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RESEARCH RULES!!!

“Research is essential for continued improvement in the feedback game experience, as well as the entire field of neurofeedback/biofeedback. This is why Zukor Interactive is a recurring monthly financial supporter of the ISNR Research Foundation and why a portion of the sales of each of our feedback games will go to industry-sponsored research. We believe that anyone who cares about this industry will support research, either financially or by donating their time to research projects. Do you?”

– Samuel Turcotte, President & CTO, Zukor Interactive

ZUKOR INTERACTIVE AND OUR WORLD-CLASS SYSTEM PARTNERS SUPPORT RESEARCH

“I have seen the critical need for a greater body of neurofeedback research during my time at the VA and, more recently, in private practice. Having research to back up what I know to be clinically beneficial helps us establish the legitimacy of the field. Research also helps my clients to commit to the number of sessions they need for effective results. However, we desperately need more, larger and better-designed research projects that answer clinicians’ questions and support our clinical experience.”

– Allen Novian, PhD, LMFT, LPC, Novian Counseling & Neuroeducation
Chief Clinical Advisor to Zukor Interactive

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Online Supplement

The following material may be found in the NeuroConnections Supplement at http://isnr.org/neurofeedback-info/neuroconnections-newsletters.cfm.

References to Neurofeedback-Assisted Stroke Rehabilitation Revisited
References to Neurofeedback for Hemiplegic Stroke: A Case Report
References to Transcranial DC Stimulation

NeuroConnections is the official publication of the International Society for Neurofeedback and Research and the Association for Applied Psychophysiology and Biofeedback Neurofeedback Division. Opinions expressed herein are those of the respective authors and do not necessarily reflect the official view of ISNR or AAPB. ISNR and AAPB are not responsible for the products or programs of private companies advertised herein.
Letter from AAPB NFB Division President

The Dance of Discovery

We’re at the fiftieth anniversary of the publication of Thomas Kuhn’s *The Structure of Scientific Revolutions* (and it’s been about that long since I read it). In honor of the occasion, a new edition is being published, and it was just reviewed in the journal *Science*. The success of the scientific enterprise has been ascribed to the development of the “scientific method,” but the various attempts to define this more closely have not met with universal accord. Coming closest, perhaps, is Karl Popper’s formulation that the path forward in science is found by way of devising falsifiable hypotheses. The consequence is rather weak truth claims, with a proposition surviving merely by being not yet falsified. Thomas Kuhn urges that attention be paid instead to how scientists actually work, and that means looking at the history of science. No simple conceptual framework for the ostensible “scientific method” emerges.

What tends to drive the resolution of scientific puzzles by and large is the encounter of a “success” or a finding that demands attention for its novelty, perversity, or theoretical import. The finding is embodied in a new formulation, or paradigm, that at that moment may have very little going for it in terms of truth claims. It merely serves at that first juncture to structure further inquiry. Initial progress is typically made largely on an empirical rather than a theoretical basis. At this stage, it is far too early to require that the new paradigm make better predictions than the old or that it should meet the most rigorous tests of validity. For example, it was a long time before the Copernican/Galilean heliocentric cosmology yielded better predictions of planetary orbits than the existing Ptolemaic geocentric view, for which the epicycles had been worked out in great detail.

All that is required of a new paradigm is sufficient success to drive the spirit of innovation forward, and a climate in which divergent perspectives are tolerated. What distinguishes scientific enterprise then, is not so much what it mandates, but rather what it allows. The true scientific spirit gives wide latitude to the exploration of novelty. These explorations are then pruned—like a dendritic tree—by the successes and failures they encounter. Anomalies are undoubtedly prominent early on, but they are tolerated and held in conceptual suspension for eventual resolution. In the early phases of an emerging paradigm, even the criteria for what constitutes good evidence are up for debate.

Reference to the existing paradigm is of little help because the old and the new may well be incommensurable, possibly involving large conceptual shifts that alter the meaning of terms and engage a new vocabulary. Eventually the new paradigm may ascend to dominance, but this process will hardly ever be marked by the surfacing of incontrovertible evidence. “No process yet disclosed by the historical study of scientific development at all resembles the methodological stereotype of falsification by direct comparison with nature.”

All of this has just been recapitulated over the last half century in our field of neurofeedback. What started out naturally enough as a largely empirically driven effort was soon distorted, however, by encounter with the prevailing paradigm of pharmacology, allied as it is to enormous market power.

This tilted the playing field and subjected our nascent field to withering criticism and to premature “truth tests.” These tests all emerge from the existing paradigm, and hence belong to the category of incommensurables. The placebo-controlled design, the reversal design, and the sham-controlled design are all inappropriate to our method—even though these designs can in principle yield “success,” and have even done so. They all short-change the matter under test. In the nascent realm of personalized medicine and of individualized training, all group designs are inappropriate. This is most obvious in matters such as stroke rehabilitation, the theme of the current issue. Every case of stroke has its uniqueness. None can be treated in a formulaic way. More generally, all tests of a therapy that take the therapist out of the picture are inherently invalid. This should be insisted upon forthrightly, and we proceed from there.

It is those who are knowledgeable...
about the new paradigm, and who are practiced in it, that are the best judges of how its validity should be tested. Criteria of justification likely have to be tailored to the subject under study. All it takes is a look at history to see that this process has once again borne fruit. This field has been established entirely by empirical methods, by means of ad hoc truth tests, and essentially without recourse to the formal research methodology that is bequeathed to us from drug research.

With the growth of the field, however, the insistence on orthodoxy in research methods is even being internalized within the field. The result of this imposed orthodoxy is that innovation, broadly conceived, will not be nurtured within the field as it should be, and may once again have to seek safe harbor elsewhere, just as at one time the neurofeedback people had to separate from the AAPB to found their own organization in order to flourish.

And to top it all off, the palpable hostility within the field to emerging ideas is often justified in the name of science. How is it possible that the lesson of paradigm-breaking science has been so quickly lost to those who have just lived through it? Instead, the pioneers are pining to be admitted to the pantheon of orthodoxy. Having made their own contribution, they implicitly declare this scientific revolution to be essentially over.

The matter was put well in a letter to Science News back in 1990:

“Lest we become too arrogant, science needs to be reminded occasionally that it does not know everything yet. It is the duty of any scientist who discovers a new phenomenon to first prove to himself that it is real, and then, when this is done, to tell the world so that others can prove or disprove the reality.

“There are always some scientists who cannot accept change easily. This by itself is not a serious problem and is sometimes useful. However, a few of these people are too arrogant to allow the scientific method to follow its most productive course. They take the initiative in alerting the world that the proposed idea is not only wrong but being proposed by incompetent people. In the process, they frequently distort the available information to make their case. They sow confusion and raise issues that distract from the real problem of learning how nature behaves. When they have their way, the work to uncover the reality does not even start or, if started, is not published unless the results are negative.”


Siegfried Othmer, PhD
Letter from ISNR President

When you are courting a nice girl an hour seems like a second. When you sit on a red-hot cinder a second seems like an hour. That’s relativity. —Albert Einstein

Now that you have had your science lesson for the day...I find it a wonderful reminder of the hopefulness of humanity that someone of Einstein’s intellect still had a sense of humor, even about one of his greatest discoveries. It also reminds me of the many, many smart and generous people who make up the Society that is ISNR. Over my two tours of duty on the Board of Directors, and now as President, I have seen the time and energy that is committed to the organization with no pay and, as often as not, with no recognition. As is always the case, the organization is only as good as its people. And if that is indeed the measure, then we are a truly phenomenal organization!

However, the truth is, all organizations change. Members come and go. As we have noted this past year, some leave us permanently and we have only their gifts and legacies to remember them by. Some, because they have a shift in the priorities of their lives. One of those who has contributed much for a long time, who is among those making a shift in his priorities and entering a new stage of life, is Cory Hammond. If you were at the conference in Orlando you heard Cory being recognized for his many contributions as he was given the M. Barry Sterman Lifetime Achievement Award. What wasn’t mentioned at the time, was Cory’s intention to “slow down” and spend more time in activities related to family and friends. What this means for ISNR is that two important tasks are going undone. Cory has been the editor for the Clinical Corner in the Journal of Neurotherapy. Cory has done the challenging work of soliciting and editing the many helpful and enlightening clinically oriented articles over many years. I can’t say how many have told me this is often the first part of the Journal they turn to. The Journal of Neurotherapy is somewhat unique, in that we not only routinely publish scientific research articles, but also publish articles that are specifically relevant for the clinician. This is also a place where new methodologies, modes, and means of treatment are discussed for the first time.

A second critical task that has occupied Cory over the years, has been the regular management and updating of the Comprehensive Bibliography on the ISNR website. This has been, and continues to be, one of the most frequently referenced resources on the website. Professionals access it to see what research has been published related to a specific diagnosis or condition, and clinicians frequently refer clients to it as a demonstration of the legitimate research and information available on the effectiveness and use of neurofeedback.

The reason for my highlighting Cory and his contributions is to say we need the membership’s help. We need some folks to step up and volunteer to take on these tasks. The Comprehensive Bibliography is daily becoming less comprehensive as articles continue to be published, but there is no one currently actively involved in updating the list. There also is now no one actively soliciting and helping to provide clinically oriented articles for our Journal. So my call to the membership in this edition of the President’s letter is for volunteers. We need some help now! If you have any interest in helping with these important tasks, please contact me or any member of the Board, and we will get you connected to the right person. And that reminds me, did you hear the one about Tesla and the cat?

Randall Lyle, PhD

Welcome to our spring 2013 issue. In this issue we examine bio/neurofeedback applications in stroke rehabilitation, past and future, ranging from the well established to the cutting edge. The story begins with the discovery in 1830 by German physiologist Emil du Bois-Reymond of the electrical action potential emanating from a muscle during contraction. The first reports of successful biofeedback interventions in the treatment of stroke emerged in the early 1960’s, later bolstered by controlled EMG biofeedback trials by applied researchers such as Basmajian and Wolfe.

It would take another decade before neurofeedback pioneer Margaret Ayers began to direct her ample clinical talents and spirit of scientific inquiry to the problem of stroke rehabilitation, a history that her protégé, Penny Montgomery, shares with us in the current issue. Later in the issue, Corey Hammond weighs in with a contemporary case history, demonstrating the continued relevance of Ayers’ seminal work in his own successful treatment of a recent stroke survivor. Thanks also to Jay Gunkelman for a thoughtful commentary, highlighting the limitations of MRI or CT scan for understanding of the functions impacted by stroke, Jay discusses an integrated approach combining task activation ERP analysis with traditional qEEG to inform rehabilitation planning, and offers up the term iQEEG as an appropriate label for this approach.

In response, a compelling body of evidence of the brain’s capacity for neuroplastic repair following stroke, rehabilitation clinicians are increasingly turning their attention to neuromodulatory techniques for their power to bolster traditional rehabilitation outcomes. It is
Dear All,

Welcome to the spring 2013 edition of NeuroConnections. This has been a very complex and challenging time since the last letter. The forces of nature proved that there isn’t any easy method for predicting outcomes of wind and rain. The Eastern Seaboard, populated with millions of people, received a hit that continues to reverberate throughout our lives. Each day in our practices we work to alleviate the pain that the client is suffering. That is usually one person at a time. When there are many, many people experiencing the same pain and looking to

Roger Riss, PsyD

Letter from ISNR Co-Editor

Fitting that we acknowledge Len Ochs, a pioneer in these methods, within the current issue, with a remarkable tale of recovery as told by master LENS clinician Stephen Larson. David Siever follows with a brief tutorial on transcranial direct current stimulation (tDCS), an emerging neuromodulation method which, in early studies, appears to boost response to traditional stroke therapies by a factor of perhaps 20%. Already approved for clinical use in Europe, tDCS is enjoying an explosion of interest among US researchers for its potential to speed motor and language recovery in stroke survivors.

Lastly, we turn our attention to a family of hybrid devices, which are not only transforming the landscape of the contemporary stroke rehabilitation unit, but are also expanding the boundaries of bio and neurofeedback in ways that traditional practitioners may barely recognize as part of their own heritage. We will learn that biofeedback is showing up in surprising places in the modern rehabilitation clinic, providing patients with a means to direct and control their interface with therapy robots, virtual reality training environments, and external stimulation devices which rebuild connections between muscle and brain. In the process, biofeedback is leaving its comfortable niche of the past, and increasingly entering into the mainstream of the stroke patient’s and therapist’s daily routine.

We hope that you enjoy this issue as much as we have enjoyed bringing it to you.

Roger Riss, PsyD

Dear All,

Welcome to the spring 2013 edition of NeuroConnections. This has been a very complex and challenging time since the last letter. The forces of nature proved that there isn’t any easy method for predicting outcomes of wind and rain. The Eastern Seaboard, populated with millions of people, received a hit that continues to reverberate throughout our lives. Each day in our practices we work to alleviate the pain that the client is suffering. That is usually one person at a time. When there are many, many people experiencing the same pain and looking to
us for the answers, the balancing of our own lives takes on a more difficult arc. And then the Sandy Hook Elementary School shootings drove us even deeper into the examination of our lives, focus, and mission.

I do not have any idea what your thoughts are on the mental health system, especially in the United States, I can only give you mine. We, in the Neurofeedback world, have at our command such powerful means of helping those in deep pain and to often quickly bring them to a balanced, productive state. We are a tiny group when one compares our numbers with the rest of the mental health and medical groups. At the same time, we do seem to do a heck of a lot of good for a formidable number of people. Recently, a client said “I think this instrument (happened to be Neurofield) should be in every school and used extensively.” It isn’t that the means to help people who are in such pain that they must destroy others is not available; the methods are available. Connecting the methods, clinician, and patient is where the journey gets screwed up it seems. I am sure many of you work with teachers, school psychologists, and counselors, and have witnessed firsthand the utter nonsense of keeping from them the diagnosis of the child and the methods being used to assist the child. We know this is tied to the old studies of expectations of teachers (they apparently cannot be trusted with the true diagnosis of a child). In the medical field we are still trying to get the idea that the mind and body are interactive and the whole person needs to be treated at the same time; well, in the educational field the concept that the educational needs are completely separate from the mental health needs of the child is likewise vigorously defended. Thankfully, in our field we treat the whole person and that means we are pioneers in assessing and working with any number of medical, educational and even work personnel and providing the training that changes the brain and whole person to a more productive and happier being. We must continue to find ways to work with more clients and more systems that interact with the people who are in psychological and medical pain. I firmly believe there is always a solution; we just have to keep trying until we find those that work.

I acknowledge this letter is not my usual, in which I sort of give you a highlight of the articles in this edition. I could not ignore the two major incidents that have marked the last months here in the East and in the world and try to communicate the need that we have to fill.

Sincerely,

Merlyn Hurd, PhD,
BCN Senior Fellow

AAPB Earns Five-Year Reaccreditation!

AAPB has long been accredited by the American Psychological Association (APA) as a provider of continuing education credit. In its recent renewal application, AAPB was awarded the highest level, a full five-year accreditation, for its CE provider status!

So, what does this mean to you as a member of AAPB and the biofeedback Community? It means that AAPB is in the position to offer all of its conferences, workshops, webinars, the AAPB Home Study program, and other educational programs to provide continuing education (CE) credits in accordance with the APA standards. It also means that AAPB can act as a sponsor in accrediting educational programs for APA-recognized CE credits offered by other organizations.

Many of the AAPB state and regional chapters have taken advantage of this benefit in the past. We hope that more will participate going forward as a result of this status. One of the most important aspects of this accreditation status is that you have the opportunity to attend some great educational programs and use the CE credits to maintain your license, BCIA certification, and other CE requirements.

It also means that AAPB can serve as a sponsor in providing CE credit to organizations outside of its own organizational structure such as ISNR, BCIA, and other qualifying organizations. To do so, the organization must submit an application, similar to the process that would be utilized by APA, but at a much reduced cost.

This reaccredited status is provided based, in part, on the high quality of the programs that are offered by AAPB. We are very pleased with this recognition and hope that it will translate to new growth for the educational opportunities that we offer! So, if you read this article is before the March 2013 AAPB Annual Research Conference, and if you have not yet registered, we encourage you to take another look. The highlyrated educational content is such that it should not be missed! Of course, if you read this article after the Portland meeting, be sure to watch the AAPB website at www.aapb.org for our ongoing series of webinars throughout the year! Past webinars have covered such important topics as ethics, PTSD, stress management, addiction and compulsive behavior, anatomy, HRV and others. Be sure to check the websites of the state and regional biofeedback societies for programs that they offer with CE credit as well.

As this reaccreditation attests, quality education is paramount within the goals and objectives of AAPB. We look forward to continuing to meet your educational needs.

David L. Stumph, IOM, CAE

Membership Benefits Based on Three Key Values: Information, Opportunities and Access

ISNR’s membership persisted in building momentum as 2012 came to a close. Membership grew by six percent this year, including a large contingent of professionals from over 30 countries, representing the highest membership rate in our history. It’s clear that as we evolve and grow, ISNR’s members will continue to regard us as their professional home.

Today ISNR is a leader in clinical practice, educational applications, and research in applied neuroscience. We stand for scientific innovation, communication, and collaboration. We are an organization that engages members, encourages understanding of brain physiology, and informs the public about improving lives through neurofeedback. We are leading advocates in explaining the role brain regulation modalities play in affecting behavior and in pursuing research into better understanding of brain function.

We are dedicated to building a strong and diverse membership that’s committed to clinical and ethical guidelines for the practice of applied neuroscience. We are endeavoring to evolve and advance the organization to better serve our worldwide membership by providing information resources for the public and professionals, and by promoting scientific research through our peer-reviewed journal.

We know how important our programs are to our members and how critical they are to our mission of promoting the self-regulation of brain activity for healthier functioning through neurofeedback and biofeedback.
Jonathan E. Walker, M.D.

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

EEG / QEEG interpretations, analyses and reports with protocols using the modular activation / coherence approach to allow practitioners to achieve superior results

Dr. Walker personally reads each QEEG Service includes phone consultation with Dr. Walker

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This summer, the ISNR Board of Directors commissioned a comprehensive membership survey to evaluate whether current programs and services adequately support member needs and expectations. The survey included assessments of membership benefits, the annual conference, and publications, as well as educational and networking opportunities.

The results from this survey are clear. Our members overwhelmingly report satisfaction with their ISNR membership benefits, based on three key values: information, opportunities, and access.

**Information**

ISNR members appreciate our long-standing tradition of providing the most current research in the study of neurofeedback. The *Journal of Neurotherapy* draws upon divergent expertise to report relevant findings from the diversity of disciplines and to offer interpretive commentary. ISNR members receive:

- A free subscription to *The Journal of Neurotherapy* online and in print.
- Online access for all *Journal* issues—volume one through the current issue.
- The opportunity to publish research in the Journal.

Our members depend on ISNR for the information they need—whether it’s the latest research, news, or techniques. One of the most important benefits of membership includes:

- A free subscription to our newsletter, *NeuroConnections*—a joint publication with the AAPB Neurofeedback Division.

**Opportunities**

ISNR’s annual conference brings together hundreds of professionals from more than 30 countries for thought-provoking presentations, educational programs, and certification programs. ISNR members benefit from an enhanced professional life and also from money saving deals that include:

- Reduced rates to ISNR annual conferences and workshops.
- Discounts on ISNR-published books, DVDs, and annual conference presentations.

The professional standards of our annual conferences, workshops, and educational tools are widely respected because they promote the highest standards of professional practice, ethics, and education.

**Access**

In a competitive environment, one of the best pathways to career advancement is to be recognized by one’s peers. ISNR members enjoy a variety of leadership-building capabilities through participation in the governance board, journal peer review, the newsletter, and special interest groups. ISNR membership makes the connections that matter by providing:

- Opportunities to connect with the most prominent professionals in the field.
- Access to an online member directory.

**Join**

I encourage you to renew your membership and to continue your involvement in our dynamic organization in 2013. Membership dues allow us to provide exceptional, intellectually stimulating programming.

If you are like me, renewing your membership can easily be forgotten amongst all the other tasks on your “to do” list. So take a few minutes now and renew your membership for 2013 by logging on to the ISNR store at: http://isnr.mybigcommerce.com/categories/Membership-%28Join-or-Renew%29/

If you have already renewed your membership, please spread the word and tell your colleagues about the many benefits of ISNR membership and how it can help enrich their career in the field of neurofeedback.

**Want more information?**

Detailed membership benefit information may be found at: http://www.isnr.org/membership/benefits.cfm. Joining is easy. Online applications are available at: http://www.isnr.org/membership/member-applications.cfm. You can join online or mail your application to ISNR, 1350 Beverly Road, Suite 115, PMB 114, McLean, Virginia, 22101. If you still have questions, please contact me at cyablonski@isnr.org or call 703-848-1994.

**On the Horizon**

In 2013, I hope you will make plans to join us for the 21st Annual Conference in Dallas, Texas from September 18–22, with pre-conference workshops September 16–18 and free vendor seminars on September 22.

I value your input, your ideas and your suggestions. This is your organization and your involvement is essential. So please feel free to contact me to discuss any issues. My email is cyablonski@isnr.org.

With best wishes,

*Cindy A. Yablonski, MBA*
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Patient Self Reports: Alpha-Stim® vs. Drugs

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<th>Alprazolam (N=836)</th>
<th>Alpha-Stim (N=114)</th>
<th>Alpha-Stim (N=398)</th>
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<td>77%</td>
<td>78%</td>
<td>81%</td>
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Percent of Patients Reporting Improvement

Patients who reported a positive response according to WebMD Drug Surveys and Alpha-Stim® Service Member and customer surveys. Alpha-Stim® Data from 1991 Military Service Member Survey (N=132) and Alpha-Stim® Patient Survey (N=2,740). Conducted by Larry Price, PhD, Associate Dean of Research and Professor of Pharmacometrics and Epilepsy, Texas State University, Pharmaceutical Survey Data from www.WebMD.com/drugs. Accessed on October 26, 2011.

In the USA the FDA restricts this device to sale by, or on the order of a licensed practitioner. It is sold over-the-counter throughout the rest of the world. Side effects occur in less than 1% of people and they are mild and self-limiting consisting mainly of headaches and skin irritation on the ear lobe electrode site.

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A stroke is defined as the loss of brain function due to sudden change in the integrity of the blood supply to the brain. One of two types of stroke may occur; Ischemia (lack of blood flow) is the result of a blockage, while hemorrhage (bleeding) results from an open or ruptured blood vessel. The affected area of the brain cannot function, which results in the inability to move limbs on one side of the body, loss of part of the visual field, or inability to form speech. (Domnan 2008)

Stroke symptoms differ depending on the area of the brain affected. The major arteries involved in stroke include the anterior, middle, and posterior cerebral arteries. Ischemia or hemorrhage usually occurs on only one side of the brain and the symptoms give clues as to the side involved. If the stroke is in the right hemisphere, there is often paralysis on the left side of the body, memory loss, difficulty with vision, and a change in behavior toward a more reactive and impulsive style. Left-sided strokes result in paralysis on the right side of the body, memory loss, speech and language problems, and a more cautious behavioral style. (American Heart Association, Inc. 2012)

According to the American Heart Association, a stroke occurs approximately every 40 seconds in the U.S. That means about 795,000 Americans suffer a new or recurrent stroke each year. Stroke is the number four cause of death, with more than 137,000 people dying of stroke per year—or about one every four minutes. (American Heart Association, Inc. 2012)

Brain wave activity and patterns are seen in EEG recordings. EEG changes after stroke tend to be focal, with generalized slowing and loss of normal background activity. A reduction in overall amplitude with sharp activity and intermittent delta activity typically occurs. (Faught 1993)

The screen shots below of EEG records were obtained using a Neuropathways EEG Imaging machine.

By contrast, note the increase in spikes and sharp waves in the EEG shown below resulting in higher slow wave amplitude.

According to the National Stroke Association, it is a myth that only older adults have strokes. Although people over 65 are at higher risk of stroke, a person of any age can have a stroke, including teenagers, children, newborns, and even unborn babies. Estimates vary, but it is believed that stroke affects about 6 in 100,000 children. (Fact Sheet: strokes in infants and children. American Heart Association 2012).

Changes in the EEGs of children with strokes resemble those of adults with strokes, but with an even greater increase in slow wave amplitude. This may be due to the fact that healthy
Neurofeedback has been demonstrated to bring about a permanent improvement in brain function and symptoms following stroke in both adults and children. Below are two EEG recordings from the same 70-year-old man. The first is post-stroke and before neurofeedback training. The second shows EEG changes after seven months of neurofeedback training at T4T6.

That children can also benefit from neurofeedback training was reported in an early 1995 study by Ayers. Six children, ages seven months to fifteen years, received EEG feedback and showed improvement in the areas of foot dorsiflexion, concentration, short-term memory, and had fewer mood swings compared to a control group of six age-matched children. (Ayers 1995)

In an interview by the Nielsen Recovery Group in 2006, Ayers was asked about her controversial use of suppression (rather than enhancement) neurofeedback protocols. Her response was based on a prescient understanding of the brain’s function as primarily inhibitory in nature. She asserted that inhibition is the paradoxical behavior necessary for life. Every field, from engineering (with inhibitory gates) to biology and physics (with inhibition required to maintain homeostasis) relies on this behavior. She explained that all sensory information arriving at the sensory cortex passes through the sensory nuclei on its way to the cortex. Neurons within these nuclei receive inhibitory messages from cells with short axons. This is called surround inhibition. It is this inhibition that sets the stage for homeostasis. Sohn and Hallett describe surround inhibition in a 2004 article. (Sohn and Hallett 2004)

While there is some disagreement as to where sub-cortical GABA interneurons develop, it is agreed that they regulate both the development and function of the cerebral cortex. (Wonders and Anderson 2005).

When there is damage in the cerebral cortex following a stroke, the center of the damaged area demonstrates a reduction in beta activity and an increase in slow wave activity in the theta (4–7Hz) and delta (0.5–4Hz) frequency bands. This slowing in the damaged area is accompanied by an increase in fast beta activity in the area peripheral to the damage which reflects the brain’s attempt to bring the frequency of slow wave activity back to a more normal and functional speed. There is also a significant increase in inhibition that interferes with the re-establishment of beta activity in the damaged area. Thus, EEG changes following a stroke include an increase in background slow wave activity, spikes, and slow waves in the theta and delta frequency bands.

Using mice as subjects, Clarkson, et al, have described the role of inhibition in stroke recovery. Following the strokes in mice, tonic neuronal inhibition increased in the peri-infarct zone. This increased tonic inhibition was mediated by extrasynaptic GABAA receptors and was caused by impairment in GABA function. To counteract the heightened inhibition, the authors administered in vivo a benzodiazepine inverse agonist specific for α5-subunit-containing extrasynaptic GABAA receptors at a delay after the stroke. This treatment produced an early and sustained recovery of motor function. Genetically lowering the number of GABAA receptors responsible for tonic inhibition also proved beneficial for recovery after stroke, consistent with the therapeutic potential of diminishing extrasynaptic GABAA receptor function. By diminishing inhibition, beta activity increased, thus establishing more normal function. (Clarkson et al 2010).

This study shows that after stroke, motor function is improved with biochemical regulation of inhibition. Motor function is also improved with neurofeedback training. Slow wave suppression protocols train the brain to suppress EEG activity that should not be present, such as excessive theta that inhibits normal beta wave activity. It has been demonstrated that a reduction in theta activity results in an increase in the production of beta frequency activity and improves motor function. (Putman 2001). Noting that the same outcome results from both ap-
proaches, it is possible that both perform the same function. Neurofeedback training actually regulates inhibition, making homeostasis possible and indeed probable. It becomes even more likely when we add an understanding of the role of thalamocortical loops in neocortical dynamics. Thalamocortical loops are described as mechanisms through which the thalamus functions as a hub from which any site in the cortex can communicate with any other such site or sites. (Llinas 2003).

Lubar has described the role of neurofeedback according to the principles of neocortical dynamics. (Lubar 1997). He states that neurofeedback is a technique for modifying resonant loops in the cortex that are both excitatory and inhibitory. An understanding of this mechanism can explain the results of neurofeedback training. He further explains that based on this concept, one need not train more than one or two channels of EEG to bring about therapeutic change utilizing resonant looping already set up in the brain. We are in a very exciting time in the understanding of cortical mechanisms and the role of neurofeedback in restoring brain function. In only eighty-eight years, we have come from the very first EEG recording made by Hans Berger (1924), to now understanding cortical mechanisms and the role neurofeedback plays in changing brain activity. Information has grown exponentially in the past few years, making those of us who have been in the neurofeedback field since its beginning, almost 50 years ago, wish we could continue to be a part of this exciting adventure. It is to be hoped that those who remain in the field fully appreciate they now have the knowledge and technology to bring about extraordinary changes in the brains of those they serve. Referring to neurofeedback, Ayers’ concluding remarks in the Nielsen interview reflect her belief that neurofeedback “… is the greatest tool in history.”

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Dr. Penny Montgomery began her career in biofeedback in 1968 at the University of Texas Health Science Center. She received her Ph.D. in Behavioral Medicine Psychology from the University of North Texas, Doctor of Alternative Medicine from Rio Verde University, and is a BCIA Senior Fellow.

She co-authored with Dr. Kenneth Gaarder Clinical Biofeedback: A Procedural Manual for Behavioral Medicine and, with the late Dr. Margaret Ayers, with whom she worked for 15 years, Whispers from the Brain. In addition, she has published more than 30 research papers in the area of biofeedback, lectured in the visiting professor program at the Cleveland Clinic, has been a consultant to the Department of the Army, and has lectured and consulted extensively over the past 40 years.

Dr. Montgomery may be reached by email at pennymont@mac.com.
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Neurofeedback for Hemiplegic Stroke: A Case Report

D. Corydon Hammond, PhD

Abstract
This paper describes the neurofeedback treatment of a woman seven years after a stroke left her hemiplegic. Marked improvement was obtained.

Background
The patient was a 45-year-old female who, at age 38, had a large brain aneurysm which measured just over 2 cm and arose from the broad base at the bifurcation of the right internal carotid artery. It was found on a CT scan, and surgery was done in which the aneurysm sac was punctured without bleeding. After surgery the bone was reattached and the burr holes covered with Silastic burr hole caps and a titanium mesh was screwed to the area of the bone where the decompressive craniectomy had been done, to fill that site.

The surgical discharge stated, “At surgery, there was some concern that there was slight kinking of the origin of the middle cerebral artery. I studied this and felt that I could not improve the situation. Postoperatively, the patient did not move her left side. An immediate angiogram was obtained showing that the middle cerebral artery did not fill. At this point, because of the length of time that had passed between the clipping and the angiogram, I did not feel that I could improve the situation by returning the patient to surgery and removing and re-attaching the clip. The patient developed a dense left hemiplegia. She was kept intubated for a few days. A postoperative CT scan showed a right middle cerebral artery infarct with mild mass effect.” After surgery, the patient remained in the hospital for 6 weeks, then went to rehab and physical therapy. Over a lengthy period of time she had overcome significant swallowing problems and facial droop.

Seven years post-stroke, the patient was brought to my office in a wheelchair by her elderly mother. She has severe spasticity in her left hand and arm, with them in a tight spasm against her chest and her hand tightly clenched. Baclofen was not being used at that time in her treatment, and the neurosurgeon had many years previously suggested cutting nerves to relax her arm, but she had resisted having this done. She could not move her left arm or hand, and had only very slight ability to move her left leg, ankle, knee, or toes. She was experiencing some short-term memory problems and cognitive decline, and had basically given up reading, apart from the TV Guide. She spent 95% of her time in a wheelchair, but a physical therapist had taught her, with a brace on her left ankle and the use of a cane with a large, square base, to throw her hip. Thus she could awkwardly get around a little bit in her kitchen. She was taking three pills a day (which cannot be identified at this time in her record) to help with bladder control, but she still experienced incontinence four to five times daily. Due to the specific nature of her disability and the costs involved, it was decided to begin treatment without doing a quantitative EEG.

Treatment
Treatment was done using a Neuropathways EEG neurofeedback unit which had a sampling rate of 250,000 samples/second, digitally filtering the EEG signal, with a common mode rejection ratio greater than 110dB wideband and greater than 120dB at 60 Hz. The treatment plan was based on what I had learned in workshops and personal mentoring from one of the first pioneers in neurofeedback, Margaret Ayers (1999). First, we focused on the spasticity in her left hand and arm. This was done by using a sequential montage and placing electrodes about 1¼” in front of and behind C4, the area of the homunculus associated with these areas of the body. The sessions were 30 minutes long, inhibiting 4–7 Hz while reinforcing 15–18 Hz only very mildly, because too strongly reinforcing beta can produce negative effects (Hammond & Kirk, 2008). Because the patient’s elderly mother drove her to appointments from three hours away, we conducted two sessions in a day, once a week, with a three- to four-hour interval between sessions. Over the course of six or seven sessions, her arm relaxed and moved from a tight spasm against her chest to resting comfortably on her lap, and her hand opened and was relaxed. She expressed profound relief. Further treatment sessions over this area did not, however, result in functional use of her left hand and arm.

Treatment next focused on improving mobility. To improve functioning in her left leg, a sequential montage was again used, placing electrodes over the area on the homunculus associated with the legs. Thus, an electrode was placed about ¼” in front of and behind Cz and we trained with the same inhibit and reinforcement parameters. This protocol was used in the morning sessions. In the afternoon sessions, we used a protocol designed to improve physical balance (Hammond, 2005). This protocol utilizes a sequential montage with the electrodes placed low on the back of the head directly below O1 and O2, positioned so that the bottom of the electrode comes to about the middle of the inion ridge, but does not go below it. Once again, 4–7 Hz was inhibited while mildly reinforcing 15–18 Hz. The EEG in this unique location is quite small, and it is important to watch for EMG contamination and encourage the patient to keep the neck relaxed.

Treatment consisted of a total of 50
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sessions. At the conclusion, the patient was mobile around the house without her wheelchair or the aid of a cane; also, she was regularly going on walks with her mother of up to one mile, where she would take her cane. Cognitive functioning improved as well, and she began reading regularly again. As a control procedure in her treatment, I had not told her that the balance protocol also could produce improvements with incontinence or swallowing problems. At the end of our treatment she spontaneously expressed delight in the fact that her incontinence had improved. Now, instead of having 4–5 “accidents” daily, incontinence was a rare occurrence and she had reduced her medication for bladder control from three pills daily to one pill a day. At last contact the patient was working with a vocational rehabilitation counselor pursuing vocational retraining.

**Conclusion**

Up to this time, neurofeedback literature on stroke rehabilitation has been limited to case studies (Ayers, 1995, 1999; Bearden, Cassisi, & Pineda, 2003; Cannon, Sherlin, & Lyle, 2010; Doppelmayr, Nosko, Pecherstorfer, & Fink, 2007; Hammond, 2005; Putnam, 2001; Rozelle & Budzynski, 1995; Wing, 2001). These reports appear very encouraging, but further controlled research is needed to determine how efficacious neurofeedback can be with this population.

**About the Author**

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References are available in the supplement at: [http://isnr.org/neurofeedback-info/neuroconnections-newsletters.cfm](http://isnr.org/neurofeedback-info/neuroconnections-newsletters.cfm).

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**Functional Analysis in Stroke**

*Jay Gunkelman*

In stroke it is often important to document the areas involved with the cerebrovascular accident (also called a “brain attack”), whether hemorrhagic (bleed) or thrombotic (embolic).

The medical image from an MRI or CT scan of the client’s head will show the primary area involved, but it is far from a description of the functions impacted. A local lesion may interrupt a pathway allowing distant locations to be networked together. These connectivity disturbances are a functional change in areas that often have no local indication of disturbance.

In the EEG, slowing in the general area of white matter disturbance from a stroke may be seen, though this is not specific to the function disturbed, or even specifically diagnostic of the stroke, as tumors and other lesions can cause a similar change in the EEG.

*A local lesion may interrupt a pathway allowing distant locations to be networked together.*

The default mode network function of the brain is seen at rest with eyes open or closed, but to see function more fully; you also look at the brain on task. Though some databases have added tasks, they are poorly constrained, and may merely be mental math or reading with no control over the difficulty of the material. In our work we use specific Go-No-Go tasks, and with these standardized tasks a different picture can emerge. The ongoing EEG is recorded on these Continuous Performance Test (CPT)-type Go-No-Go tasks, but the EEG is randomized by the timing and it is “averaged out” by the analysis of the specific Go or NoGo events.

The Event-Related Potential (ERP) actually can look at the full range of systems used in the CPT. It starts with sensory detection of early event-related changes in potential, including thalamic and cortical arrival of the specific sensory inputs (both auditory and visual), as well as the sensory processing of both the dorsal and ventral processing stream as the client detects both the “what” and the “where” of the stimulus. The detection of novelty or distractors, the motor engagement or inhibition of motor responses, as well as the comparison of the motor response or inhibition to the model of expected behavior (error detection) all are part of the functional analysis of the qEEG information.

In stroke, the specific steps of administrative response to stimuli can be tracked, including subtle changes in the basal forebrain and cingulate seen in error detection and working memory of the task, as well as thalamic relay and surface cortical functions required for proper CPT response behavior, whether inhibition of a response or the actual execution of a required motor response.

The functional qEEG is a more complete evaluation than a resting-state EEG evaluation using simple eyes open or eyes closed. The detail of which neural pathways are involved in both motor responses and also withholding of responses can be specifically evaluated. The EEG and ERP together form the fQEEG, which is much better than either EEG or ERP taken in isolation.

The ERP is much more diagnostically specific than the EEG, but ERPs are often less useful in designing a clinical intervention than the EEG. Together they form a powerful evaluation and treatment design tool: the fQEEG.
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Stroke, according to the CDC and the American Heart Association, is the third leading cause of death in the United States. Strokes happen every 45 seconds, and six and a half million Americans are currently living with only part of their brain functioning due to stroke. The causes, and the varieties, of stroke are legion, but the only feature on which medical authorities agree, is that they are generally caused by a malfunction of the cardiovascular system in relation to the brain.

Our brains consume about a quarter of the entire energy of the human body, usually delivered in the form of glucose and oxygen, by the circulatory system. The web of neurons, and the glial matrix in which they are embedded, are as exquisitely delicate as they are highly functional. Even small and brief failures of the delivery system, called ischemias, can lead to cognitive impairments that may be permanent, while larger blood clots that deny oxygen and nutrients to key areas of the brain, lead to massive and major debilities: loss of language, memory, motor functioning, and massive paralysis. Even more serious are aneurisms, bleeds that may affect whole brain regions or entire hemispheres; or, when they shut down deeper areas that control vital functions, simply cause death.

The stroke that is the major subject of this article was extremely massive, enough to be life threatening, and take out most of Michael Schacker’s left hemisphere, including Broca’s area, which generates speech, and much of the left motor cortex, leading to paralysis of his right hand and leg. With the permission of Barbara Dean Schacker, Michael’s wife, I have used the title of her website: “Stroke Family,” because when a loved person is “stroked,” the whole family also is “stroked,” or traumatized. Barbara is also a co-author for this article, and we begin with her narrative of how she developed the method called The Sensory Trigger that she evolved to help her own father with his stroke many years ago. (So, as well as Michael’s dramatic recovery, this article brings in the “back-story” of a remarkable, loving, and resourceful spouse.) My other collaborator on this article is Dr. Victor Zelek, the neuropsychologist at The Northeast Center for Special Care, who first brain-mapped Michael and saw to his care during his year and a half at this excellent and groundbreaking facility for care of the brain injured.

In 2010, Dr. Zelek and I presented Michael’s case at the International Society for Neurofeedback and Research (ISNR) annual conference in Denver, along with the brain maps and clinical data accompanying this article. The room was packed with neuroscience professionals, including Norman Doidge, author of The Brain that Changes Itself, and I was able to have a really interesting discussion with both Dr. Doidge and Harvard’s Dr. Alvaro Pascual-Leone, one of the founding researchers of the (once controversial, now widely accepted, theory of “neural plasticity,” about how neurofeedback has a special contribution to make to the restoration of brains damaged by traumatic brain injuries (TBIs) of any kind. Michael’s case gripped the audience because of the extent of his intellectual and creative prowess before the stroke: writer, composer, musician, political activist. With a little help from his friends (and some neurofeedback), Michael Schacker may win his way back to the richly brilliant cognition he had with a whole brain instead of half. Before we turn to Michael’s breathtaking recovery, let us look to the back-story.

Barbara Schacker

That day, I had no idea that my life was about to change forever. I remember standing in left field playing softball during recess on a beautiful spring day in Nebraska when my teacher suddenly appeared and motioned me off the field. “You have permission to leave school immediately,” she said gravely. “Your sister is waiting for you in her car.” Climbing into the car, I immediately knew something terrible had happened. My sister said, her voice trembling, “Daddy has had a stroke. They don’t know if he will live. He is in the hospital with Mom.”

When we arrived at the hospital in Lincoln, my sister and I rushed to the entrance. At the door, I was asked, “How old are you?” “I am 13.” I replied. The nuns declared, “She is under age, she can’t come in. You have to be 15 to enter the hospital. That is the rule.” My sister took me back to the car to wait while she rushed inside. It was midday. I waited and waited, but no one came for me. I moved into the back seat to lie down. It was late at night, about 12 hours later, when my sister returned to find me hysterically whimpering in a fetal position—curled up in the back seat. She had finally gained permission to bring me in.
I remember walking down the clean, echoing halls to my father’s room to see him in a coma. My mother seemed to be in a trance, until we went to the women’s room where she broke down and sobbed, “What will I do without him?” All the things my father was to me, all the things he had done with me, flooded into my mind. The times he let me drive sitting on his lap around our plowed wheat field, even though I was only six years old. Because of this early training, I already knew how to drive, even though I was only 13. The time there was a tornado that raged right over our house, taking the chimney and a beloved weeping willow tree while we huddled together in the basement, his strong hand holding mine and reassuring me to “have faith, all would be well.” Teaching me to hammer and saw so I could build a tree house—all by myself. This kind, intelligent, affectionate father, a robust 48 years old, now lay crushed by a massive stroke that had come out of nowhere. It didn’t seem possible; it was completely unreal.

Coming out of the coma, he was in the hospital three months before returning home to be taken care of by my mother. We were told he would never walk or talk again. He didn’t understand anything we said and was totally silent. The neurologist told us he had “global aphasia.” As a young woman I liked books and wanted to know the answers to my questions, so I looked this up in the Encyclopedia Britannica—it said that this condition was “incurable.” Yet something just didn’t fit, even though I couldn’t explain my discomfort at the time. I remember mother telling me “Barbara, you have to just accept it. He will never talk again.” But something in me didn’t believe her, didn’t believe the speech therapists, and didn’t believe the Mayo Clinic that declared him “untreatable.”

About a year later, something was wrong with the car and he couldn’t tell us what it was, although he knew what was broken. He took a pencil and piece of paper and with his left hand (his right hand was paralyzed by the stroke) drew a complete diagram of the car engine with one area circled. Everyone was amazed he could do this. Obviously he had all his memory, but he just couldn’t talk! I took the piece of paper with this drawing to the mechanic who confirmed that it was, indeed, the clutch plate—the part that he had repaired. The fact that he could draw this diagram struck me, and I thought, “There is a way I can help him. There must be some way he can talk again.” Although I didn’t know it at the time, this was the beginning of a whole life’s journey and the discovery of what I would later call, The Sensory Trigger Method.

Years later, at 21, I was working in the Lincoln Public Library as head of the printing department. I had a young daughter, Jennifer, who at age two was learning to talk. I became fascinated with the work of Maria Montessori. Montessori, a French woman, was one of the first women to receive a medical degree. She was to become famous for discovering a way to teach brain-damaged children in the asylum how to talk, read, and write, using the sense of touch. Her theory that children can be guided to master their senses, even with learning disabilities and brain damage—that they could master fine motor coordination and develop abilities that may at first not be apparent, or may be lacking in their development, but are the very basis and foundation of adult intellectual and verbal abilities—this made a deep impression. Hunggrily, I went on to devour books on the evolution of language and the history of writing. There was something more there than just wanting to know how to help my two-year-old express herself!

Then one day, while I was clearing off my desk, it suddenly came to me—of course! The drawing that my father had made, it must have to do with that. He could communicate with pictures—pictureograms—like the cave drawings at Lascaux, France. The recovery of language could follow an evolutionary pattern. He could communicate with pictures—that is the starting point! Being an artist, I quickly took card stock and cut it up into small cards and drew pictures on each one. “Vernon”—a picture of my father, “point to”—a hand pointing, “cup”—a picture of a cup. I made one for myself and other objects: a door, a chair, a hammer, and an apple. The opinion of the speech therapist who had had asked my father to “point to the cup” and then diagnosed him untreatable because he couldn’t do that, suddenly became unimportant—this new way I had discovered became my new challenge.

Armed with my new set of cards and a few objects, I duplicated the speech therapist’s test, only this time it would be non-verbal, we would be communicating with pictures—and with touch! I laid the cards down in sequence on the desk before my father. “Vernon” “point to” “cup.” Avoiding body cues, I didn’t give it away by looking at the cup. I waited. Silently, I placed my finger down to touch each card in sequence. I showed him how to do the same with the index finger of this left hand. He followed my demonstration touching each one in sequence, left to right. Then his eyes lit up and he pointed to the cup! And even though, when I asked him to point to the door or the chair, he looked at me blankly, when I used the cards, he understood and could answer by pointing to the correct object from the group of objects that were on the table or in the room.

I then launched into intensive re-
A few years later, studies revealed that we talk with both sides of our brains as young children, before the brain specializes. More studies came out that indicated that the right hemisphere could learn to talk. As I continued to research, I found a study that showed that aphasia is rare in Asian populations. Because their writing is pictographic, their brain symmetry develops differently and active speech is probably stored on both hemispheres. It has also been found that bilingual people who have a stroke or brain injury on the left side often can speak their second language, but not their native tongue—especially if they learned it after the age of seven. Their brain stored the new language on the other side of the brain. Wondering, I thought, “What if we can make a new dominant hand? What if we can access the passive speech center through its corresponding hand, and get it to talk again?”

So, after 18 months of working with my picture language cards, mostly independently with the help of my devoted mother, my father said his first spontaneous word—it was nine years post-stroke. It was the first and only documented recovery from global aphasia.

He progressed from being able to say only about nine words, to being able to repeat words and copy them, writing the letters under the picture. As the long months passed, it seemed hopeless that he would ever be able to talk spontaneously, however. But then about 18 months later, watching the report on TV on the Mars explorer expedition, he drew a picture of the solar system and near the fourth planet wrote the word “Mars” and then pointing to it said “Mars.” He then found that he could say “Mars” whenever he wanted to. That was the beginning.

In 1973, I moved to California to find employment, and left my father to work on his own. I remember coming back home to visit him; he looked up at me and said, “Amazing.” Tearfully, I told him it truly was amazing! As his speaking vocabulary increased to over 700 words, he was able to go downtown on the bus by himself, go to the grocery store and the post office, even the bank. He became an artist with his left hand and showed his works in the state capitol building.

I went on to create the first talking software for aphasia recovery in 1988. It ran on an Apple IIe computer with an Echo Speech device. My husband, Michael helped me design and produce the program, while speech pathologists reviewed the plan and tested it at U.C. Davis Medical Center. In 1991, Reader’s Digest published, “Prisoner of Silence” written by Geeta Dardick—one of those “amazing stories.” Also, in 1991 Johns Hopkins University awarded me a Certificate of Achievement as the creator of the first talking software for speech therapy and aphasia recovery—I had won over 2,000 other entries in the competition. The program, called “Breakthrough to Language,” sold to the speech therapy field, as well as to special education, and by now has been sent to over 50,000 adults, to help them recover their speech.

This success was to be short-lived however. Computer technology was advancing so rapidly that we were forced to upgrade. The investors, however, didn’t understand the need to upgrade the software to high-resolution graphics. It would cost a half-million dollars to duplicate the program—it would be too costly. Without the upgrade, the program became obsolete. Sadly, we closed the company and retired the program.

Michael’s Story

Michael Schacker
My husband Michael and I decided to move to Woodstock, New York in 1995 to start over. There, we would be close to writers and publishers, as well as old family networks that could help us. After about a year, I realized that a whole new program could be created as an online program. I learned to program in HTML and created, with Michael’s help, the first online talking software for speech recovery in 1998. I focused more on self-help for stroke and brain-injury survivors, and added different Sensory Trigger programs and techniques that were not computer-based. I created StrokeFamily.org, a website devoted to helping stroke and head injury survivors recover their speech, rebuild their body, and prevent stroke. The “Let’s Talk” software program sold well and helped many people recover their speech. I continued my research and wrote guides and paper programs to make the “Whole Speech Practice Kit.” We didn’t know then how important this work would be to our own family—that catastrophic traumatic brain damage would strike again.

In the spring of 2008, Michael had just finished the last edit on his book, A Spring Without Bees, how colony collapse disorder has endangered our food supply, a book that the publisher realized was so important, it had to be rushed to publication. After over-working under unusually high stress for nine months, he did not look or feel well. His complexion was ashen, even though he ate well, and he had been falling asleep in the middle of the day, sometimes sleeping for hours. The way he walked was almost lopsided and weak. Michael shrugged this off as exhaustion and the fact that he had not had time to exercise for months. Melissa, our daughter, and I were worried about him; our premonitions told us there was something quite wrong with him. When we pleaded with him to see a doctor, he dismissed us. “No, that’s not necessary,” he said, “I’ll just get back in shape now that the book is finished.”

Then, on the evening of April 2, 2008, Michael developed a sudden severe backache. He said he wanted to lie down for a while and went upstairs. For some reason I followed him, deciding to work on my computer near him while he rested. Suddenly he said, “Barbara I’ve got this terrible pain in my back—it’s getting worse. I don’t know what’s going on.”

“Is it like a spasm?” I asked. He groaned: “No, it’s a like a knife ripping into me!” Instantly, I was on the search engine, typing in “heart attack, back pain.” It came up as a symptom. “Does your shoulder hurt?” “Yes” “Do your fingers tingle or feel numb?” “Yeah, a little.” He struggled to sit up but fell back on the bed. “It’s getting worse—I can…I can…feel…it—in my chest.” Now he was having trouble getting the words out. Suddenly I remembered the nightmare I had three nights earlier. In the dream I had come home to find Michael dead on the floor in a pool of blood with a hole in his heart that looked like a bullet wound. A strange feeling came over me that this was not just a bad backache. “You’re going to the ER right now!” “Why?” he asked in disbelief. “Because you’re having a heart attack, and you have clear signs of impending stroke.”

“There isn’t time for the ambulance—we live too far away, and it might take an ambulance a half-hour to get here.” I knew from my research that there is usually only a 20-25-minute window of opportunity to get to the hospital to receive life-saving treatment. “Melissa!” I called as we rushed out the door, “Call the hospital—the number is on the computer screen—tell them a heart attack and possible stroke will be in the ER within 20 minutes—tell them to have the stroke team and expert cardiologist ready and the CAT scan scheduled for use.”

Michael now was noticeably limping and leaning heavily toward his right side as he quickly got to the car. “I’ll have to speed to get there in time.” I said. Now Michael wasn’t talking, but making struggling sounds next to me in the front seat. I drove at 70 miles an hour with my hazard lights flashing. “What am I doing?” I thought to myself.

“You know this is the only way to save his life.” was the answer I heard in my head. “You’re going to be okay, Michael.” I said, as I reached over to hold his hand. Miraculously, there happened to be no one on the highway as we sped toward Kingston, and strangely there were no patrol cars to pull me over—and all the lights turned green. No one was even in our lane as we streaked down Broadway and turned into the hospital ER at 8:30 p.m.
As it turned out, we made it just in time. If we had been 10 minutes later he would have died. When the CAT scan revealed a problem with the aorta (an artery that feeds the brain) and not the heart, Michael was immediately airlifted to Albany Medical center where he was rushed into a 7-hour high-risk operation to install an artificial artery before it burst, and he would bleed to death.

He survived the operation, but the next day it was clear he had massive brain damage to his left hemisphere, and even some to his right hemisphere. The neurologist showed me the scan and my heart sunk. Almost his entire left hemisphere was gone. I knew what I was looking at. The area of damage looked like it was about 20% greater than the amount that would wipe out his speech and comprehension. The doctors said, “There will be no speech.” “What about speech therapy?” I asked. The top neurologist shook his head and paused, “It’s not possible…there will be no speech.”

Numbly, I walked away wondering, “Is he one of those I can’t help?” Then, trying to reassure myself, “Well, I have helped my father who was declared untreatable and I have helped countless other ‘hopeless’ cases—maybe. He is an expert musician—maybe he has mixed dominance—his right hemisphere was virtually untouched—I have to believe! I just have to believe in him, in God—in myself. And if I don’t know how to help him, even if I don’t know the answer, I promise, I’ll find it!”

Holding Michael’s left hand, and leaning close to him, I started at once with the Sensory Trigger Method. He was still in his coma. I talked to him and told him not to give up, that he knew that I would help him recover, and that one day he would walk and talk again. I talked to him for at least 30 minutes each day, while stimulating his left hand. My mind raced with all the things I needed to do…with all the things I had to do and all the things I could do.

Michael had to relearn how to breathe. His lungs had been shut down so long, that when the heart was restarted, they were collapsed and no longer worked. He spent the first month connected to life support with an artificial breathing machine that made horrifying sounds as the air was sucked in and then was forced out through the plastic-rimmed tube in his throat. There were all kinds of tubes; feeding tubes and drainage tubes sprouted from all parts of his body. A large piece of his skull on the left side had to be removed to relieve the pressure from the swelling of his brain. His head bulged out grotesquely on that side for a few weeks, and then, as the swelling went down, it caved in.

This left brain had collapsed and he couldn’t make any sounds at all. But he could nod his head for yes and no if I held his left hand. I showed this to the nurses who were flabbergasted. They couldn’t get answers out of him…but I could. I could, because I was using the Sensory Trigger Method that was going to the undamaged right hemisphere, while normal speech without the touch signal only went to the damaged side.

It was the end of April, and he had just been moved out of critical care at Albany Medical Center and into an acute care program at Sunnyview Rehabilitation Hospital in Schenectady, when the publisher sent the prepublication copy of his book. Melissa and I drove there to visit him almost every day, and this day we had his book in hand. When we arrived in his room I brought out the book and placed it in his hands. He wept as he realized he had lived to see his book published. I put the book in his left hand and held the book for him on the right side as Melissa turned a few pages. He looked hard at the print and I noticed his eyes scanning from left to right. “I think he is trying to read,” I said. After a while I noticed the light of recognition in his eyes as his eyes efficiently moved from left to right and he broke out in a huge smile. “I think he is reading! Are you reading, Michael? Can you read?” He looked at me and made a little sound. “You can read, Michael, can’t you? You can still read!” and he nodded slightly, his eyes shining!

After he put the book down and we had all taken a break, the thought came to me to try to get him to say the word “read.” So I went back in, took up his left hand, and told him, “I believe you can say ‘read.’ Let’s try it!” I let him watch my face. I said softly, “read,” exaggerating my facial expression. “Uh” he responded. “Read,” I repeated. “You can do it—rrrrr—eee—d.” “Uh r uh…dah,” he said. “Good! You made the r sound!” Over and over again we tried. I held his left hand while working with him. Finally, in about an hour—it seemed like eternity—he said the word “read.” “READ! Yes! Read! You said the word read!” I exclaimed. Melissa and I hugged him and cried and he cried too—he had said his first word. The doctors and nurses were so amazed—no one could believe it. “It’s very unusual—so early on,” one physician said blandly. But for me it was a sign—he could recover his speech.
Months passed and all he could say was “read.” “Read—read—read,” he would say it for “I love you.” “Read,” he would say for “yes,” and “read” he would say for “no.” It was his all-purpose word. He said, “read” for everything. This is because he not only had aphasia, the loss of speech—he also had apraxia and dyspraxia—repetitious speech not tied to meaning, unintelligible speech, and partially intelligible speech. He even had left-hand motor apraxia, which meant that he could not gesture to something that he wanted. He would reach over and pick up a pencil when he meant to pick up his spoon. He was far more damaged than my father had been. He would have to overcome all these different speech disorders.

Speech therapy was not really successful at Sunnyview. He could repeat the days of the week after the therapist and repeat numbers. He then could recite the numbers from memory in order up to ten, but not say them, or verbally identify them, on his own. Yet, with the sensory trigger method, words were popping out spontaneously, infrequently, but there all the same. When this happened, they came out perfectly clear—though often they were not retained and were seemingly never used again. He progressed to being able to repeat more and more words spoken to him. I challenged him and got him to say—much to his delight—“regeneration,” “synchronicity”—the words he had talked and written about in his unpublished book, “21st Century Transformation.” We gave him all kinds of books to read. One day he said, “sentient beings” spontaneously! And then, excited by the change in political climate in the country, “Obama!” (With great enthusiasm.) This was after he had moved into the Northeast Center for Special Care with their Traumatic Brain Injury Recovery Program.

The Northeast Center or NCSC as it is affectionately called, is an innovative, skilled nursing facility—perhaps one of the most innovative in the country. The
idea here is that traumatic brain injuries take a long time to recover from, and that most severe brain injuries don’t recover fully because the brain is not stimulated in creative ways. The Center features an extensive and extraordinarily successful art therapy program and music therapy program. Michael, being an amazing musician, not only performed as lead singer, he composed and recorded his own original music, playing the violin, guitar, and mandolin with virtuosity. In their music program he was allowed to be the “star” and could sing the words to his songs clearly, while the Center’s band learned and performed the music complete with musicians, and back-up singers who were also recovering from strokes and brain injuries.

I petitioned the Center to have a computer placed in Michael’s room so he could run my “Let’s Talk” program, alongside their speech therapy program. It was an informal adjunct speech therapy program. This at first raised eyebrows, but as he progressed more rapidly, with the Sensory Trigger Method and the programs in the kit, the speech therapist was allowed to include it in her therapy sessions with him. As predicted, in six weeks, his brain had grown new connections, and now more spontaneous words were coming back every day. His tendency to say the word “read” for everything slowly faded away into the background. I started sentence practice with him, beginning with three-word sentences. And then, six months later, he began to say spontaneous phrases and some sentences on his own...words that were not practiced, but words and sentences coming from the right hemisphere speech center’s ability to grow new connections in the brain.

Around this time we gained permission to take Michael out of the Center to receive special treatment given by Dr. Stephen Larsen. Ironically, we had been great friends of Stephen and his wife Robin. Here it was—miraculously—synchronously, you might say, our friend Stephen, who had successfully treated the severely brain-injured with LENS neurofeedback and other neuro-regenerative therapies!

When Michael started with LENS, he couldn’t walk more than a very short distance, and was still bound to his wheelchair. A few short months after treatment at Stone Mountain, he was walking with assistance into the office with a hemi-walker cane, sitting down in a regular chair, and walking back out to the car! And now, at almost three years post stroke, he walked into the office without his cane and with just a little assistance from his aide and me, to help him get up the steps and steady his balance.

Dr. Larsen mapped Michael’s brain and has kept careful records—charting the increase in brain wave activity that is now traveling through of its own accord in the previously “dead zones” of the left hemisphere. It appears that the two therapies have a synergistic effect when it comes to speech, as both methods make new pathways in the brain. Both confirm the new paradigm of brain function—the plasticity or learning capability of the injured brain—the brain that can heal itself.

Now, I often think of the thousands—perhaps hundreds of thousands of stroke and head injury survivors who have not been so fortunate to have the Sensory Trigger Method or neurofeedback therapy, and are still trapped in the silence of aphasia or the repetitive meaningless speech of apraxia and dyspraxia.

I have videotaped his progress at each step, each breakthrough in his recovery, and have published these clips on Stroke-family.org for others to get a sense of what real speech recovery looks and sounds like. He still has dyspraxia and makes some mistakes in his speech. He still works to break through the blocks to what he is trying to say. Yet, he continues to recover with brain regenerative approaches like LENS neurofeedback and the Sensory Trigger Method. And, remarkably, more and more independently. Michael becomes more like himself every day.

**Turning the Lights Back On:**
**Stephen Larsen Describes his Treatment of Michael Schacker**

When I first visited my friend in the Northeast Center, I was overwhelmed with sensations: the open, inviting lobby
was flanked by two lofty atria, filled with plants—and with the artwork of patients. A woman played respectable jazz piano on an electronic keyboard in the lobby. But even to get to the elevators, one passed dozens of patients who were extremely compromised by their injuries. A few people seemed caught in a private conversation with themselves; some others shouted and gesticulated. A few were high functioning enough to engage in personal conversations. One man told me quite lucidly of his accident that had left him wheelchair bound and with a complicated brain injury.

Michael was on the second floor, in a large room with his own bathroom and a window. All around was tangible evidence of his political and environmental affiliation: a picture of Barack Obama, nature scenes, lots of books, a computer, and a keyboard. Not bad for a hospital! Michael wore a purple football helmet because of his open skull. One false move in the bathroom, one lunge from his bed to the wall, and the brain damage could be incalculable. Day and night he had to wear that helmet. But he seemed to remember me, and greeted me warmly. His smile was lopsided, but very genuine. The only words he could say were, “read, read, read!” But he smiled with great warmth and enthusiasm as he said it. Barbara had already been working with the Sensory Trigger, and the imagery work that she had developed to help her father. There were cards around and she could start up a computer program. Michael was working cheerfully and willingly every day.

I was in the process of negotiating a professional agreement to come into the center and treat Michael, when the founding director, Anthony Salerno died, and there were major administrative shifts. Barbara decided to schedule weekly appointments for LENS sessions at our center, about 15 miles away from the Northeast Center, on an outpatient basis.

Michael arrived in a wheelchair, wearing his purple helmet, and smiling broadly at the outing. Dr. Zelek had already done a qEEG on Michael at the Northeast Center. He confessed to me that he was “trepidatious because the q requires a cap and the cap covered the place where Michael had no protective

Figure 5: The two sets of maps show Michael’s brain a year apart, and after about 25 LENS treatments. Note the Injury areas in both the qEEG images and the LORETA.
skull. But the map was completed, and confirmed the extent of the damage that the CAT scan had also shown.

I was thrilled when Michael’s skull plate was restored in November of 2009, after about 25 neurofeedback treatments, because it meant the re-traumatization caused by the restorative surgery was now behind us, and we could begin to treat the area underneath (where theoretically there was no brain) in earnest.

The comparison of the qEEGs one year apart is shown on the previous page. Michael is now able to walk into the center with a cane, and has a vocabulary of several hundred words. The LORETA map also shows profound differences.

As of this writing, Michael Schacker has been living in his own apartment, on disability, with caretakers coming in to help him on a schedule. He is growing stronger every day. He was recently able to walk into the center without his cane, and with a little assistance. He is able to read, use the computer, and is practicing to regain control of his right hand with the Ramachandran box we have had built for him.

The earlier version of this story was published as part of Chapter 12 in my book The Neurofeedback Solution (Healing Arts Press, 2012). Last year Michael was able to perform musically in front of a crowd of about a thousand at our annual Beltane Festival. His language comprehension is really normalized, though his language production is still halting. He can put short sentences together. The man who, according to his neurologists, would “never walk or talk again” is doing both. In late 2011, in one of our neurofeedback sessions, Michael kept emphasizing, “Stephen, Book! Book!” I replied, “Michael, your bee book is wonderful, and influencing people all over America and Europe—but he was insistent. Then Barbara said, “Oh no, I think he means the book he’s been working on for 30 years!” “What’s that?” I asked.

After I read it, almost completely finished, except for the last chapter, I knew it was publishable, and talked my friend John Graham at Park St. Press into publishing it. (Barbara finished the last chapter from some other writings, Michael re-read and approved the whole thing, and I wrote an introduction. The handsome hardbound edition of Global Awakening (Jan 2013 Park St. Press) is a work of genius, and not to be missed!

For more information on the Sensory Trigger, visit StrokeFamily.org or SensoryTrigger.org.

About the Author
Stephen Larsen, PhD, LMHC (NY), BCN, is Psychology Professor Emeritus, SUNY, director of Stone Mountain Center, PC, and, with his wife Robin, co-founder of the not-for-profit Center for Symbolic Studies. He is the author of 10 books currently in print, among which are: The Healing Power of Neurofeedback (2006) and The Neurofeedback Solution (2012). He also wrote, with Robin, A Fire in the Mind, the Authorized Biography of Joseph Campbell, the award-winning The Fundamentalist Mind (2007), The Mythic Imagination (1990) and The Shaman’s Doorway (1976.) He has presented many papers and chaired symposia at ISNR and AAPB conferences, and published articles in both The Journal of Neurotherapy, and Biofeedback.

At Stone Mountain’s annual Beltane Festival, Michael has been able to sing—accompanying one of his own CD’s, in front of 1,000 people.
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

SINGLE-BAND MAGNITUDE TOPOGRAPHIES

- Delta Theta Alpha Beta
- Microvolts
- Standard Deviations
- 3.0
- 0.0
- -3.0

AVAILABLE SERVICES

Full Package: #’s 1-7: minimum recommended for Neurotherapy
$225.00
Includes electronic copy. Priority mail is $20 extra.

Full Package: #’s 1-6: Without report (1-5 only)
$195.00
Includes electronic copy. Priority mail is $20 extra. If one database used the minimum is $75.00

01) NX Link - NYU/E. Roy John Normative Database (Eyes Closed)
$70.00
A) NX Link Discriminant Analyses: ADD, LD, Depression, Memory/Dementia, Substance Abuse, Head Injury, Schizophrenia/Thought Disorders

02) EureKa3! - NovaTech EEG LORETA Analysis System and Adult Normative
$70.00
Database - Eyes Closed

03) Neuroguide - R. Thatcher Normative Database
$70.00/each
A) Eyes Closed Linked Ears Z-scores // Eyes Closed LaPlacian Z-scores
B) Eyes Open Linked Ears Z-scores // Eyes Open LaPlacian Z-scores

04) Neurorep - W. Hudspeth QEEG Analysis System
$70.00/each
A) Eyes Closed - Weighted Average, Z-scores, Magnitude, % Power, LaPlacian, Average Spectrum, coherence, connectivity
B) Eyes Open - Weighted Average, Z-scores, Magnitude, % Power, LaPlacian, Average Spectrum, coherence, connectivity

05) Thatcher TBI Discriminant Analysis and Severity Index
$70.00
06) Thatcher Learning Disabilities Discriminant Analysis and Severity Index
$70.00
07) Clinical Correlations and Neurotherapy Recommendations by Bob Gurnee
$70.00

08) Conventional Medical EEG - Read by Neurologist
$125.00
09) EureKa3! – NovaTech EEG LORETA Analysis - Eyes Open-Non Database
$70.00
10) Neurorep - W. Hudspeth QEEG Analysis System: Task
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Weighted Average, Z-scores, Magnitude, % Power, LaPlacian, Average Spectrum

11) Supervision and Training Hourly Rate
$100.00
12) Extra set of Printed Maps sent priority mail
$35.00
13) Electronic (sent via FTP or E-mail) and Paper Copies of Maps sent priority mail with package purchase
$20.00
(Standard package only include electronic or paper copies of maps not both)

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

Total value: $630

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Established 1982
Transcranial DC Stimulation

Dave Siever, CET

Transcranial DC Stimulation (tDCS) is a recent neurorehabilitation technique to descend upon psychiatry and psychology. While still considered investigational by the FDA, and officially limited to research use within the United States, tDCS has shown itself to be effective in treating a wide variety of maladies, and holds great promise in clinical and rehabilitation applications. It is easy to use and only mild undesirable side-effects have been observed. There is essentially nothing that tDCS is contraindicated for, excepting use during pregnancy and with patients who have history of seizure as precautionary measures. tDCS is not to be used on those who have with a pacemaker or other implanted life-preserving bio-electric appliance. Technically, tDCS is easier to use than other neurostimulation techniques such as rTMS, or neurofeedback. However, researchers and clinicians aspiring to engage in tDCS must be knowledgeable in the neural networks associated with various training sites and potential impacts of training.

History of tDCS

In 43 AD, Scribonius Largus, a physician of the Roman Emperor Claudius, described a detailed account of the use of the (electric) torpedo fish to treat gout and headache. Since that time, a number of scientists experimented with electrical stimulation in hopes of treating various maladies as well as bringing people back from the dead. It was the invention of the battery that made DC stimulation or faradization, as it was termed at the time, possible. In 1755, French physician Charles Le Roy, wrapped wires around the head of a blind man in hopes of restoring his eyesight (Pascual-Leone, & Wagner, 2007).

Duchenne de Boulogne (Figure 1) became the first to systematically use electricity in the diagnosis and treatment of disease. He even brought a woman back from the “dead” after she was in a coma-state from carbonic oxide poisoning, by using an early form of cardiac electro-shock (Pascual-Leone, & Wagner, 2007).

In the USA in 1871, Beard and Rockwell published their book on the medical uses of electricity. They presented arguments for the use of galvanization (the term for DC stimulation at the time) for a variety of indications, as shown in Figure 2.

In the late 1700s to early 1800s, Giovanni Aldini (Galvani’s nephew) reported experiments using galvanization to treat psychosis, depression and even revive the dead. He later went on a traveling road show demonstrating the use of electricity for bringing cadavers back to life. It is thought that this showmanship may have been the cause for damaging the reputation of electrical stimulation for the next 100 years. In the 1960s, animal experiments using weak DC stimulation on the exposed cortex showed that neuronal activity could be altered immediately, and that these changes would last for several hours. These studies marked the true beginnings of transcranial DC Stimulation (tDCS).

Much of the original tDCS research has been done by Nitsche and his colleagues at the University of Gottingen in Germany. Other authors include: Fregni, Pascual-Leone and Boggio from Beth-Israel Deaconess Medical School (Harvard), plus Antal, Kincses, Hoffman, Kruse, and a dozen or so studies a year were published. A very thorough literature review covering the years from 1998-2008 with tDCS studies all categorized was completed by Nitsche et al., 2008).

In about 2005 the fascination with tDCS was growing and by 2008, it took off and the studies published accelerated almost exponentially. According to PubMed (see figure 3), in the time span from 2000—November of 2012, more than 500 tDCS studies were completed. This would make the grand total of tDCS studies in the range of 650-700 studies as of the time this article was written (November, 2012). In my analysis, most con-
This includes human, veterinary and animal studies where the study focus was merely an examination of the potential of 65 mv, by 5-10 mv, to anodal stimulation depolarizes the neuron. The negative electrode, termed the cathode, hyperpolarizes it. Stagg, et al., 2009, used magnetic resonance spectroscopy to observe that anodal stimulation decreases local GABA (an inhibiting neuromodulator) levels, thus increasing neuronal activity, while cathodal stimulation primarily decreased local glutamate (an excitatory neuromodulator), thus reducing neuronal activity. It is also shown that anodal stimulation increases cerebral blood flow (Zheng, et al., 2011), beta and gamma brain wave activity (Keeser, et al., 2011). Cathodal stimulation reduces cerebral blood flow while increasing delta and theta brain wave activity. The rule-of-thumb is that brain activity (as measured with FMRI or PET) under the anode is enhanced by roughly 20% to 40% when the current-density.

### Technical Aspects of tDCS

The positive electrode is termed the anode. Popular theory suggests that anodal stimulation depolarizes the local neurons from their typical resting potential of 65 mv, by 5-10 mv, to 55 mv, which in turn will require less dendritic input to fire (depolarize) the neuron. The negative electrode, termed the cathode, hyperpolarizes the neuron slightly and it will require increased dendritic input to fire it. Stagg, et al., 2009, used magnetic resonance spectroscopy to observe that anodal stimulation decreases local GABA (an inhibiting neuromodulator) levels, thus increasing neuronal activity, while cathodal stimulation primarily decreased local glutamate (an excitatory neuromodulator), thus reducing neuronal activity. It is also shown that anodal stimulation increases cerebral blood flow (Zheng, et al., 2011), beta and gamma brain wave activity (Keeser, et al., 2011). Cathodal stimulation reduces cerebral blood flow while increasing delta and theta brain wave activity. The rule-of-thumb is that brain activity (as measured with FMRI or PET) under the anode is enhanced by roughly 20% to 40% when the current-density.

<table>
<thead>
<tr>
<th>Table 1: Categorical Breakdown of Studies Using tDCS</th>
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<tr>
<td>NOTE: Several of these studies had dual purposes such as looking at some sort of rehabilitation of stroke patients. In these cases, the primary study (in this case stroke) was used as the category.</td>
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<tr>
<td>Motor Studies 70 These include all types affecting basic motor and fine motor control. Also includes MRI and other imaging studies during motor activities.</td>
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<td>Motor Learning 10 These include studies where a motor skill is learned.</td>
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<td>Somatosensory and Tactile sensitivity 15</td>
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<td>Memory 18</td>
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<td>Working &amp; Episodic (declarative) memory 18</td>
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<td>Recall of names 6 This includes human, landmark and other names.</td>
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<td>With Parkinson’s 2</td>
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<td>Cognition 10</td>
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<td>In general 10</td>
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<td>Reducing fake memory 1</td>
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<td>Neuro-plasticity of various types 20</td>
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<tr>
<td>Insight 2</td>
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<td>Decision-making 3</td>
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<td>Semantic dissonance 1</td>
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<td>Behavior inhibition 4</td>
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<td>Decort and lying 3</td>
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<tr>
<td>Threat detection 1</td>
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<tr>
<td>Risk-taking 1</td>
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<tr>
<td>Personality 1</td>
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<tr>
<td>Emotion 25</td>
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<tr>
<td>Depression 25 Most of these studies focus on F3 anode and/or F4 Cathode.</td>
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<tr>
<td>Bipolar 3</td>
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<td>Psychiatric Disorders 6</td>
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<td>Schizophrenia 6</td>
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<td>Obsessive Compulsive Disorder 1</td>
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<td>Cognitive Disorders 2</td>
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<td>General 2</td>
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<tr>
<td>Stroke 38 These include aphasia, motor dysphasia, declarative and working memory and mood.</td>
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<td>Parkinson’s disease 5</td>
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<td>Alzheimer’s and Dementia 12</td>
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<td>Planning 1</td>
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<tr>
<td>Traumatic Brain Injury 2</td>
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<td>Epilepsy 6</td>
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<tr>
<td>Pain 30 These include migraine, fibromyalgia, and chronic post-operative pain. Most involve the motor cortex and/or the prefrontal cortex as well.</td>
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<tr>
<td>Pain of various types 30</td>
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<td>Migraine 5</td>
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<td>Visual-spatial 5</td>
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<td>Visuo-motor 5</td>
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<tr>
<td>Auditory acuity and pitch discrimination 4</td>
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<td>Tinnitus 10</td>
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<tr>
<td>Somato-sensory 2</td>
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<tr>
<td>Addiction 7 These include drug &amp; alcohol addictions and eating disorders.</td>
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<td>Drug &amp; alcohol 10</td>
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<td>Appetite cravings 4</td>
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<td>Autonomic 3</td>
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<td>Blood pressure, heart rate, breathing 3</td>
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<td>Physiology 19 An EEG examination including brain wave categories, coherence, connectivity and other measures.</td>
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<td>Visual Evoked Potentials 5 These focus on VEPs during and following tDCS.</td>
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<td>Theoretical models 20 A look at theoretical models including electric field distributions, head-models of current-density.</td>
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<td>Brain Function 19 These studies use tDCS to stimulate various regions of the brain to help determine the functions of these regions.</td>
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<td>Cerebral Blood Flow 3 These studies look at the influence of tDCS on regional cerebral blood flow.</td>
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<td>Neurotransmitters 16 An examination of the neurotransmitters influenced by tDCS. These include, L-dopa, GABA, dopamine, NMDA, cortisol, etc.</td>
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<td>Glucose 1</td>
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<td>Ethics 4 A look at the ethics of tDCS, its safety, its efficacy and reliability of sham conditions.</td>
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<td>Comfort 3</td>
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<td>Methods 15 An examination of the best practices of electrode sizes, electrode distances and frequency of use for the best long term holding.</td>
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<td>Ring method of applying tDCS 4</td>
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<td>Other Studies 1</td>
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<td>Dreams 1</td>
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<td>Seizure 1</td>
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<tr>
<td>MRI 1</td>
</tr>
<tr>
<td>Cerebellum 4 These studies involve locomotion and ataxia.</td>
</tr>
<tr>
<td>Spinal Cord 5 Peripheral pain control and motor enhancement.</td>
</tr>
<tr>
<td>Animal 15 Examined memory, pain, neurotransmitters, neuroplasticity, etc.</td>
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<tr>
<td>Literature reviews 44 Included reviews on brain disorders, depression, efficacy, future prospects, pain, stroke, animal and general conclusions.</td>
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<tr>
<td>IAC Stimulation 10</td>
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These studies include studies where a motor skill is learned.
current density (concentration of amperage under the electrode) exceeds 40 µa/cm² (260µa/inch²). The cathode reduces brain function under the electrode site by 10% to 30% at the fore-mentioned current density. Anodal stimulation is the most common form of tDCS, as most application requires enhanced brain function (with the exception of pain).

The brain-stimulating electrode is termed the active electrode, whereas the circuit-completing inactive electrode is called the reference electrode. In most of the studies, the reference has been placed over the contralateral orbit (above the left or right eye). However, most studies have neglected to look at the inhibiting or boosting effects that the reference electrode might have on the brain regions where it is placed. Some recent studies, and in particular a study by Nitsche et al., (2007), show that it is better to have a small stimulating electrode and large reference electrode. This way, the current density is high under the treatment electrode and low under the reference electrode. This arrangement allows the reference electrode to be placed most anywhere over the scalp without it affecting brain function beneath it. Most studies have used stimulation at 1 mA of current through 7 cm x 7 cm (49 cm²) electrodes (there are 2.54 cm in one inch, therefore a 1” square electrode is 2.54 cm x 2.54 cm = 6.45 cm²). Fregni and his group at Harvard have suggested using a shoulder for the reference placement. I also advocate using a shoulder placement except possibly for treating depression, where the active electrode (anode) is placed over the dorsolateral prefrontal cortex (F3 on the 10-20 electrode montage) and the cathode over F4.

Nitsche and Paulus (2000) found that a minimum current density of 17 µa/cm² was needed to excite motor neurons. Studies involving other regions of the brain have suggested that 20 to 25 µa/cm² are needed to excite neurons under the electrode. One depression study using anodal stimulation at F3 noted alleviated depression using 1 mA into a 35 cm² electrode (28 µa/cm²). Iyer et al., observed that when stimulating the left prefrontal cortex there was no effect on verbal fluency with a 1 mA current, but significant improvement at 2 mA (current density of 20 µa/cm² vs. 40 µa/cm²). Two depression studies by Boggio, et al., 2007a; Boggio, et al., 2007b) also used 2 mA.

Safety considerations when using tDCS
A literature review by Poreisz, Boros, Antal, & Paulus, (2007) verifies that low-intensity transcranial stimulation is safe for use in humans and that it is linked with only rare and relatively minor adverse effects. Contrast-enhanced MRI and EEG studies have also found no pathological concerns associated with application of tDCS (Iyer et al., 2005; Nitsche, et al., 2003). Although patients with history of seizures are routinely excluded from current tDCS studies, no instances of epileptic seizures caused by tDCS have been observed in humans (Poreisz et al., 2007), and tDCS has actually been used to treat seizure (Fregni et al., 2006; Soon-Won, 2011). The safety of tDCS use in pregnant women and children has not yet been investigated.

The most common side effects observed with tDCS are mild tingling (71%), moderate fatigue (35%), sensations of light itching (30.4%), slight burning (22%), and mild pain (16%) under the electrodes (Poreisz et al., 2007). Some subjects report headache (12%), trouble concentrating (11%), nausea (2.9%), and sleep disturbances (1%) (Poreisz et al., 2007). Burn-type skin lesions following administration of tDCS have been reported (Palm, 2008), but typically only occur when standard techniques have not been followed. Visual sensations (phosphenes) are sometimes experienced with frontally placed electrodes. There is nothing unpleasant about phosphenes, but they may be avoided by using devices which gently increase the current at startup and vice-versa at the session end. tDCS delivered at levels of up to 2 mA and administered according to current stimulation guidelines (Nitsche, 2008) has been shown to be safe for use in both healthy volunteers and patients (Iyer et al., 2005). Again, current density is the most important consideration when using tDCS, as even 1mA will cause significant discomfort and skin burns if the electrode used is small or a part of the sponge is dry.

It is important that the tDCS device is current controlled. This means that the device will automatically adjust the voltage up and down as skin resistance changes so that the current always stays the same. For instance, if the resistance of the skin is 10,000 ohms, then 10 volts will be needed to “push” 1 mA through the body. If the connection becomes poor and jumps to 20,000 ohms, then the device will automatically increase the voltage to 20 volts in order to push the 1 mA current through the body. At some point the device must be able to determine if the connection is too poor at which time it shuts down automatically to avoid injury.

To demonstrate the degree of which non-regulated current can vary, I tested a 9-volt battery supplying a 1½” by 2”(4 x 5 cm = 25 cm²) tap-water wet sponge anode at F3 and a 2”x 4”(5 x 5 cm = 25 cm²) wet sponge cathode on the left arm and found that at the onset, the current flow was 300 µa (0.3 mA) making a current density of 15 µa /cm² (300/20cm²). By applying a mild pressure on the arm electrode, the current rose to 600 µa. When I increased the anode at F3 to 2”x 4”, the current was 600 µa and rose to 1.2 mA when pressure was applied to the shoulder electrode. The currents in both situations are well below the necessary clinical value of 40 µa /cm², and therefore not effective. The variance was 2 to 1 just by applying mild pressure to the electrode. When I soaked the electrodes (1½”x 2” and 2”x 4”) in a 5% salt solution (about 1tsp in 100ml of water), the current was a whopping 3 mA, (current density of 150 µa /cm²) and
I felt painful stinging on my forehead as the current density was much too high. If the reference cathode was also used on the head instead of the shoulder, there would have been a significant inhibition effect around it.

**tDSC Devices**

There are presently only a few standalone tDSC devices available. They are: the Eldith DC Stimulator by Neuro Conn, of Germany, which sells for $10,500 US, the HDCkit, (Italy), distributed by Magstim, which sells for just under $10,000, the StarStim (Spain), also about $10,000, the 1x1 tDSC by Soterix (USA) at about $4500 and the OASIS Pro, by Mind Alive Inc., (Canada), which sells for about $600.00 US. There is one device selling for $380, but it uses a metal mono-headphone plug which violates FDA safety standards, as insulated plugs are mandatory. A static shock through the exposed plug might rattle the brain quite well. I assume that all tDSC systems are officially available in the USA for investigational use only, as there is no evidence of tDSC device for sale within the USA prior to 1976, when medical device regulations were strengthened. As demonstrated above, tDSC devices should be current controlled, in that the device automatically maintains the exact current (amperage) set by the practitioner, regardless of electrode and body electrical resistance. It helps if the tDSC device is programmable and supports sham stimulation as described in research. The OASIS Pro™ by Mind Alive Inc., has these features plus the added benefits of providing cranio-electro-stimulation (CES) and microcurrent electro therapy (MET) for muscle work.

Currently, the accepted maximum current for human use is 2 mA and often 1 mA or less is used. Consistent with published safety guidelines, the OASIS Pro has been tuned with the electrodes provided, so that at 1 mA stimulation, the active electrode delivers 50 µa/cm² while the reference electrode produces 18 µa/cm². Table 2 shows the current density using various sizes at 1 and 2 mA currents.

### Depression, Mood, and Brainwave

The left hemisphere activates (and therefore suppresses alpha electrical activity as seen on an EEG) with happy thoughts and the right hemisphere activates (suppresses alpha) with negative thoughts. Right-brain strokes spawn cheerful survivors while left-brain strokes leave the survivor with depression (Rosenfeld, 1997). This supports the “happy-left” and “depressed-right” scenario. Other studies (Davidson, 1992; Coan & Allen, 2004), as well as my own observations, have shown increased left frontal alpha concurrent with a negative outlook. As one could expect, people with unresolved trauma are plagued with negative thoughts, often waiting for something bad to happen to them. Therefore, what one thinks also has a reinforcing impact one’s degree of depression.

### Quantitative EEG (QEEG) Assisted Treatments

One method to help determine electrode polarity and placement is to use a 19-channel qEEG and a normative database to assess where brain activity may be too little or excessive. This can be determined by observing delta, theta and alpha activity. If any of these brain wave frequency bands are excessive, then as a rule of thumb, I would use anodal stimulation. If beta brainwave activity is high, I would choose cathodal stimulation.

**qEEG Based tDSC (an anecdote)**

Obsessive-Compulsive Disorder (OCD) is a condition in which a person obsesses on an item and has compulsions to return to the point of obsession as a means to help reduce the anxiety associated with the obsession. One of the regions involved in OCD is the cingulate gyrus. OCD can develop from both an under- or over-aroused condition of the cingulate; therefore a qEEG is helpful in forming a diagnosis. The following case is of a woman person who was struggling with OCD, in which her prominent obsessions were hoarding. Despite having a house...
and garage full of junk, newspapers, and magazines (and despair about her condition), she could not stop herself from collecting even more stuff nor bring herself around to clean her home.

Figure 4 shows an eyes-closed magnitude qEEG in 1 Hz bins. Notice the high magnitude, slowed alpha (9Hz) activity along her cingulate gyrus (the pink strip). I have observed nearly identical activity for “tappers” and “counters.” For this type (a “no-brakes” cingulate), I would suggest placing the anode at various positions along the cingulate from FZ to PZ to boost cingulate activity as shown in Figure 5. For those with the beta (“foot on the gas”) type of OCD, use the cathode along the cingulate to bring the beta down.

Clinical Research Regarding Stroke

Given that the majority of the tDCS studies are of academic interest in learning about the brain and evaluating the effects of tDCS on healthy individuals, it is difficult to argue efficacy in the clinical population and instill faith in practitioners. Fortunately, stroke, one of the severest of neuropsychological maladies, has been studied extensively with tDCS.

In a sense, there are three generations of treatment techniques regarding stroke. They are:

1. First generation motor improvements.
2. Second generation motor improvements.
3. Studies involving cognitive aspects of stroke.

First Generation Studies

These studies involved anodal or cathodal stimulation over the motor cortex of healthy individuals or in those with stroke over the affected sensorimotor region. The reference electrode (anode or cathode) was typically placed over the contralateral orbit as indicated in the literature review by Nitsche, et al., (2008). This was the traditional way of doing things for many years. Unfortunately, the negative effects of extra-orbital (prefrontal) cathodal stimulation were largely ignored.

Second Generation Studies

These studies came about with the discovery by Vines, Nair & Schlaug (2006), that cathodal stimulation over the motor strip of healthy adults improved finger sequence movements on the contralateral side. The reference electrode (anode or cathode) was typically placed over the contralateral orbit as indicated in the literature review by Nitsche, et al., (2008). This was the traditional way of doing things for many years. Unfortunately, the negative effects of extra-orbital (prefrontal) cathodal stimulation were largely ignored.

These studies involved anodal stimulation over the lesion sensorimotor region in one group of sessions and cathodal stimulation over the contralesional region in another group of sessions. This eventually morphed into simultaneous anodal stimulation over the lesion region with cathodal placement over the contralesional area.

Third Generation Studies

These studies focused on other aspects of stroke such as apraxia, aphasia, depression, and memory. Anodal stimulation has been applied to the left dorsolateral prefrontal cortex for improving attention, with significant result lasting for several hours (Kang, 2009), for improving working memory (Jo, et al., 2009), and for treating depression (Bueno, et al., 2011). Anodal stimulation over Broca’s area has been used by Marangolo, et al., (2011), to treat apraxia and by Baker, Rorden & Fridriksson, (2010), to treat aphasia. Baker’s group also found that placing the electrode...
most closely over healthy parilesional areas yielded the best improvements, as did Fridriksson, et al., (2011), using MRI-guided electrode placement.

The practice of bilateral stimulation involving cathodal (contralesional) stimulation proved to be an even more effective means of inducing improvements in speech and language. It does so by inhibiting the Broca’s and Wernicke’s homologous areas (right hemisphere), which have a tendency to dominate the lesioned area and thus impede rehabilitation. Kang, et al., (2011), observed improved naming in 10 aphasia stroke patients when the cathode was placed over the right Broca’s homologue area and the anode was placed over the left supra-orbit (which may have had some influence over the left-side Broca’s area). You, et al., (2011) found that bilateral stimulation of Wernicke’s area and its associated homologous area improved comprehension of speech. A very good review on aphasia and language was completed by Monti, et al., (2012).

Literature reviews by Chrysikou & Hamilton, (2011), and Schlaug, et al., (2011), explored the benefits of anodal stimulation over the lesional area with simultaneous cathodal stimulation over the homologous (non-lesional) area. A very good literature review of tDCS in relation to stroke by Schlaug, et al., (2008), is available as a pdf file for free on the Internet. This review includes diagrams of electrode placement montages, regional cerebral blood flow images, and diffusion tensor imaging of a lesioned hemisphere vs. a non-lesioned hemisphere.

**Electrode Placement**

Electrode positioning is best chosen by observing a Brodmann Area chart (Figure 6), and then overlaying it with a 10-20 or 10-10 Electrode Placement Guide as shown in Figure 7. To learn more about functionality of the brain regions for determining electrode placements, visit: www.skiltopo.com. You will see a wonderful compilation of Brodmann maps with a detailed explanation of the functions for most Brodmann Areas.

**Studies**

**The Frontal Cortex**

There have been several studies of the right dorsal-lateral prefrontal cortex (DLPFC) in around F4. Anodal (+ve) stimulation with simultaneous cathodal (-ve) stimulation at F3 has been shown to reduce risk-taking behavior, whereas anodal stimulation only was not effective (Fectueu, et al., 2007a and 2007b). A study by Beeli, et al., (2008), showed that cathodal stimulation at F4 with the anodal reference over the contralateral mastoid, increased impulsiveness by 215%. Electrodermal responses to a simulated roller-coaster ride increased by 164%, which the authors referred to as a “being in the present” effect, although it could reflect a lack of pre-frontal inhibition over the amygdala (Ledoux, 1996). Cathodal stimulation at F4 with a left-orbital reference produced social improvements, in that the subject’s propensity to punish unfair behavior was reduced significantly (Knoch, et al., 2008). Figure 8 shows F3 electrode placements for boosting organizational skills and planning ability (Smith & Clithero, 2009), and for reducing depression. If a brain map warrants it, the same size cathode may be placed over the right DLPFC at F4, as shown in Figure 9. With F3 anodal and F4 cathodal stimulation, cigarette cravings were reduced by 21% (Fregni, et al., 2007), alcohol cravings were reduced (Boggio, et al., 2008), and junk-food cravings were reduced (Fregni, et al., 2008). Although there were better results using the F3 anode/F4 cathode arrangement, an F3 cathode/F4 anode electrode placement was, surprisingly, also quite effective.

Figure 10 shows the electrode placements for improving attention. The anode is placed on FP1 or FP2, with a contralateral shoulder cathode. A large anode electrode could also be placed across FP1 and FP2 at 2 mA, with a neck-placed cathode. Chi & Snyder (2011), found an
innovative way to increase insight. They used a well-known experimental paradigm involving “matchstick arithmetic.” Participants were asked to correct a false arithmetic statement, presented in Roman numerals constructed from matchsticks, by moving one stick from one position to another position without adding or removing a stick. Part of the reason that insightfulness is boosted is because the left temporal lobe is inhibited, as shown in Figure 11.

Figure 12 shows electrode placements for boosting socializing, including facial recognition and body language. A study by Kadosh (2010) showed that anodal stimulation at P4, with the cathode at P3 (Figure 13), improved math ability, but did not test to see if the cathode caused verbal impairments.

Perception Studies
At least 15 studies have examined the effects of tDCS on the visual system (Antal & Paulus, 2008). The main conclusion from these studies is that anodal stimulation of the visual cortex (Figure 14) improves contrast discrimination and visual-motor reaction times, while cathodal stimulation reduces these abilities. The benefits from improved visual processing may include art, painting, home decorating, flying jets, and action sports, etc. Cathodal stimulation at O1 and O2 has been shown to reduce the pain level of migraine (Antal, 2011). Figure 15 shows the electrode placement for improved auditory processing and pitch discrimination. (So you just might sing better in the choir.)

Motor Strip and Pain Studies
There are many motor strip studies showing that anodal stimulation at C3 or C4, with a contralateral supraorbital reference, enhances fine motor control, while cathodal stimulation impairs it. A motor-strip study of stroke patients by Lindenberg, et al., (2010), found that by placing the anode over the motor-strip lesion and the cathode over the contralateral motor-strip, motor function increased by 21% and lasted for one week.

There are several studies on the treatment of pain. Most of them found that anodal stimulation of the motor strip, exactly as the motor enhancement studies with a contralateral supraorbital placement of the cathode, is ideal for reducing pain on the opposite side of the body (Fregni, 2006), as shown in Figure 16. Here, the A1/C1 placement would suppress pain in the right side and vice-versa for A2/C2. It seems counterintuitive that boosting the motor strip would reduce pain—or could the reference electrode over the prefrontal lobe be involved?

Mendonca (2011) found that the pre-frontal lobes play a much larger role in pain modulation than was realized in previous studies. He found that either anodal or cathodal stimulation over the supra-orbit, with a neck reference, reduced pain equally well (Figure 17). However, the supra-orbit is the location of the prefrontal lobes. This is a sensitive area for attention and impulse control, and he did not assess the effects of stimulating here. But because Beeli (2008), found that impulsiveness and emotional reactivity increased substantially with cathodal stimulation, it makes sense, in my opinion, given that either anodal or cathodal stimulation work equally well, that it would be wise to use anodal stimulation over the supra-orbital regions and place the cathode at the base of the neck.

Figure 18 and 19 depict 3rd generation applications of tDCS, especially where applied to language and speech deficits resulting from stroke.

Conclusion
Transcranial DC Stimulation is site specific and, like neurofeedback, may be used to up-modulate or down-modulate any region of the brain. tDCS is also easy to use and doesn’t require the constant attention of the therapist, thus allowing the therapist to engage in talk therapy, some forms of biofeedback and/or collect client information during the treatment.
produces immediate and lasting sharpness and reasoning of mind. Prior to 2000, few studies considered the effects of tDCS beyond a few hours. In the past 10 years, however, an increasing number of studies administered tDCS on a daily basis for one to two weeks and then performed follow up testing a week or two later. Most studies showed lingering improvements. One depression study observed a holding effect 30 days later (which personal experience confirms).

It is important to be aware that sometimes there can be undesirable trade-offs. For instance, up-training insightfulness may impair auditory pitch discrimination. Up-training math ability may induce Wernicke’s aphasia. Down-training the right frontal lobe when treating pain could increase impulsiveness and anxiousness, while down-training the left dorsolateral pre-frontal cortex may bring on depression. However, between the existing research and my personal experiences, I suspect that with appropriate training, tDCS will become a common clinical approach to neurotherapy.

**About the Author**

Dave Siever, CEO of Mind Alive Inc., of Edmonton, Alberta, Canada, has been studying the mind and designing Audio-visual Entrainment (AVE), Transcutaneous Electro-neural Stimulators (TENS), and cranio-electro stimulation (CES) devices since 1981. He originally developed the Neuropulse II (TENS device) to relax the jaws of TMJ patients and the DAVIDI (AVE device) to help performing-arts students overcome stage fright. Dave later began developing transcranial DC stimulation devices in 2007. Dave has lectured and provided workshops with leading psychological institutions including the Association of Applied Psychophysiology and Biofeedback, the International Society of Neurofeedback and Research, the College of Syntonic Optometry, the American College for the Advancement of Medicine, Walden University, the University of Alberta, the Open University-England, A Chance to Grow Charter School, the International Light Association, accredited Biofeedback Training Programs, and other venues.

References are available in the supplement at: http://isnr.org/neurofeedback-info/neuroconnections-newsletters.cfm.
Training Programs from the Leader in Biofeedback

2013 CERTIFICATE PROGRAMS

5-Day Professional Biofeedback

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4-Day EEG Neurofeedback

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Spring 2013

Robotics in Stroke Rehabilitation: A New Mission for Bio/Neurofeedback?

Roger H. Riss, PsyD

Across the country and over the planet, a new breed of robots is invading therapy gyms and rehabilitation hospitals. Unlike the robot armies of science fiction, they are being warmly welcomed as one of the most visible signs that the science of neural plasticity is transforming rehabilitation medicine, offering new hope of recovery to those who have experienced stroke. Moreover, this new generation of robots represents an important new development in our field; for they are directly under the mental control of the patients they are tasked to serve, not via the telepathy of science fiction, but rather via the science of biofeedback and brain-computer interface.

A decade or more ago, if you had asked a rehabilitation therapist how they assist the stroke survivor, they might have told you that their job was to help the patient to learn to make best use of what abilities they have left. As evidence has mounted of the brain’s capacity for neuroplastic repair, therapists have learned that this characterization of their work was far too modest. Therapists are adapting their practice to a new reality: the brain’s capacity to reorganize and their propensity to fire together as a group grows stronger with each repetition, until that neural network becomes “hard-wired” in the brain, through a process known as long-term potentiation.

What drives this process? More than 60 years ago, the visionary Canadian psychologist Donald Hebb was first to propose the notion that “cells that fire together wire together.” He proposed that each time a group of brain cells fires in unison to perform a task, forming a temporary neural network, the strength of their connection and their propensity to fire together as a group grows stronger with each repetition, repetition leads to repair. In a series of landmark studies beginning in the 1960s, psychologist Edward Taub provided tangible evidence that that repetition leads to repair in a series of innovative animal studies. Imitating the effects of a stroke, Taub deprived monkeys in his lab of unilateral hand grasp by inducing a surgical lesion within their motor cortex. He then placed the monkeys in a specially designed jacket which prevented them from using their unaffected hand, and simultaneously offered them food treats in the form of peanuts at the bottom of a test tube. After weeks of repeated efforts to pick up the peanuts, the monkeys not only began to regain use of their affected hand, but also demonstrated measureable growth of new brain cells within the surgically damaged motor control areas of the brain. By contrast, monkeys who were not forced to use their surgically damaged limb demonstrated additional loss of hand function accompanied by evidence on neuroimaging of atrophy over the lesioned cortex (Taub E., Ellman S.J., Berman A.J., 1966).

The emerging science of neuroplasticity is teaching us that the brain is more resilient to injury than we’ve ever thought before. Throughout life, the brain is constantly responding to experience and has the capacity to reorganize and create new neuronal pathways to optimize its function. After a stroke, for instance, the brain engages in a process of massive reorganization, transferring control of abilities to spared regions, or from one lobe to the other. The brain’s solution to the problem of reorganization following a stroke may look very different from one individual to another, depending upon the location and extent of the lesion (figure 1). Immediately after stroke, both patient A and B show widespread and diffuse compensatory activation patterns as they attempt right hand movement. For patient A, who had a smaller and more focal lesion, control of right hand movement gradually returns to spared regions of the left motor cortex by three months post stroke. For patient B, who had a more massive left hemisphere stroke, control of right hand movement has completely shifted to the right hemisphere by three months post stroke.

Neuroimaging techniques such as fMRI have opened a new window to the brain, allowing scientists to observe the processes of neuroplastic reorganization as it unfolds over time. A neuroplasticity “rule book” is beginning to emerge. Rule number 1, not surprisingly, appears to be “use it or lose it.” But therapists are learning that not all kinds of practice are equally effective, changing the way ther-
apy is done after a stroke. For example, a new rule of thumb is that neuroplastic change requires massed practice and hundreds to thousands of repetitions, far more than can be accomplished in a 30-minute therapy session, potentially driving up rehabilitation costs to unaffordable levels. Optimally, repetitions should also be very specific and precise, facilitating stimulation to the exact same neural networks with each repetition.

One solution to the need for precision control with very high repetition rate has been the emergence of stimulation therapies such as the Bioness H200 Wireless Hand Rehabilitation System illustrated in figure 2. The system delivers low-level electrical stimulation to activate the nerves that control paretic muscles in the hand and forearm, helping the hemiplegic stroke patient to gradually regain function via thousands of repetitive stimulations of the neural connection between the affected limb and brain, promoting cortical reorganization and gradually allowing muscles to function without the system.

Another approach to the need for precise control with very high repetition rate has been the emergence of computer-controlled therapy robots (figure 3). Robotic therapy technologies offer several critical advantages. They can guide the recovering stroke patient through the highly-precise repetitions which optimize neuroplastic reorganization of the brain, and allow patients to get in many extra hours of practice and hundreds of repetitions to supplement limited time with their human therapist.

While first generation neurostimulation and robotic systems serve as useful therapy extenders, they have proved to be of only modest effectiveness. Because they are “closed circuit” systems, they are neither able to monitor the patient’s performance, nor to provide corrective feedback. Delivering only passive repetition, they fail to satisfy the final two “rules for neuroplasticity” for they neither engage active attention to task nor optimize patient motivation. Therapists have attempted to work around these limitations by instructing patients to actively “imagine that you are moving your hand on your own.” However they soon became frustrated by patients’ feedback that over time they became bored with the task and were essentially letting the machine do the work for them.

Bio/neurofeedback has stepped in to provide a solution to this problem. Second-generation robotics and neurostimulation systems are now emerging, which utilize an “open circuit” architecture offering two-way communication between man and machine. Rather than treating the patient as passive participant, second generation systems monitor EMG or EEG to assess the patients level of participation and engagement. The patient actively controls machine by initiating EMG or EEG activity according to criteria, and the system machine is triggered to offer assistance with the exercise only as long as the patient remains actively engaged. Moreover, many systems monitor patient execution, offering corrective feedback to assist the patient in refining the quality of their movement patterns.

One second-generation device incorporating biofeedback principles is the MYOMO (MyOwnMovements) mPower System, pictured in figure 4. Sensors on the skin’s surface detect the user’s muscle signal as he or she attempts movement, and the device automatically calibrates to the strength of the muscle signal detected. The device detects any trace muscle activity which the patient is able to produce, and then employs advanced robotics to reward the patient with feedback in the form of actual motion. The patient is guided through a series of functional exercises and eventually extends use into routine daily tasks.

The efficacy of EMG-triggered electrical neuromuscular stimulation for the
recovery of hemiplegic arm function was explored in a study by Korean researchers (Lee, et al 2003), who reported that subjects greater than one year post stroke demonstrated evidence on MEP mapping, of significant neuroplastic expansion of motor cortex output areas of the brain, after only four weeks of training (figure 5).

InMotion Robotics, invented by leading scientists at MIT, is another example of a second-generation biofeedback-guided robotic system using purposeful yet engaging computer games to maintain motivation during progressively more challenging use of the affected arm.

More than 40 peer-reviewed studies indicate up to twice the rate of therapy gains compared to traditional therapy alone. Pre-post training plots, below, demonstrate dramatic improvements in fine motor control following training for one patient.

EEG driven robotics

Patients whose hemiparesis is so severe that they are unable to generate even trace voluntary muscle activity have another option, in technologies such as iCTuS, which rely on EEG to detect imagined movement via the mirror neuron system. When you execute an action, such as grasping a ball, a cortical network is activated involving both motor and premotor cortex. A similar network activates during observed or imagined action.

The overlapping cortical networks involved in action execution, action observation, and motor imagery are known as the mirror neuron system. Scientists

Continued on page 46
Heart rate variability (HRV) consists of the beat-to-beat changes in the intervals between consecutive heartbeats. Reduced HRV appears to be a marker for increased vulnerability to physical and psychological stressors, and disease. Training to increase HRV may improve the body’s ability to recover from a challenge and promote resilience by engaging critical control systems. Heart rate variability is one of the most promising biofeedback techniques to emerge in recent history. HRV biofeedback is probably efficacious for the treatment of asthma, and possibly efficacious in the treatment of chronic obstructive pulmonary disease (COPD), depression, fibromyalgia, heart disease, heart failure, hypertension, posttraumatic stress disorder (PTSD), and unexplained abdominal pain. We are only beginning to realize the many possibilities of using this easy-to-learn and easy-to-teach technique.

Our neurofeedback community has started to integrate HRV biofeedback into neurotherapy because it appreciates how complex systems can work together to produce health and recognizes the potential synergies between these modalities. They’ve told us that the learning curve is not so steep and that the results are quickly apparent.

“Setting the standards for education and training” is a phrase that is closely tied to BCIA. Both the Association for Applied Psychophysiology and Biofeedback (AAPB) and the International Society for Neurofeedback and Research (ISNR) trust us to create the standards for the clinical practice of biofeedback and to outline and define the science, history, and theory; entry level competency standards for clinical work; and how biofeedback fits in with other medical or psychological tools.

Because of the increased awareness and usage of HRV in many clinical or performance settings, BCIA chose to build an HRV certificate program. Did I misread that? Was that a typo? It distinctly says certificate, not certification—why? As you know, BCIA already has three certification programs. After careful review, following the lead of The Institute for Credentialing Excellence (ICE), we decided that our field needs an HRV certificate program.

A certification program ensures that an applicant has at least demonstrated entry-level competence, involves mentoring and training requirements, and recertification to demonstrate the acquisition of advanced knowledge. In contrast, a certificate of completion demonstrates that an applicant has successfully completed a didactic workshop based on fundamental science that meets our rigorous academic standards. Following a needs assessment, we concluded that an HRV certificate of completion would better promote BCIA’s educational mission than a new certification. A certificate could increase the rigor of didactic programs teaching HRV biofeedback and enhance the expertise of academics, technicians, performance specialists, and licensed professionals who utilize HRV biofeedback.

HRV biofeedback has diverse applications—everything from cardiac rehabilitation to peak performance. Since this modality belongs to so many professions, we developed didactic education standards instead of educational requirements. We wanted to ensure that everyone who completes our certificate program studies the learning objectives based on the same peer-reviewed studies. We reasoned that the construction of an expert-designed Blueprint of Knowledge is essential for the modality of HRV biofeedback to gain recognition and credibility within the health care field. BCIA has three decades of experience in developing curricular standards for our biofeedback, neurofeedback, and pelvic muscle dysfunction biofeedback certification programs.

BCIA enlisted a task force of some of the best and the brightest HRV clinicians, educators, and researchers to help us decide what every entry-level provider should know. They addressed many challenging questions. What is the fundamental science of HRV biofeedback, independent of commercial bias and personal perspective? What anatomy/physiology and instrumentation coverage is necessary to competently provide this training? How strong is the scientific support for each of the current HRV biofeedback applications? Finally, how do we design a curriculum that is accessible and relevant for providers with incredibly varied educational and professional backgrounds?

The HRV task force built a 15-hour Blueprint of Knowledge that is divided into the content areas listed below:

1. Cardiac anatomy and physiology (1 hour).
2. Respiratory anatomy and physiology (1 hour).
3. Autonomic nervous system anatomy and physiology (.5 hour).
4. Heart rate variability (2 hours).
5. HRV instrumentation (3 hours).
6. HRV measurements (2 hours).
7. HRV biofeedback strategies (4 hours).
8. HRV biofeedback applications (1.5 hours)

The HRV certificate of completion is intended for BCIA certificants as well as individuals with no clinical background...
who provide optimal performance training using this modality. All applicants must:

1. Agree to abide by BCIA’s Professional Standards and Ethical Principles (PSEP).
2. Complete a BCIA-approved HRV biofeedback didactic program.
3. Pass a nationally-standardized online exam based on the 15-hour Blueprint of Knowledge.

We invite all current BCIA-accredited didactic training programs, universities, and new educational providers to submit their applications showing how their curriculum fulfills our HRV biofeedback Blueprint of Knowledge.

BCIA’s Certificate of Completion in HRV biofeedback demonstrates that one has successfully completed an educational program recognized by our field.

Certificants may use the 15 hours of continuing education to fulfill recertification requirements and/or maintain their health care license, depending on state regulations. We believe that building and maintaining educational standards is crucial for all biofeedback modalities to gain respect from the health care community and to take their rightful place alongside other recognized treatments.

Robotics in Stroke Rehabilitation continued from page 44

at University/ETH Zurich, Switzerland noted that after a stroke the motor system is often damaged, reducing ability to execute actions on the affected side of the body, while the mirror neuron system which activates with imagined or observed movement is typically spared.

Interactive Computer-based Therapy System (iCTuS) (figure 7) is a virtual reality system designed to maximally activate the mirror neuron system. The patient’s real arm movements are monitored via sensors and mapped onto virtual arms appearing on the computer display. If the patient cannot move their affected limbs due to a stroke, then the movements of healthy arm can be substituted and mapped to the affected virtual arm, providing the brain with a display of imagined movement in the affected hand.

By repeated “exercising” of the mirror neuron system, they seek to promote reorganization of the brain to restore movement of the affected hand.

The movements of the patient’s hands are tracked using custom-designed modular gloves (figure 8) which can be easily fitted even to patients with edema or spasticity. The system is size-adjustable for use by children and adults from the age of 6 upwards.

Practice is embedded in entertaining tasks (figure 9). Therapists can adjust the mappings of real movements onto on-screen virtual movements, implementing different therapy modes:

- Paretic virtual limb mirrors healthy real limb movement (mirror therapy).
- Paretic virtual limb follows healthy real limb movement (reverse mirror therapy).
- Paretic virtual limb moves twice as far as paretic real limb (movement boosting).
- Healthy virtual limb cannot move at all, patient must move using paretic limb only (constraint-induced movement therapy, CIMT).

Once a highly underutilized intervention in hospital settings, bio/neurofeedback technology is making a surprising re-appearance in hospital hallways as an integral feature of the latest generation of robotic and virtual reality systems designed to promote cortical reorganization following stroke and brain injury. While the preceding review was limited to discussion of upper extremity motor rehabilitation systems, similar technologies targeting language, memory, and cognitive rehabilitation are under development.

About the Author
Roger Riss, PsyD BCB, BCN, QEEGT, is a neuropsychologist with over 25 years’ experience in rehabilitation of stroke, brain injury and other neurological illness. He is co-editor of NeuroConnections and has served on the AAPB neurofeedback division board of directors.

References
Look for the 2013 Mini Grant Announcement Coming Soon!

Interested in PTSD research? The Foundation has created a listserv to bring like minds together. Email us at executivedirector@isnr-researchfoundation.org if you would like to participate.

Thank you to Cynthia Kerson and Mike Cohen for donating their speaking honoraria to the Foundation.

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WHY CHOOSE BIOGRAPH INFINITI?
A CLINICAL SYSTEM YOU CAN RELY ON

Prioritizing data validity and reliability
One of BioGraph's key strengths has always been to put your data's safety first. The system is crash proof and ensures optimal signal quality, integrated electrode impedance and artifact rejection are essential to clinical work. If you cannot rely on quality sensor data, you cannot ensure valid statistical analyses or demonstrate positive clinical outcomes.

Streamlining for ease of use
Version after version, in response to your feedback, we've made our system easier to use in order to help you work more efficiently. Version 5.5 introduced Quick Start functionality, which puts all the complexity of recording sessions into a simple desktop icon. Version 6.0's many enhancements radically expand your clinical choices.

Engaging yet contingent multimedia feedback
As a multimedia biofeedback and neurofeedback platform, BioGraph's audio and visual capabilities are exceptional. From the ability to manipulate MIDI audio files to the BioFun games, we have constantly maintained that enjoyable and engaging feedback is important, but accurate, informative and contingent biofeedback is essential.

Enhancing reporting capabilities
As a clinical software platform, BioGraph Infiniti analyzes your data accurately and generates easy to understand reports that make your clients' clinical progress evident. Release after release, we have created many specialized Application Suites to provide you with training and reporting screens for standard and user-definable protocols. The CardioPro Infiniti HRV Analysis module goes even further in providing high-end reporting capabilities.

Join us on...

BioGraph Infiniti 6.0 runs on the most reliable and reliable acquisition software in the world. Highly accurate sensors and low noise cables ensure quality signals, so you can focus on your work. Conforming to strict ISO and medical device policies and regulatory registrations across the world, including the newly released IEEE Recommendations for Neurofeedback Systems, the Infinit platform meets the highest standards in the field and beyond.
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