the fan's slow oscillations.

More relevant examples might be the long time-scale phenomenon of task planning that depends on the short time-scale phenomenon of mental focus, or the way a dancer improves the fluidity of his or her overall movement by focusing on such momentary phenomena such as the movements of joints, the shifting of their balance, and the mastery of their gestures. In both cases contributions over a 1/2 second interval affect the phenomena that exist over a 5 to 10 second interval. In both cases the brain must discern the small changes of which the larger phenomena are composed.

Transients

There are two sources of transient phenomena in the EEG: episodic neurological events, such as the SCP, and the time relaxation of the amplifier's circuit due to an abrupt change in the signal. The origin of the first transient is in the brain, the origin of the second transient is in the amplifier. In both cases the signal generates multiple harmonics, and in both cases training the originating event can be attempted using an AC or a DC amplifier.

Training with a DC amplifier aims to train base-line changes in the overall electrical potential and presumes the importance of this aspect of the phenomena. Training with an AC amplifier removes changes in the base-line and presents only harmonic components over the epoch that is observed. Training based on the signal's harmonics presumes these harmonics capture some mechanism of importance. The character and the duration of the displayed phenomena are different in each case.

A transient signal has both impulse and harmonic features. It can be fully represented as either a changing, instantaneous, DC potential, or as a complete set of harmonics measured over a period of time. Each approach has the potential to train different aspects of the signal. The two training approaches are complementary, each reflecting aspects of the transient signal missing from the other.

Conclusion

Assuming the SCP activity is sufficiently "loud" in low frequency harmonics, so as to enable the brain to distinguish it from other cortical activity, ILF training may impact some aspect of it. Transients also generate high frequency components ignored by ILF feedback that play an important role in describing the signal, and may play an important role in training. Training the whole SCP would require the simultaneous reward of a mixture of many frequencies, not only the distinctive long-term DC polarity changes reflected in the zero frequency component of the EEG signal. If there are important cranial rhythms in the 0.1 Hz region, and if the slow harmonic components of the SCP are of importance, then these are best trained with a slow response AC amplifier.

The important point is that ILF training is mathematically plausible, may provide a window onto the training of SCP phenomena, and may also have repercussions for the training of slow, periodic cortical rhythms that may or may not be related to the SCP. Regardless of the signal's origin both DC and AC training can be conducted, and each training modality focuses on different aspects of the signal.

Contact: ls@tengerresearch.com

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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.

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The Brain's Idle State

Cynthia Kerson, PhD

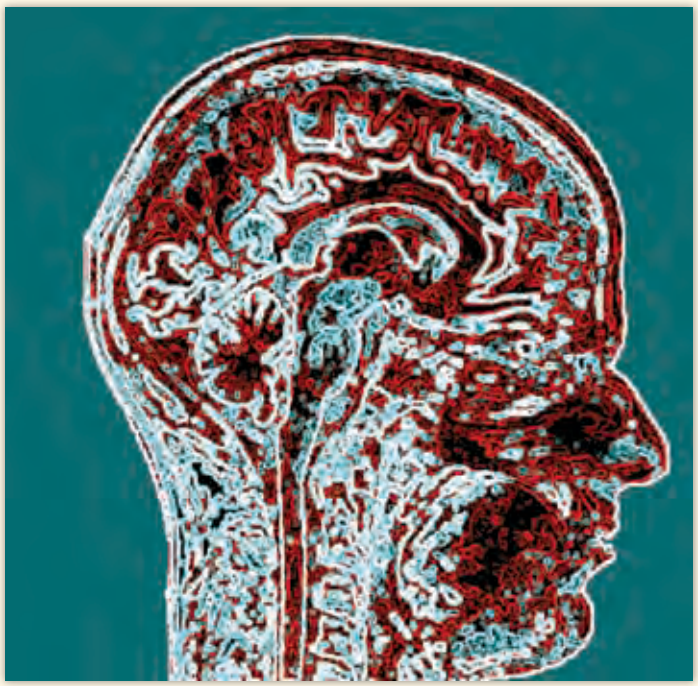
Many of us think the eyes closed resting state while recording the brain is an idle state. Some of us look for the dominant frequency and this state is often used to compare to other tasks, including eyes open, hyperventilating, math, reading, meditating, etc. Do you believe the eyes closed state to be an idle, or good example, of a resting state?

Resting eyes closed does of course reflect dominant alpha idling frequency of the visual cortex.

Also, ec is useful for comparison with other tasks due to ability to better control for movement artifact.

However, it is well known that eyes closed eeg actually has lower test retest reliability (stability) than task related eeg, probably reflecting variability in arousal, level of cognitive engagement etc during ec.

Roger Riss, PhD



Eyes closed rest is certainly not an idle state. In the fMRI literature this state is now referred to as "mind-wandering," that typically involves active thinking and problem-solving about personally relevant issues with or without awareness that one is doing so (so-called meta-awareness.)" In our meditation studies, in which we are specifically looking to see just how quiet the brain can get, we have abandoned eyes-closed rest as a baseline in favor of a minimal but standardized attention task such as counting a repetitive sound. Visual fixation on a crosshair is still widely used in fMRI research whenever the experimental task involves visual stimuli.

David Hubbard, MD

E. Roy John and myself submitted a grant to identify EEG/EP differences between age matched normal controls and learning disabled children to the National Science Foundation in 1972 in which we wrote one sentence about resting eyes closed and eyes open EEG and 40 pages detailing active tasks. A year or two later NSF scheduled a site visit to evaluate the progress of the grant and we could only complete analyses on the eyes closed and open EEG data and found about 97% discriminant accuracy with three independent cross-validations. NSF site visitors were pleased and continued the funding and we spent the next three years analyzing the active task data and never got above about 85% discriminant accuracy. The results of these analyses were published in John, E.R., Prichep, L.S., Ahn, H., Kaye, H., Brown, D., Easton, P., Karmel, B., Toro, A., and Thatcher, R. *Neurometric Evaluation of Brain Function in Normal and Learning Disabled Children*. The University of Michigan Press, Ann Arbor, Mich., 1989.

We were puzzled by this and assumed that the reduced sensitivity of active tasks was because there was more artifact and less ability to control experimental conditions across subjects, e.g., different people interpret task instructions differently, differences in stimulus intensity and display distances, etc. Myself and E. Roy John continued to publish active task research but over the years we always found that the resting eyes closed and eyes open conditions were clinically very sensitive and much easier to obtain.

This paradox of weaker sensitivity with active tasks was not solved until the late 1990s when fMRI showed a decrease in overall brain energy consumption in an active task in comparison to resting eye closed or resting eyes open states. The decrease in metabolism in an active task was dramatic and large and was confined primarily to the bilateral temporal lobes, bilateral hippocampus, precuneus, posterior cingulate and the medial frontal lobes and was later called the "Default Mode Network" (DMN). fMRI and EEG/MEG studies further elaborated that only a relative small reorganization of non-DMN neural groups were involved in active tasks and this relatively weak effect was difficult to detect and required a lot of averaging. Even after averaging it was seen that widespread brain regions were involved in many different tasks but in a coordinated and sequential manner over very brief time intervals. See Marcus Raichle's recent review of the DMN in "The Brain's Dark Energy," Scientific American, March 2010.

The DMN has been shown to increase its activity upon self reflection or self-evaluation and what is called "time travel" when people ruminate about the past and the future while they are not engaged in a task. It appears that this is a very important property of the human brain, i.e., to use the resting moments for self-reflections, for

thinking about the future and the past, for planning for the future, for recombining old memories and thoughts. Thus, eyes closed and eyes open conditions are not actually periods of brain "rest", quite the contrary these are conditions of heightened brain activity and this is part of the reason that resting EEG studies have reported very high sensitivities in resting conditions over the years and why so much of modern day fMRI and PET studies are focused on eyes closed and eyes open resting conditions and why this focus will continue into the future.

Robert Thatcher, PhD

It is idle thinking...but somehow resting for the eyes only, for lack of information to process.

Dr. Victoria L. Ibric

It's clear from fMRI research that any 'resting' state, whether eyes open or eyes closed, is an active state and areas involved in the default mode network will be active to quite a high degree.

I'd say that the 'resting' eyes open and/or eyes closed states are our baseline that we use to compare to known values rather than as a baseline against task. We have some pretty good data that shows what the EEG during these states should look like, including peak frequency, etc.

and so if we see differences I see that as an opportunity to ask questions to find out why there are differences from expected values and patterns.

John Anderson

An eyes closed resting state is unconstrained, and many people are "busy" mentally during their recordings.

Eyes closed seems a good heuristic for "resting state," but I think we are really trying to tap into the default network. In our minimaps with trauma clients at the Trauma Center in Boston, we find a pretty consistent pattern of beta and high beta increase in the eyes closed condition. Clearly this is not a "resting state" for these brains, but more likely an activation state, and perhaps indication of a poorly working default network. More remains to be discovered.

Richard L. Jacobs, Psy.D

The rhythmic dominant background EEG feature parietal-occipitally with eyes closed that attenuates with eye opening is the person's alpha, by classical neurology and EEG definition. This can be from as slow as 3-4 Hz to as fast at 15-16 Hz.

In the fMRI literature this state is now referred to as "mind-wandering," that typically involves active thinking and problem-solving about personally relevant issues with or without awareness that one is doing so.

The well done research on the background rhythm suggests that it is not all the same factor or component, as the slower alpha corresponds with hypoperfusion, but the faster end of the same person's alpha spectral peak(s) can be a positive perfusion, though not a significant level of hyperperfusion.

This suggests that the background alpha is not a single entity, and the perfusion finding suggests that it is not all "resting."

An eyes closed resting state is unconstrained, and many people are "busy" mentally during their recordings. The only areas that can generally be said to be idled or at rest are the primary visual cortex areas posteriorly, where the alpha "blocks."

These observations in no way can be used to suggest the brain is generally resting or idled elsewhere topographically, as the EEG perfusion correlates of local function would attest.

Wolfgang Klimesch's lab in Salzburg has done some great EEG studies with NF changing alpha tuning. Their work also would suggest that the alpha rhythm's tuning can be a critical feature associated with semantic/declarative memory, with faster alpha associated with superior performance on semantic memory tasks.

Faster and slower alpha are not well characterized by the databases, as they do not tell you things are faster or slower than expected, but rather tell you there is too much or too little power at various frequencies. Although the naming convention would suggest it tracks the peak, the peak frequency metrics don't measure peak frequencies, as the calculation is for a "centroid" or "mean" frequency, and not the peak power content in the band.

If perfectly normal alpha is slowed, the databases will say it has too much power at a slower frequency… when the power for alpha may be perfectly normal, just too slow… thus Z-scores can misguide an interpretation if frequencies are not appropriately tuned.

One of the more critical pieces of knowledge about someone's EEG is whether it is tuned too fast or too slow, and the impact of this on the cortical perfusion/function.

Jay Gunkelman, QEED

We obtained subjective reports from subjects after eyes-closed and eyes-opened recordings and behold, there is a directed focus on regulating eye movements, body movements, tongue, etc. These were coded by three independent raters and all coded as attention/self-regulation, additionally, attention to not becoming bored or internal state is also attention. Therefore in the absence of a direct stimulus, the default network is an attentional process. Any individual using EEG/fMRI/PET advises the subject or patient of the process and that movement needs to be minimized. No one simply tells the subject to rest, at least to my knowledge. Thus, we can conclude that this is a cerebral directive engaged in self-monitoring, self-mentalizing and not simply resting as the name may infer. We should have this ready for pub (already published in transactions of the human computational intellegence modeling proceedings) and of course in my dissertation. This is an important point of clarity that needs further investigation.

Rex Cannon

Tests are Simple When it Comes with Neuro Therapy

Kianna Sarvestani, Robby Stevens, The New School

Introduction

The purpose of this project is to find out if Neuro therapy helps kids perform better on tests. Both of our parents are specialized in Neuropsychology. Both of us have been interested in this for a long time. This prompted us to do an experiment on Neuro Therapy. Does Neuro therapy help kids perform better on test. We wanted to know if eleven subjects received Neuro therapy then 75% of them will perform better on test

Abstract

The hypothesis that Neuro therapy helps kids perform better on attention tasks was tested. The project gave subjects a test before Neuro therapy and after Neuro therapy. The purpose of this project was to see if with a short, incomplete session, you can see an increase in scores. In real life individuals have repeated sessions of Neuro therapy and, after many sessions, improvements are seen. The results showed that 91% of the subjects performed better on the test after Neuro therapy. Also the subject's performance did improve by an average of 10 percent overall. Neuro therapy right before a test may help performance. Future studies should include a control group to see how much of the change is the result of doing Neuro therapy and how much subjects improve with practice alone.

Materials and Methods

Material List

1. Stopwatch
2. Attention test
3. Pencil
4. Black pen
5. Red pen
6. EEG biofeedback machine

Experimental Procedure

1. Subjects will be brought in to the testing room
2. Then the researcher will explain the directions of the attention based test
3. After explaining the directions the researcher will say "Go" the subject will be underlining the letter or symbol that is at the top of the page
4. The researcher will wait forty seconds then say "Stop"
5. The researcher will repeat steps four and five two times
6. After taking the test they will walk over to the EEG biofeedback machine
7. Exfoliating Gel will be placed on both ears and one on the top forehead
8. Then the researcher will wipe off the gel
9. After that they will stick the sensors in a different gel
10. Then the sensors will be placed on the ears and top of forehead
11. The subjects will form pictures on the screen when their brain does the correct thing
12. After twenty minutes the subjects will be unhooked from the machine
13. The subject will then be taken to the area or room where they started testing
14. The researchers will explain the attention based test directions
15. Then they will repeat steps four and five four times

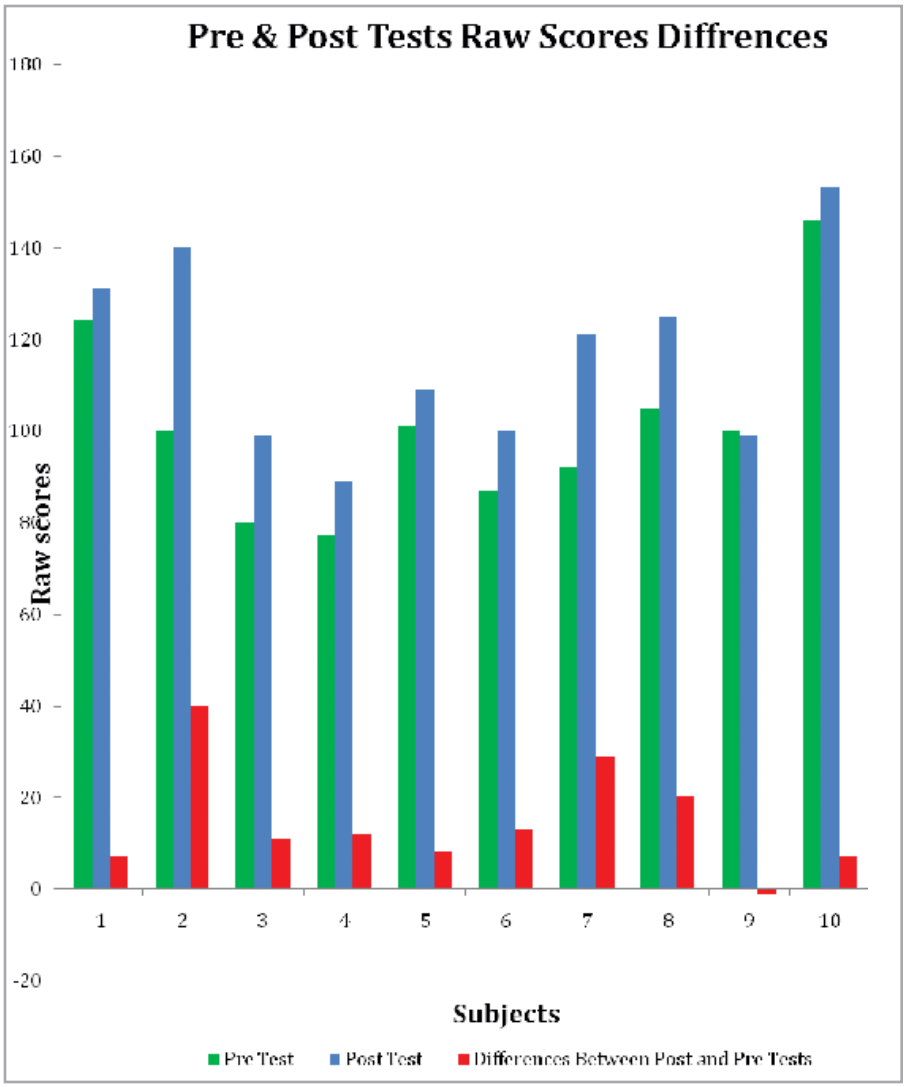
Before giving the Neuro therapy treatment, we gave each subject an attention based test. After the Neuro therapy treatment we gave alternative forms of the same attention based test. We then compared the performance of both tests. About 91 percent of the subjects did better after Neuro therapy than before. Although not all the subjects made a significant improvement in their tests after Neuro therapy, about 64 percent of them did. By significant we mean a more than a 10 percent increase in the average test score after Neuro therapy. Only one subject failed to improve their test average after taking Neuro therapy though there was no significant decrease in performance. The differences in the pre and post test raw scores are also impressive. In the second graph it shows the sum of the four pre tests, the sum of the four post test scores, and the difference between the two. All of the subjects that proved to perform better after Neuro therapy improved by a difference of seven points or more. One subject improved on the raw data by forty points and an average 11.75. The improvement on average is a good amount but it is

even easier to see when you compare the raw score data.

Discussion

The subjects in this study showed improved test performance after Neuro therapy. In Neuro therapy brain wave activity is measured by Electroencephalograph the subject receive feed back through the electrodes that helps them learn to change their brainwave activity. Through the computer game the subject plays advances only if they are producing certain brain wave patterns. In a clinical setting, the subjects get repeated sessions, for about 20-40 minutes, over a six month time frame to see improvement. In this experiment, the subjects showed improvement after one 20 minute session. Neuro therapy is a treatment for Attention Deficit Hyperactivity Disorder. The test we used in this experiment measures attention and the improvement the subjects showed is consistent with reports that Neuro therapy can improve attention. There were several variables that we could not control for in this experiment. The variability of the testing environment and the amount of background noise differed for each subject. However, the test conditions were generally similar for both pre and post tests. There were also interruptions with parents and teachers coming in and out. Another important variable that we could not control for was practice effect. The scope of our project prevented us from including a control group. Future research should include a group that does not set valid Neuro feedback/Neuro therapy. That group could then be compared to the Neuro therapy group to see if there is more improvement in the Neuro therapy group. The field of Neuro therapy is interesting and this study shows that

About 91% of the subjects did better on the test after Neuro therapy than they did before Neuro therapy.



Conclusion

About 91% of the subjects did better on the test after Neuro therapy than they did before Neuro therapy. The findings were consistent because of the high rate of improvement demonstrated. The subjects were also observed to concentrate and focus better on the post-test. Sixty-four % of the subjects made a significant improvement in their test performance following Neuro therapy and only one subject failed to improve. Our hypothesis was upheld and shows that this therapy can help improve test scores on attention test even in average 6th Graders.

Acknowledgments

There are many people we would like to thank and with out them are project would not be completed. First we would like to thank Ms. Anne Stevens for providing the equipment for this project and showing us how to use Neuro therapy. We would also like to thank Ms. Marry Ann Scott for providing the test and helped us with the research and helped us carry out are experiment. Also we would like to thank are adult sponsor Ms. Carolyn for helping us through the whole project and with the forms. Also we would like to thank Ms. Marry Kim for helping us with the graphs and data sheet. Lastly we would like to thank all the subjects who participated in this experiment.

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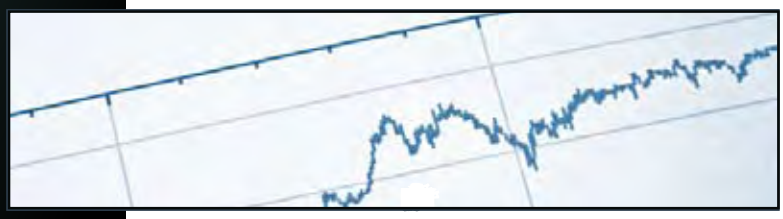

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Biofeedback of Slow Cortical Potentials in Epilepsy

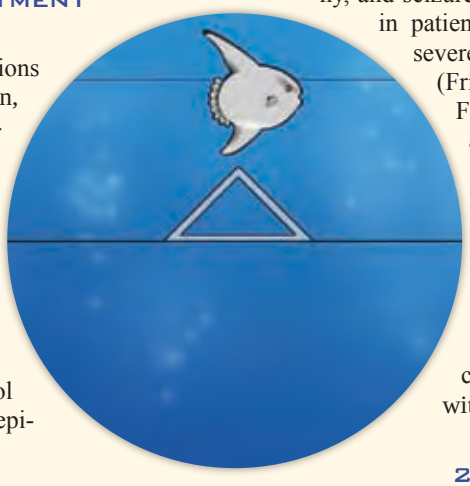
Sarah Wyckoff, MA
Institute of Medical Psychology and Behavioral Neurobiology, University of Tübingen

Epidemiology, Classification, and Prognosis

Epilepsy is one of the most common neurological disorders and affects people of all ages. According to the World Health Organization (WHO) (2009) approximately 50 million people have epilepsy worldwide; 4 to 10 per 1000 people, with an annual incidence of new cases around 40 to 70 per 100,000 people in the general population. In the United States, the 1-year prevalence is 7.1 per 1000 people (Hirtz et al., 2007) and the annual direct and indirect costs of epilepsy for the estimated 2.3 million prevalent cases in 1995 were approximately $12.5 billion with a majority of funds concentrated in patients with intractable epilepsy (Begley et al., 2000). Epilepsy may be caused due to head injuries, infections, tumors, congenital and genetic conditions, and cerebrovascular disorders. However, for six out of 10 people diagnosed with the disorder there is no known cause (WHO, 2009). Regardless of individual origin, epilepsy is a chronic disorder of the brain characterized by recurrent seizures resulting from a disturbed balance between excitation and inhibition of neurons located predominantly in the cerebral cortex. Partial seizures are the product of activation of neurons "focused" in one or more areas of the cortex with and without loss of consciousness. Generalized seizures involve activation of neurons in both hemispheres and include a variety of subtypes.

Medical Treatment Options

Current treatment options include medication, neurosurgical interventions, and behavioral management techniques. Studies indicate that up to 70% of patients newly diagnosed with epilepsy can successfully be treated (complete control of seizures) with antiepileptic drugs and following two to five years of successful pharmacological treatment about 60% to 70% of patients can discontinue medications without relapse (WHO, 2009). However, data collected from community and hospital based studies indicate that one-fourth to one-third of epileptic patients develop intractable, or drug-resistant, epilepsy (Kwan & Brodie, 2000). After the failure of multiple antiepileptic drugs and continuation of uncontrolled seizures patients may wish to seek surgical interventions. Although up to 70% of neurosurgical patients may become seizure-free for a prolonged period of time, most patients experience relapse after surgery (Kwan & Sperling, 2009). These findings highlight the need for cost effective alternatives and adjunct interventions to treat patients that fail to respond to pharmacological and neurosurgical methods.



Behavioral Interventions

Behavioral interventions focusing on identification and control of seizure antecedents, relaxation training, contingency management, and application of countermeasures have been found to produce a significant reduction of seizures compared to control groups at 10-week, 1-year, and 8-year follow-up (Dahl, Brorson, & Melin, 1992). Biofeedback of physiological signals and EEG rhythms has also been successful in the treatment of epilepsy. Diaphragmatic respiration training with percent end-tidal CO_2 biofeedback produced EEG normalization, restoration of respiratory synchrony, and seizure frequency reduction in patients with moderate to severe intractable epilepsy (Fried, Rubin, Carlton, & Fox, 1984; Fried, Fox, & Carlton, 1990). Galvanic skin response biofeedback training also elicited a significant reduction of seizures compared to a sham control biofeedback condition in patients with treatment-resistant epilepsy (Nagai, Goldstein, Fenwick, & Trimble, 2004). A recently meta-analysis of EEG biofeedback studies, indexed in online research databases between 1970 and 2005, provided data on seizure frequency changes related to treatment. The results indicated that EEG operant conditioning of sensorimotor rhythm (SMR) and slow cortical potentials (SCP) were able to produce a significant ($p < 0.005$) reduction in seizure frequency for patients with treatment-resistant epilepsy (Tan et al., 2009).

Self-Regulation of SMR

The investigation of EEG biofeedback as a treatment for intractable epilepsy has a 40-year history, with research in the United States focusing primarily on the augmentation of SMR with or without simultaneous inhibition of slow frequency rhythms (See Sterman in this issue) and research in Europe focusing on the self-regulation of SCPs. SMR training has its roots in animal studies and its efficacy as a treatment in epileptic populations was discovered accidentally. In the early 1970s, Sterman and colleagues successfully used EEG operant conditioning to reward increased production of specific 11-15 Hz rhythmic activity over the somatosensory cortex in alert but motionless cats (review, see Sterman, 2000). Following their EEG training, these cats were included as subjects in a study investigating the convulsive properties of toxic compounds used as rocket propellants. While non-conditioned cats in the study were found to have convulsions with exposure to an established dose of the toxic compound, the protective effects of SMR-training appeared to increase seizure thresholds for the conditioned cats. This hypothesis was tested and, once published,

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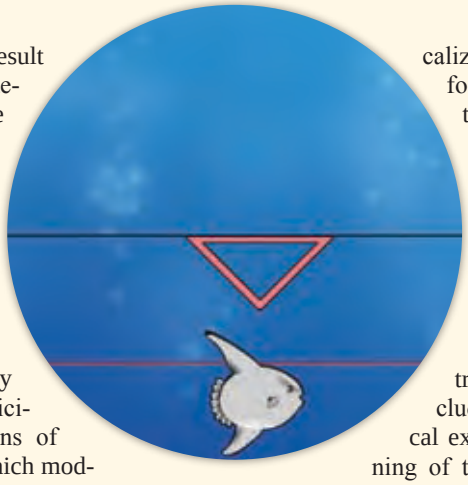
Slow Cortical Potentials in Epilepsy
continued from page 27

the findings stimulated research in several laboratories. The resulting body of research indicates that the entrainment of thalamocotical regulatory mechanisms through SMR training has the capacity to reduce neuronal excitability, blunt the impact of transient neuronal discharges, and stabilize state characteristics, thereby countering the abnormal and excessive synchronous neuronal discharges and cortical hyper-excitability observed in epileptic populations.

Slow Cortical Potentials

Following roughly the same timeline as the early SMR researchers, Birbaumer and colleagues began developing a physiological model in which slow cortical potentials (SCPs) of EEG reflect the threshold regulation mechanism of cortical activation and inhibition. In simple terms, negative SCP shifts increase the firing probabilities of a cell assembly, while positive SCP shifts inhibit this activity. These shifts are present in EEG signals below 0.01 Hz, last from 300 ms to several seconds, and play a critical role in the preparatory distribution of sensory, motor, and attentional resources. In a series of experiments, both healthy and clinical participant populations were able to learn self-regulation of negative and positive SCP shifts over central electrode sites (Birbaumer, Roberts, Lutzenberger, Rockstroh, & Elbert, 1992; Holzapfel, Strehl, Kotchoubey, & Birbaumer, 1998) as well as demonstrate simultaneous control over right-left-hemispheric differences (Birbaumer et al., 2000; Birbaumer et al., 1988; Kübler et al., 1999; Kübler, Kotchoubey, Kaiser, Wolpaw, & Birbaumer, 2001; Wolpaw, Birbaumer, McFarland, Pfurtscheller, & Vaughan, 2002).

In patients with epilepsy, negative SCP shifts have been observed proceeding and during ictal discharges and positive SCP shifts following seizure termination (Ikeda et al., 1997). These findings led researchers to conceptualize epilepsy as a problem in restraining the hyperactivation of neurons in which increased cortical negativity decreases an individual's threshold for paroxysmal activity. Therefore, it was hypothesized that training epileptic patients to suppress negative SCPs would attenuate epileptic discharges resulting in seizure frequency reduction. To date, two multicenter studies have shown that patients with epilepsy are able to learn self-regulation of SCPs and as a result have significantly decreased incidence of seizures (Rockstroh et al., 1993; Kotchoubey et al., 2001).



Rockstroh and colleagues (1993)investigated twenty-five patients with drug-refractory epilepsies that participated in 28 sessions of SCP feedback in which modulation of SCPs in both positive and negative directions was rewarded. Eighteen patients continued to monitor seizure frequency during a 1-year follow up period revealing that compared to baseline levels, seizure incidence was significantly lower during the follow-up period ($P < 0.01$). Six patients reported post-treatment seizure frequency below the 8-week median baseline values, seven showed seizure frequency reductions, and five did not report changes in seizure incidence. Kotchoubey and colleagues (2001) investigated the impact of SCP feedback, respiration training, and anticonvulsive medication treatment on seizure frequency. Thirty-four patients participated in 35 sessions of SCP feedback in which modulation of positive and negative shifts were randomly distributed. Cortical positivity was required in 50% of trials for the first 20 sessions and 67% for the last 15 sessions. Overall, significant seizure reduction was reported for the SCP ($P < 0.05$) and medication conditions but not for the respiration training. For the SCP training group the overall seizure frequency rate decreased by one-third, the rate of simple partial seizures decreased by one-half, and some patients became seizure free. Stability of SCP self-regulation was observed at 6-month follow-up and seizure reduction was maintained at 12-month follow up (Tan et al., 2009).

In an fMRI investigation of SCP training, positive SCP shifts produced a deactivation blood oxygen level-dependent (BOLD) response in comparison to baseline state around the training electrode, frontal lobe, and thalamus for successful regulators (Strehl et al., 2006). A variety of factors have been identified to impact SCP training outcomes. When training groups were subdivided on the basis of cognitive ability, low versus high intelligence, patients with lower IQ attained better clinical outcomes (Kotchoubey et al., 2001). However, localization of the epileptic foci appears to mediate the negative correlation between cognitive performance and seizure reduction. Strehl, Kotchoubey, Trevorrow, and Birbaumer (2005) identified three variables that accounted for 70% of treatment success, including: (1) level of cortical excitability at the beginning of treatment, (2) epileptic focus, and (3) personality variables. Successful outcomes may be enhanced in patient populations without large negative SCP amplitudes pre-training, without a left temporal epileptic focus, and in those scoring low on life satisfaction and high on stress reactivity. Despite these findings, it is important to note that patients with left temporal epileptic focus yield successful outcomes following SCP training, with several reporting reductions and/or remission of seizure incidence during follow-up assessments (Kotchoubey et al., 2001).

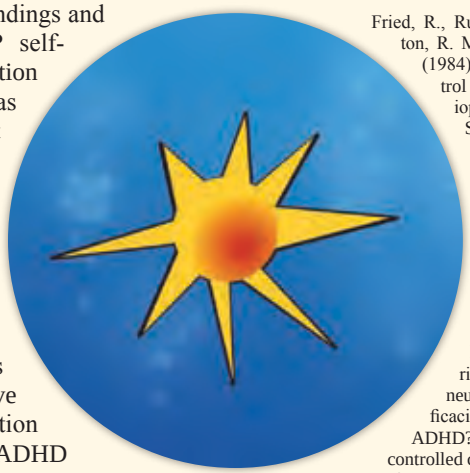
Training Protocol

SCPs are recorded at the vertex (Cz), referenced to the mastoids, with a skin resistance of 5 – 10 kΩ. The time constant is set at 10 seconds and the data sampled at 128 Hz (cycles per second). Because the eye acts as a dipole and produces a significant source of artifact in SCP signals, online correction for EOG components (blinks, horizontal and vertical movements) are essential. Patients are seated in a comfortable chair in front of a notebook or desktop computer for training sessions, with or without the clinician present in the training room. The trainee is cued by a graphic symbol to "activate" or "deactivate" their brain. The movement of the feedback symbol reflects the degree of the individuals SCP shifts: feedback moving upward as the result of a negative shift and downward as a result of a positive shift. The distribution of required negative and positive shifts are randomly set at 50/50 during initial training phases and increased to approximately 30/70 during later training phases. Successful activation and deactivation is indicated with a visual reward. To generalize newly acquired regulation skills to everyday life situations, 25% percent of all trials serve as "transfer trials" in which no feedback is presented during the active training phase but level of success is indicated with the visual reward system.

Feedback trials last 8 seconds and 100 - 120 trials constitute one session, approximately 1 hour long. In research settings, SCP training typically occurs over several blocks of intensive daily sessions (weekends off - 20+ sessions) for a three-week period, followed by a 3 to 8 week break in between training sessions. During their breaks patients practice SCP self-regulation in everyday situations. To facilitate home training and generalization of SCP regulation, patients are instructed to imagine producing the activation and deactivation shifts and assisted with a visual of the training screen via cue card or DVD. Current investigations of SCP training in ADHD populations at the University of Tübingen are investigating the use of a biweekly training schedule.

Conclusions

In conclusion, the research findings and clinical application of SCP self-regulation for seizure reduction in patients with epilepsy has been significant (Rockstroh et al., 1993; Kotchoubey et al., 2001; Tan et al., 2009). Self-regulation skills and seizure reduction have been shown to have long-term and stable effects (Kotchoubey et al., 1997; Tan et al., 2009). Additional research findings and treatment success have been observed in the application of SCP feedback training in ADHD populations (Arns, de Ridder, Strehl, Breteler, & Coenen, 2009; Gebensleben et al., 2009; Leins et al., 2007; Heinrich, 2004; Strehl, Leins, Goth, Klinger, & Birbaumer, 2006), for migraine reduction (Siniatchkin et al., 2000a; Siniatchkin, Kropp, & Gerber, 2000b; Kropp et al., 2002) and communication with totally paralyzed but intellectually intact patients in locked-in states (Kübler et al., 1999; Kübler et al., 2001; Vaughan, Wolpow, & Donchin, 1996). Additional clinical trials and controlled studies are needed to validate SCP feedback as a treatment method for a broader range of conditions and populations. If you would like to learn more about SCP training for epilepsy and other neurological disorders you are encouraged to examine the studies, meta-analyses, and reviews cited within the text, as a majority are available for full text download online.



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Underlying Treatment Issues in Neurofeedback as Exemplified by Treatment of Seizure Disorders Part 2

Len Ochs, PhD

There are a number of unsettling aspects of working with people who have frank and latent seizures. Some of them are:

- Fear of seizures themselves.
- Fear of operating out of scope of practice.
- Fear of triggering a seizure.
- Lack of ability to estimate where the patient is in the course of his or her treatment.
- Lack of perspective about when to stop treatment if it looks as if there is no progress. Related to this is lack of perspective about what is happening if the patient is not responding.

I will discuss also how to:

- Give choices to patients about progressing through neurofeedback treatment.
- Recognize when seizures may be latent before the start of neurofeedback.
- Recognize when seizures look immanent but are really improbable.
- Anticipate with the patient the potential, and even probable course of treatment

This paper is based on my own work with dozens of patients with seizures, and my consulting with others who are working with people with seizure problems. I'll address these and other issues. I'll address both the unsettling aspects of these issues as well as ways to work with complex individuals with complex problems.

Fear of Seizures

Seizures, like death, can carry powerful and completely unnerving connotations. Many clinicians become unglued by partial or generalized convulsions, perhaps by the complete subservience to them of both patient and clinician. This goes well beyond being prepared and knowing how to manage these events. Even knowing how to respond to them, while helpful, doesn't quite capture the emotional reaction to them. To understand the quality and depth of your own reaction, look at the topic of "seizures" on www.youtube.com. Look at the huge variety of seizure videos. Notice your own reactions as you do. This will give you a sense of what you are up against on an emotional level as you think about working with seizures.

Scope of Practice Issues

To determine your scope of practice check with the licensing board that regulates your licensure. You must be a licensed healthcare practitioner to operate biofeedback systems (unless you are operating it under prescription from a licensed healthcare practitioner). You must have the specific education and training *beyond* your licensure coursework to validate to the outside world that you are legally and ethically capable of working with seizures. Finally, you will need malpractice insurance that covers both liability and *administrative problems*. Not infrequently, one's colleagues, through either misunderstanding or malevolence, question whether practitioners are operating within their scope of practice. You must be clear about what your operating board allows you to do and what your professional liability insurance covers.

Fear of Triggering a Seizure

Seizures can occur for a variety of known and unknown reasons, and are often exacerbated by a host of other factors. They rarely happen with exact predictability. I often state verbally and in writing that the prospective patient will definitely continue to have seizures once entering treatment. It's important to minimize any client perception that seizures will stop just by entering treatment.

Patients who have a seizure after a treatment will often wonder whether a particular treatment triggered the seizure. I set the expectation that we will at times apply the *incorrect* treatment which can trigger seizures if they are already a problem to a patient. I state this as a certainty for two reasons: (1) it does happen under certain circumstances; and (2) people's sensitivity can change so rapidly to the ordinary electromagnetic fields from all feedback EEGs, allergies, colds, hormonal changes, reactions to foods, and so on, that their seizure thresholds can rise and fall in unpredictable ways. So triggering seizures is unavoidable at times.

To someone having a seizure, having another one is rarely a problem. He or she knows what to do about them, and is usually matter of fact about them. To someone who *used to* have seizures but who no longer has them, having them once again is extremely worrisome. This also applies to someone who used to have tics, or migraines, or explosive problems, or odd episodes, after which they become profoundly tired.

Prospective patients need to be questioned closely about whether or not they used to have these problems. A previous history of these seizure-like problems with current CNS functioning problems means that there is a high probability that they will re-experience these seizures because they were incompletely resolved earlier. The seizures and seizure-like episodes will return for some brief period, and often become truly resolved in the process of fully increasing their functioning. At this time this is what our technology brings about. We need to tell this to prospective patients in order to give them the choice about whether to go along with our treatment approach. It is necessary for them to evaluate whether they have the familial, social, and medical supports to go through with this.

I tell them there is a very slim chance that they will have seizures or seizure-like episodes now if they never had seizures in the past. Or if they have them now they won't abruptly stop just by entering treatment. However if they *used to* have them, but have other problems of functioning now, the increases of functioning will probably come with a relatively brief return of symptoms. I stress that desire to increase function is the person's choice, and not a medical necessity. And if they come in for

treatment of their seizures, they must have had all relevant medical studies. Much of the rest of this paper is devoted to the course of treatment that they will experience for the treatment of seizures.

I then describe to the patient the probable course of changes that their seizures will follow over the course of treatment. These will be described later in this article in more detail. It is important for the patient about when, after a treatment, a seizure (or any symptom, for that matter) could be considered a "side effect" and when one might be considered not related to the treatment. I usually consider "side effect" to be limited to the 24-hours after treatment, and not afterward. Otherwise the symptom could be considered related to anything else that could occur in one's body. These might be hormonal shifts in both male and female, a sub-clinical cold that has yet to show itself, allergies, nutritional or medical changes, or in complex cases, a plausible and rapid increase in a patient's sensitivity that could later potentiate other factors.

Anticipating the Potential and Even Probable Course of Treatment with the Patient

One of the most powerfully comforting experiences for patients is the ability of the therapist to knowledgeably predict the course of treatment. To be able to do this reliably can also be one of the most supportive, uplifting, and nourishing actions for the clinician.

On the other hand while it is always possible to use phrases like "It's going to get worse before it gets better," more sophisticated patients will silently groan to themselves; you will lose credibility in their eyes by using such hackneyed phrases that they have heard before. The problem is that those who use such cute phrases probably don't think very deeply about what they are doing, or about the true complexity of working with difficult problems. For these patients, hearing these phrases has come to be an accurate predictor that they will probably not achieve significant reductions in seizure activity, except to possibly end up before another clinician who will once more use the same trite phrases.

However trite that expression might sound, it is, unfortunately true. So the task is to rephrase it so that it becomes fresh and more informative. Understanding the basis of this truth allows one to provide an explanation about why the annoyingly trite statements are true, and allows the patient to hear the statement in a different, more acceptable way. For instance, one might say "You may have seizures again for a relatively brief time if you have had residual functioning problems since your tics disappeared many years ago. But if your functioning problems came long after your tics disappeared—perhaps after a bump on the head—you probably will not have a period of seizures."

Using Similarities between the Mechanisms of Centrally Mediated Pain and Seizures to Anticipate the Course of Treatment

Understanding the course of different kinds of pain helps us to understand the course of progress with seizures.

1. Different types of bodily tissues tend to have different pain patterns. The pain from striated muscle wounds tend to reduce from the outside in, toward their centers, and gradually reduce in intensity.
2. Smooth muscle (vascular and gut) pain tends to remain high and intense as long as the wound is there; however as these wounds heal, the duration of the painful episode abbreviates each time the wound is irritated. However, if the irritation to the wound area is above a certain level then the healing of the wound stops, and the healing process starts again at the beginning. Vascular wounds, for instance need to be free from migraine for a period of 6 – 8 weeks for it to heal completely (depending on the location and the extent of the original injury). This is why someone with a cold or hormonal shift can suffer another migraine after a supposedly successful migraine treatment.
3. Seizures also seem to be a mixed type of wound. The tissues involved at seizure sites also seem to have a healing pattern, although not usually involving pain. Instead, the subjective and objective components of seizures tend to be related to the regional areas of the brain involved: consciousness or perceptual, vascular, motor, glandular, and combinations of the above. And as the areas involved heal, the extent and locations of the remaining wounds influence the kinds of subjective and objective symptoms and signs that arise. For instance, movements of all of the limbs may be involved in generalized seizures, formerly known as grand mal. The lips may turn blue; the skin may turn white. The neck may arch back. As the person heals there may be more localized movements, and less involvement of the limbs, head and neck; however *new* disturbances of exteroception may appear in the form of visual distortions (visual patterns, speckles, spots, waves, tunnels, and narrowed, darker vision) or sounds of just about any kind and intensity, gut reactions such as spasms and nausea, and so on. It is the transitioning among reactions of

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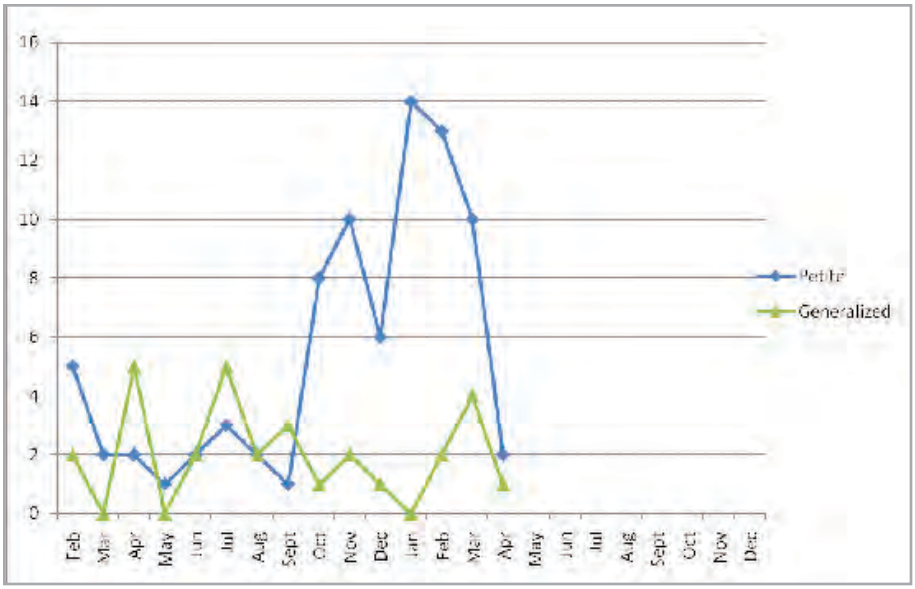

SeizureLatend and Actual Seizures

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different biological systems that signifies improvements in the status of the systems that maintain seizures.

4. It is common for some types of seizures to reduce while others increase in number. The graph in Fig. 1 shows the treatment effects of a man who entered treatment with a severe head injury in December 2009. He was at that point a paraplegic. A former athlete, he was hit from behind while riding his bike, just before he was to leave for Yale law school. He was engaged to be married at that time. In Fig. 1, the number of generalized seizures is reduced; while the number of petit mal increased each month until they finally began to drop. During this time his function has been increasing and continues to do so in cognitive and motor areas, such as calculations, social awareness, talking, and walking.
5. Unseen in the chart are decreases in the length of the postictal period. There is also much less time in altered consciousness, including fatigue and fogginess.
6. There is a toll to pay in decreased function when the brain, in an attempt to protect itself from the spread of seizures, neurochemically cuts its own connectivity to encapsulate and contain the spread of seizure. One would anticipate that re-activating communication of the brain with itself would once again lead to seizures. Instead, two huge events take place when connectivity is restored:

a. First, *there is no necessary increase in seizures when connectivity is restored*. This is a complete surprise. It would seem reasonable that the restoration of connectivity would also restore the ability of seizures to spread. In order to restore connectivity the brain has to release the suppression on the EEG that it has placed there to protect itself. Not only do amplitudes and standard deviations increase markedly and alarmingly, as if seizures were again immanent, but the raw EEG often shows spike and sharp waves, mixed with high amplitude slow waves. There may, on occasion, be increases in seizures, often of different types than those seen on entering treatment. But far from all the time. So there can be fewer seizures at the same time there is the appearance of higher amplitude, higher standard deviation, and spike and sharp wave mixed with high amplitude slow wave activity. This is often thought of as an unwelcome combination, and something to train away when using traditional neurofeedback.

b. Second, in the midst of this unwelcome psychophysiological picture, *there is almost always a significant increase in functioning and shorter postictal periods.*

When clinicians see the rises in amplitude and variability they call me in a panic. I always ask "But how is he/she functioning?" And in every case that I know about they have said something to the effect "Oh, he/she's doing very well, now." In fact, it is the accompaniment of increases in functioning with a "worse" picture of the EEG that accompanies releases of suppression and increases in connectivity. *If there were no increases in observed functioning, we could anticipate that seizures would return.*

Estimating where the patient is in the course of his or her treatment

The patient's treatment ends when he or she has had enough, not when the seizures are gone. What may be a goal at the start of treatment may not remain as the sole reason for treatment. For instance, there may a continued progression of reduced time in seizure, and much greater functioning. An example is being able to read again. At the same time the original grand mal seizures may have been translated into relatively brief sensory disturbances, and the patient wants to get on with life. On follow-up some weeks or months later – after neurofeedback treatment, the patient may be doing even better, with increases in visual processing, depth perception, visual sharpness, and field of vision.

Some of the questions to be asked are: (Answers of "Yes" come with reductions in seizure.)

a. Is the duration of the seizure or episode shortening?
b. Is the postictal period shortening?
c. Is there a difference in the specificity, complexity, and/or generality of the seizures with decreasing involvement?
d. Is there a reduction of time spent in the postictal period?
e. Is there a change in the aura, if there is one?
f. Is there an increase in general functioning level or in mood, cognition, motor integration, or energy?
g. Is the sense of humor returning? The sense of humor depends on the recovery of self trust.
h. Are there signs that one is able to make more of a commitment to a home, work, and relationship?

When to stop treatment?

a. When the *patient* decides to stop.
b. When there is a plateau of more than three days and there are midline deviations in amplitude and standard deviations, suggesting referral to an infectious disease specialist, or one who is expert in management of inflammation or toxicity.
c. When the therapist struggles for weeks to find symptom reduction. It is important for clinicians to not presume that they are missing something and to resort to more sophisticated means of assessment and treatment when stymied with plateaus during the application of neurofeedback.
d. When physician assessment of unrecognized medical factors brings the sought after relief (although finding physicians with the experience to undertake this assessment may be problematic).
e. When there is a reduction or loss of family or other close support and the experience of coming is too distressing.

In summary, when working with seizures it is helpful to understand some of the functional physiology underlying their perpetuation and spreading. It is useful to recognize the kind of dread with which seizures impact clinicians as a cue for obtaining training, supervision and consultation. And while the new information may or may not make a difference in the emotional baggage a therapist carries with him or her about neurofeedback treatment of seizures, the information can give the clinician a logic and cognitive structure to sustain him or herself when treating these dramatic events. Patients need us to have this information so that they can receive help that can make a difference for them.

BEST Practices: The Sterman-Kaiser Approach to Providing Neurofeedback

An Interview with Dr. Barry Sterman conducted by Michael Gismondi, LMHC

Introduction

Professor and researcher Dr. Barry Sterman is well known as a founding father of clinical neurofeedback for medical complaints. What has been shrouded in mystery, however, is precisely how he currently conducts neurofeedback in his private practice for epilepsy, head injuries and other neurological conditions. Due to a recent presentation to neurologists at the famed Cleveland Clinic, Dr. Sterman was urged to write a paper on the present status of the SMR training meta-research (See an excerpt from that article on page 9 this issue, Ed), as well as how his NFB techniques have evolved to the present day. The reference for this paper is Sterman (2010; in press), "Biofeedback in the Treatment of Epilepsy," *Cleveland Clinic Journal of Medicine.*

Clinicians interested in Sterman's actual NFB techniques and practices, and the rationales driving them, are frustrated to find such information to be readily available only in fragments; a paper or lecture about co-modulation here, a chapter on post reward synchronization there, and horribly out-of-date or incomplete coverage on his actual SMR training procedures in most instances. But recently, in conjunction with his software developer and long-time collaborator, Dr. David Kaiser, relationships have been forged with hardware developer Tom Collura of BrainMaster Technologies and Robert Lawson, who is resurrecting the vaunted J&J NeuroNavigator system. In addition, the Sterman-Kaiser team, collectively know as the Sterman-Kaiser Imaging Lab (SKIL), are releasing a sophisticated multi-channel NFB trainer to work with SKIL 3.0, the well known QEEG acquisition, normative analysis and reporting software. Finally, Sterman has been giving a series of trainings of his latest NFB techniques to neurologists from the Cleveland Clinic and Stanford Medical School. Clearly, it was time to corner Barry (no small feat in itself) and find out the whole story.

MG: What hardware and software do you use for your private clinic work?

MBS: Currently I use the J&J NeuroNavigator for both 19-channel QEEG and multi-channel SMR/co-modulation training, utilizing a bare minimum of four active channels, and usually more like 6, 8, 12 or more. I am using J&J's USE-3 software; I have it jerry-rigged to do what I want. Now as you know, the NeuroNavigator has not been manufactured and sold in many years, so we have done two things. One, we have joined forces with Tom Collura and Brainmaster to have all our present and future software run on the 19-channel Brainmaster Discovery. Second, we are working with Robert Lawson to resurrect and update the J&J NeuroNavigator, which is now called the NeuroSync system, and hopefully will be available later this year. There are at least two relatively unique innovations that recommend the new SKIL trainer software. On the most basic level, we insist that both the QEEG assessments and the subsequent NFB training are conducted using magnitude, not power, to avoid what we see as intolerable distortions in the frequency and amplitude data. You make mountains out of molehills when you use power and square the magnitude, and then you go on to train the mountains!

> I think of SMR this way; just as alpha gives an us a window into the status of the visual system, SMR gives us the "stand-by state" of motor system function, and that is critical to understanding any kind of seizure disorder.

MG: Even newer players in the QEEG arena like the Brain Resource Company are making that mistake with their QEEG reports.

MBS: Right, now it would be just as problematic for us to use Laplacian metrics for our SKIL QEEG analysis, which we usually do, unless certain frequencies are widely expressed, and then train in magnitude! Therefore, the new SKIL trainer is going to have the ability to train in Laplacian. In addition, we will be able to train the Brodmann area outputs. With Laplacian training you need four active channels to triangulate one target site, so you want ready access to 12 or 16 active channels at once. We like to look at and train four triangulated Laplacian sites at once. The SKIL trainer for the Discovery system will be released in time for the ISNR 2010 conference this September

MG: OK, allow me to play devil's advocate for a moment. Why should the average NFB clinician care about Laplacian and Brodmann area training, and in the process incur the expense of a 19 channel unit like the Brainmaster Discovery or NeuroSync that uses all 19 channels for QEEG mapping or training interchangeably?

MBS: Functional and dynamic localization, greatly increased precision in the comprehensive spatial localization of the *system* of sites and bands that need to be addressed at any given phase of treatment to recover function that has been lost or compromised due to lesion, head trauma, or seizure dynamics. But perhaps we are getting a little ahead of ourselves. Let's return to this question in a moment and let's start with the basics, which for me are the use of the QEEG to help organize the treatment plan.

Continued on page 35

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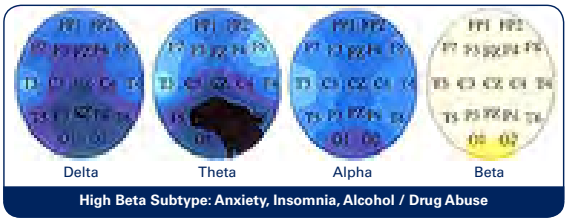
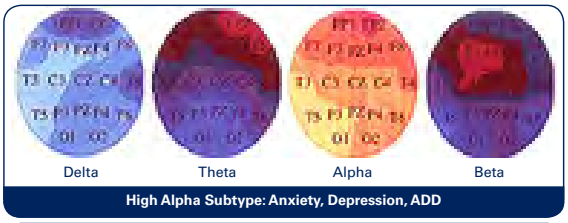
QEEG mapping service

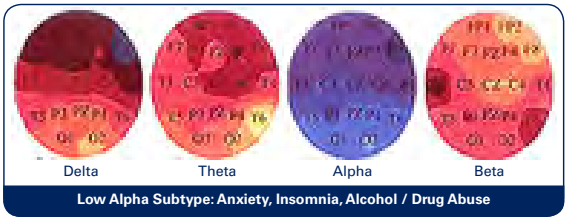
QEEG / TOPOGRAPHIC BRAIN MAPS:
Generalized Anxiety Disorder Subtypes



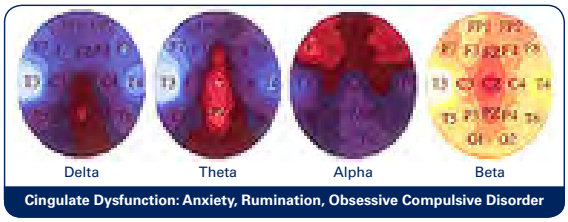
High Beta Subtype: Anxiety, Insomnia, Alcohol / Drug Abuse



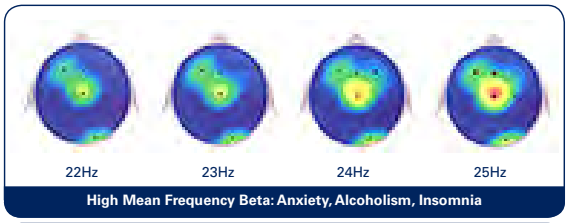
High Alpha Subtype: Anxiety, Depression, ADD



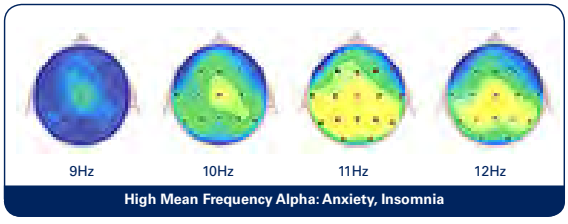
Low Alpha Subtype: Anxiety, Insomnia, Alcohol / Drug Abuse



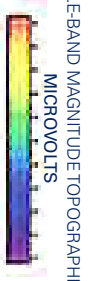
Cingulate Dysfunction: Anxiety, Rumination, Obsessive Compulsive Disorder



High Mean Frequency Beta: Anxiety, Alcoholism, Insomnia



High Mean Frequency Alpha: Anxiety, Insomnia



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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
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ROBERT L. GURNEE
MSW, BCIA:EEG, QEEG Diplomate, Director

SCOTTSDALE NEUROFEEDBACK INSTITUTE / ADD CLINIC
8114 East Cactus Road #200, Scottsdale, AZ 85260
Tel: (480) 424 7200 Fax: (480) 424 7800
Web: www.add-clinic.com Email: add@add-clinic.com
Established 1982

Best Practices
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MG: OK, back to the basics, how do you, or do you use a QEEG report to select where you will up-train SMR?

MBS: Let me say from the outset I consider SMR to be akin to getting the core temperature of a patient. I think of SMR this way; just as alpha gives an us a window into the status of the visual system, SMR gives us the "stand-by state" of motor system function, and that is critical to understanding any kind of seizure disorder. I tend to focus on C3/C4, but if a patient has had surgery or an injury at C4, let's say, I will probably be fighting tissue damage and fast wave activity there, so I want to look at an alternative way to reward SMR. So what I will do is co-modulate C4 with C3 and use different thresholds and bands and try to get a bi-lateral synchrony going. Keep in mind healthy SMR is strongly bi-laterally synchronous. Co-modulation, to me, is more than using a metric for a specific type of connectivity like say coherence. I want to get the outputs at two related sites modulated together. That,s all co-mod is anyway, coordinated spectral modulation.

MG: Well sure. Co-mod focuses upon the stability of *magnitude* fluctuation differences between two sites, just as classic coherence is the correlation coefficient of the stability of *phase* differences over time.

MBS: Yes, and because I am working with spectral outputs to begin with, which is part of what co-mod does, as opposed to coherence, since we are not interested in phase, if I can get two sites like C3 and C4 working together across the spectrum, wherever possible, I can stabilize it enough so that in a little while I can do SMR up-training despite the initial barriers. I want to link up the damaged site with the healthy contra-lateral one. And this in a way is our version of Z-Score training; I have three bands I am training for each site, I can reward SMR but at the same time determine what the exact peak slow activity is, which is usually injury or pathology related, and train it down, both at the site I am rewarding SMR, plus the peak slow activity that is prominent at neighboring sites. PLUS I actively monitor sites neighboring the injured site so that if the action shifts, if the pathology driven slowing moves from one site to the next, I am ready to now down-train it while I am rewarding SMR. And don't forget, I like to up-train SMR at two contralateral sites simultaneously if I can, so in a sense I have a multisite, multivariable training going on, akin in a way to z-score training. I am simultaneously rewarding both for co-modulation with a contralateral site and straight SMR magnitude increase at each site. I can do this easily with the SKIL 3.0 report which has single hertz bins, peak frequency data, and co-modulation data, between sites. However, it is critical to point out that the combined profile of frequency-amplitude changes sought among sites corresponds to a normal functional state that has been previously documented, and not just a list of sophisticated-sounding derived parameters.

MG: So, like Z-Score training, you are simultaneously training amplitude (magnitude) and connectivity (comodulation) variables at multiple sites. Four, or many more, sites at once?

MBS: No, four sites is our maximum for simultaneous training, and this is only progressively developed, unlike Z-Score training, I am explicitly rewarding the gradual, *successive approximation* increase in a pattern of multiple criteria, while at the same time manipulating the post reward delay of a new trial so that a discrete-trial structure is imposed. This is a very important feature of our work.

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MG: On that last point, it is interesting that the only user and developer of Brainmaster Z-Score training protocols that makes explicit this gradual increase in time above threshold is Tom Brownback, who pairs one tone for being, for example, one second above multiple thresholds, another tone for two seconds, and a church bell rings when you hit and maintain the target duration.

MBS: Time above threshold is important, but I like to break the learning process down even further. I am very interested in obtaining increases in *the rate* of scoring across both trials and sessions, as is indicated in learning theory.

MG: Clearly you use the QEEG report to tell you what slow and fast waves to inhibit as you reward SMR and SMR comodulation at contralateral sites. Do you use the QEEG to define the exact SMR band for a particular client?

MBS: Not really. The desired "resting but alert" state for the motor system is 12 to 15 Hz. Sure, if you want things more alert you will reward 14 through 18 Hz. But when you are targeting seizure process, you want to stabilize the motor or sensory-motor system first and foremost. And there is now a large amount of data to support that claim. We want to build a barrier against abnormal discharge impacting the motor pathways. So we get great results in eliminating the motor expression of seizure process, but patients can still have auras or perhaps little brief "episodes," particularly if they are temporal lobe seizure types. But the process doesn't spread, we don't see generalized seizures return.

MG: In listening to you, Barry, I am struck by the thought that what you are doing is very much *medical* neurofeedback, you seem to be working very hard to build a bridge to neurologists, if only they would listen.

MBS: Well, young neurologists are more open to what I am doing, and some are actually signing up for my trainings, particularly the ones who got their undergraduate degrees in psychology. I had dual training in physiology and psychology, so that really helps.

MG: Returning to how the QEEG informs how you treat a patient, I would imagine you don't look at the out-of-range slowing in a vacuum; you are at the same time looking at the site of injury or seizure

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activity to help you narrow your focus.

MBS: I can't help but thinking about what is causing what. With head injury you have the shearing and torqueing and stretching of the tissue and tracts, and the squeezing and compressing of the frontal lobes. Then there is the damaging of the micro-tubules in the axons that don't recover on their own, effecting the communication and information connections in the brain. You have to understand how white matter damage causes slowing in the EEG.

MG: Next question; you mentioned you sometimes suppress fast wave activity these days as part of your training protocol. Weren't you of the opinion in the past that the beta, especially as you go above say18-19 hertz, is just muscle artifact and doesn't exist?

MBS: No, I never said that. I said that one must consider where the fast activity is coming from on the head and the pattern of amplitude increase across frequency. Muscle activity tends to be localized to areas associated with underlying muscles and increases in amplitude at higher frequencies. Closed head injuries produce localized high frequency activity related to injury potentials and cortical irritability. The key is its localization and frequency pattern.

MG: In your recent Cleveland Clinic paper, you spoke of setting the post reward pause to two seconds. Do you find that to be optimal or even necessary for learning consolidation?

MBS: The reason for the post-reward pause is to impose a discrete-trial structure on the training. Earlier human studies in my lab looking at event-related EEG de-synchronization patterns established that the EEG activation associated with a learned response required 1.5-2.0 seconds to recover. It was assumed that this was the time required for essential response consolidation. We wanted to build that into our protocol.

MG: One last question. It has been pointed out that your work with laboratory cats featured the use of a bi-polar montage, and yet your current model for doing clinical work doesn't use a bi-polar montage, would you care to comment?

MBS: I'd be happy to do so. It's difficult to attach electrodes to a cat's ear. And it's difficult to put screws anywhere on the cat's head that are silent. So it was almost impossible to do referential recording, so we had to do bi-polar. Now, when we started working with human seizure patients, we continued using bipolar placements. Then we had a research grant proposal to the NIH shot down because bipolar recording prevents accurate localization of EEG characteristics, an objective we had specified. So, eventually we moved to linked ear and more recently, Laplacian, still seeking accurate localization.

MG: Darwinian adaption via "survival of the fittest".

MBS: Something like that!

MG: Thank you for your time.

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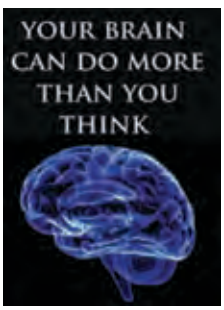

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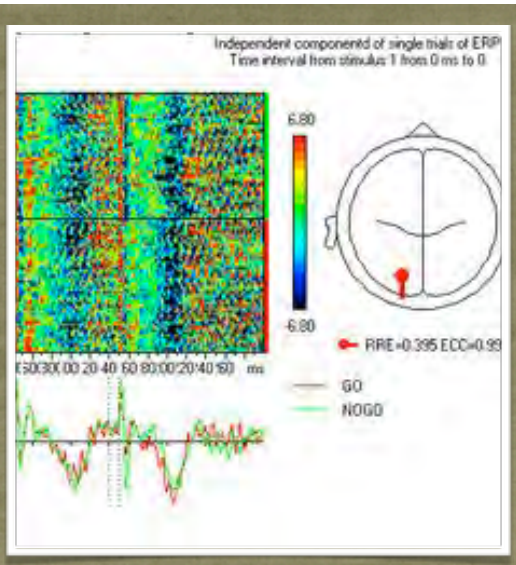

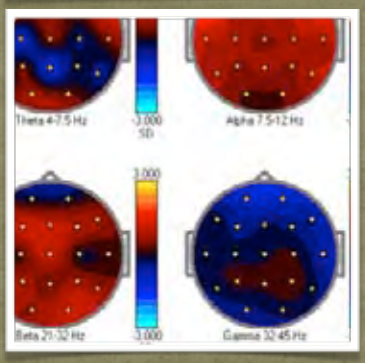

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