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Letter from ISNR President

This year marks 20 years since ISNR was founded. It seems like it should be longer, but then again, those years have gone by quickly. I was fortunate to attend the second ISNR conference that was held at the Union Plaza Hotel in downtown Las Vegas. It is interesting to compare the conferences of today to that early meeting. As I recall, there were 50–75 attendees and a handful of vendors there. It was very informal and slower-paced than conferences we are accustomed to now. Peniston’s work was being discussed then, as were neurofeedback methods and protocols, of which there were very few. And of course, the debates over whose approach was the right one had already started. Some things haven’t changed.

As I view this 20-year span of time and compare the issues the field has dealt with, I see great growth in some areas and no change, possibly regression, in others. Consider the technological advances that have taken place, particularly improvements in our amplifiers and EEG collection equipment. The size difference alone is amazing; starting with instruments the size of a small ottoman to today’s sophisticated devices that are no larger than a paperback book. Neurofeedback treatment models and methods have expanded into a variety unimagined 20 years ago. Among those things that have changed little, unfortunately, are insurance reimbursement and the need for more and better research to bolster the credibility of our claims about the benefits of neurofeedback. However, through our growth over the last 20 years, one unifying constant has remained: our dedication and enthusiasm for the healing and growth potential of neurofeedback. With our claims about the benefits of neurofeedback.

The subject of research brings me to report to you some recent activities of your ISNR Boards. The ISNR Board and the ISNR Research Foundation Board have been dialoguing about areas of mutual concern. Both Boards agree, as I know most of our membership does, that we need to continue to raise money for research in neurofeedback and other neuromodulation methods. The field still needs larger, better controlled studies on effects of neurofeedback for Autism, TBI, and other disorders. However, one regrettable trend that persists in our organization is the low percentage of members who financially support ISNR’s efforts to advance research. If we, who are dedicated to realizing the potential of neurofeedback and who worry about the lack of insurance reimbursement for our clients, are not financially supporting research in our own field, how can we expect those outside our organization to do so? I am reminding you to donate to the ISNR Research Foundation. You may do so through the links on the ISNR website or through the ISNR Research Foundation website at www.isnr-researchfoundation.org. There are many ways that you can donate, including Legacy Giving, which will allow you and your work to be remembered in the future. Donating to the Research Foundation is an investment in the continuing growth and credibility of what we do.

We can all celebrate our organization’s progress at our 20th Annual Meeting in Orlando this fall. This is sure to be a great meeting and a party to remember. The conference is being held at the Hyatt Cypress Gardens Hotel, September 19–23, 2012. The ISNR Board recently held its annual working retreat at the hotel, and we were awed by the accommodations and beauty of the facility. Lush vegetation, spacious surroundings, gorgeous pool area, a 9 hole pitch and putt golf course, wonderful restaurants, and if your room happens to be on the favored side, you can watch the nightly fireworks held at Disney’s Magic Kingdom just a few miles away. This is by far the nicest hotel I can remember for an ISNR conference. I know the Conference Committee has been working hard to produce the best meeting yet. The conference may be in Orlando this year, but it will not be a “Mickey Mouse” affair! (Sorry, couldn’t resist the bad pun.) Bring your family this year and make it a family event. While you are networking and gaining new information and ideas, your family can enjoy all of the attractions that are within a few miles of the hotel. Also, if you have photos, anecdotes, or items of interest related to ISNR’s history that you want to share with other members, please forward these to our new Executive Director at cyablonski@isnr.org, or bring them with you to the conference.

Our new Executive Director, Cindy Yablonski, MBA, was welcomed to ISNR last month. As I wrote in my last column, Cindy comes to us from the Protein Society where she has been Executive Director for the last eight years. She helped that organization grow significantly during her tenure there, and we look forward to reaping the benefits of her knowledge and experience. Please feel free to contact Cindy to introduce yourself and welcome her to our organization.

I am looking forward to seeing all of you at our 20th anniversary Conference in Orlando in September.

Richard E. Davis, MS

ISNR Mission Statement
To promote excellence in clinical practice, educational applications, and research in applied neuroscience in order to better understand and enhance brain function. Our objectives are:
- Improve lives through neurofeedback and other brain regulation modalities
- Encourage understanding of brain physiology and its impact on behavior
- Promote scientific research and peer-reviewed publications
- Provide information resources for the public and professionals
- Develop clinical and ethical guidelines for the practice of applied neuroscience

AAPB Neurofeedback Division Mission Statement
To improve human welfare through the pursuit of its goals. The specific goals are:
- The encouragement and improvement of scientific research and clinical applications of EEG technology and neurofeedback
- The promotion of high standards of professional practice, peer review, ethics, and education in neurofeedback
- The promotion of neurofeedback and the dissemination of information to the public about neurofeedback
- The division is organized for the purpose of carrying on educational and scientific objectives and is not to be operated for profit.
Neurofeedback Division Meeting Report: The Conceptual Reunification of Biofeedback and Neurofeedback

Last quarter, the theme of NeuroConnections was neurofeedback in application to PTSD. My own contribution dealt with the utility of infra-low frequency training in that application. This work has raised some fundamental questions about our standard assumptions in this field, and that led me to raise these larger issues at the Neurofeedback Division meeting at the AAPB annual conference. Again, I used our experience with PTSD to give some concreteness to these issues, to lay the foundation for the basic question about whether the division of our field into biofeedback and neurofeedback is sustainable at the theoretical level. That, in turn, raises the question of whether our existing institutional arrangements still serve our best interests. There was considerable sentiment at the meeting in favor of overt measures to try to breach the divide; however, attendance consisted of those neurofeedback practitioners who see sufficient value in the AAPB meeting to attend. We can hardly generalize from that to the neurofeedback community at large.

Let me briefly construct the argument for a reconciliation of the biofeedback and neurofeedback perspectives as it has evolved in my own thinking, given our clinical experience over the last five years. One of the early criticisms of infra-low frequency (ILF) neurofeedback was that it couldn’t be operant conditioning. That has always been a valid criticism. Such validity does not, however, dispose of ILF training. Matters are rather the other way around. Validity of ILF training disposes of the ‘universality’ of operant conditioning as the explanatory principle for neurofeedback. The validity of ILF training is no longer in question. Our clinical experience with this approach matches or exceeds our prior results over the preceding twenty years. Our burden is to recruit a different model to explain our results, and that has implications for basic assumptions we have been making all along. These have to be enlarged to encompass this kind of training. Basically, what is involved here is nothing more than what we might call waveform following, the witnessing by the brain of the time course of its own physiology. Fortunately, we have the precedent of biofeedback, where waveform following is already familiar through such techniques as Heart Rate Variability training. The mere witnessing of our own process of state management is sufficient all by itself to effect positive change. This is observable, for example, when we allow a person to observe his own breath waveform. Almost invariably, the waveform will move toward calmer, slower breathing. Listening to one’s breath via sound amplification does the same. No instruction needed. As soon as the brain becomes aware of its connection to the displayed signal it will quite naturally react to that signal and in effect “take charge” of that signal. It will extract information from the signal, but at the same time it will also try to control the signal. A feedback loop is created, and apparently there is no fundamental distinction here between the brain utilizing an external feedback loop versus an internal one. It’s just information, after all, and its means of delivery is a secondary issue. Correlation drives salience; salience drives engagement; and engagement drives the impulse to control.

Now if there is no essential distinction here between external and internal feedback loops, one might well ask why traumatized individuals don’t recover simply through the
exercise of their internal feedback loops? The answer is that most of them do. After all, only a subset of those who are exposed to similar traumatizing events end up with persistent PTSD. The rest recover by the same mechanisms that we augment and abet with external feedback. With most individuals, the deficits that finally present themselves for relief are traceable to the cumulative influence of numerous prior brain insults, each one contributing its bit to the diminution of the endogenous recovery capacity. Eventually, outside help (in the form of an external feedback loop) is needed to effect recovery. It remains to explain why bringing these external feedback loops to bear should be so rapidly effective. Paradoxically, it may be because the external feedback loops are not very good! The brain is engaging with a complex signal, or perhaps even with one that is corrupted by extraneous constituents. Further, the brain’s interpretation of the signal may also be less than perfect. Incremental errors lurk at every point in the control loop.

This means that the regulatory machinery is even more strongly challenged by the exercise than if the control loop were more ideal. And that turns out to be a virtue. An analogy may be helpful here. Consider the task of learning to drive after some practical joker slightly loosened the lug nuts on the front wheels. Control is still possible, but a tight control loop is out of the question. The driver will be much more strongly engaged with the task, and the steering corrections will have much wider swings, than if matters were as they should be. A real-world example is that of flight simulators for military and commercial pilots. Typically, the airframe control loops are deliberately degraded in crisis simulations to probe the stress tolerance of the pilot. In neurofeedback by waveform following, error correction drives the process, and the errors are there in abundance.

A second major theme that emerges from this work is the highlighting of our resting state networks. After all, in the ILF training, the brain is only getting to witness its infra-low frequency activity, which ties directly into persistent resting state activity—activity that is highly conserved and hence resistant to interference. So in our own little microcosm, our explanatory model is highly constrained; we are compelled to posit the functional re-normalization of our resting state networks as the operative mechanism. Given the ubiquity of functional connectivity deviations in the psychopathologies, it is likely that all the familiar neuromodulation technologies have, as their ultimate goal, the functional organization of our resting state networks, albeit more indirectly. This is then the objective as well of the biofeedback technologies, also more indirectly.

Our field thus far has resembled geology prior to the discovery of plate tectonics. Geology was getting done, but there was no grand organizing principle that made sense on the larger scale. The arrival of the theory of plate tectonics changed everything. The theory took more than forty years to be accepted, but such reluctance to adopt a new paradigm is only too familiar from our own experience. We’re well into our fourth decade also. The centrality of functional connectivity in psychopathology raises questions to which the neuromodulation technologies in general are an appropriate response. As usual, it is the theory that tells us what we may believe.

The third major theme is the manifest efficacy of a technique that targets function rather than dysfunction. This basic issue of appropriate targeting has been dividing the field for two decades now. In ILF feedback, the clinical condition at issue has only minimal import for the choice of a basic approach, and EEG data have no import at all. Ninety percent of all clients train under identical conditions in terms of the spectral response of the transfer function, and the remaining ten percent train nearby. The only other protocol issue is placement, and in this regard the entire clinical population sorts into no more than two standard starting placements. The target in all cases is the behavior of our resting state networks, not the particular complaint. The latter is only relevant for assessing progress through training. If the right training is being done, then symptoms that reflect dysregulation status should subside.

This approach could be called outcome-guided, to distinguish it from the other main thrust of the field, what Rob Cohen calls assessment-guided training. It is the response of the brain to the intervention that guides the individualization and optimization of the basic protocols. Over the course of training, the protocols do come to particularize to each individual, so that individual symptoms do end up driving protocol if they have not yielded to the more general approaches. The surprise, however, is just how much gets done with generic approaches using just two starting protocols. In this the ILF training is not unique. The NeurOptimal approach utilizes a single starting placement for everyone. Even what is called Live Z-score training supports this general case. If one is tracking 400 to 1200 measures, particularity is largely lost. The composite measure is closer to indicating the general dysregulation status of the individual. The target is better function rather than any specific dysfunction.

All of the standard reward-based protocols in neurofeedback have improved function as their target. And even standard inhibit-based training is really targeting improved function as well. It is simply cueing the brain as to its dysregulation status, leaving the remedy entirely at its discretion. Standard targeted coherence-based training can be described the same way. Although the immediate target may be a specific deviation, the clinical objective is not typically a specific dysfunction, but rather broader functional improvements. And finally, the stimulations employed in LENS, ROISH, and standard AVS devices are disruptive in character, leaving the brain to sort things out. These are still basically seeking general improvements in function.

This means that even though neurofeedback offers us a unique parametric selectivity that would be consistent with narrow targeting of specific deficits, this is not how things have played out so far, by and large. We may one day find such narrow targeting quite useful for some specific purposes, but most of our bread is earned these days by improving the trainee’s functional competences broadly and in that, we find ourselves very much in the same boat with traditional biofeedback.

All of the above makes the case for a substantial commonality between biofeedback and neurofeedback at the theoretical level. In other respects, there is a case to be made for complementarity at the level of practice. However, both the commonality at the conceptual level and the complementarity at the practical level contribute jointly to a mutual interest of interests within our respective communities. We are just not big enough for this division in our midst to be inconsequential.

The dominant source of division within the two branches of the field is that biofeedback concerns itself principally with autonomnic regulation and that neurofeedback takes as its point of departure a primary concern with executive function. Biofeedback has squatters’ rights on the hypothalamus, and neurofeedback claims custody of the frontal lobe. It is our ILF training, once again, that has persuaded us of the efficiency of EEG feedback even for autonomic regulation. Neurofeedback need no longer take a backseat in that regard. So, for the moment, it looks like neurofeedback is even poised to grab the crown jewels of conventional biofeedback.

From the biofeedback perspective, the case is made that autonomic regulation is foundational to the entire enterprise of physiological self-regulation. If good autonomic regulation is achieved, then broad benefits are derived—even including executive function. With the experience of ILF training, our experience is very similar. Essentially, all symptoms of cerebral dysregulation respond favorably to the training of the infra-low frequency EEG. We can bring these two perspectives into alignment by positing that autonomic regulation is also a pathway into the functional reorganization of our resting state networks. The key is not autonomic regulation per se, but rather the behavior of our system at low frequencies—i.e., resting state organization. Both peripheral biofeedback and ILF neurofeedback give us more direct access to the low-frequency domain than other methods.

The signal advantage of using measures of peripheral physiology in biofeedback is the responsiveness to state change. We can-
not say the same for the EEG, but it is clear from our experience with ILF training that one reason for its effectiveness is likewise the induction of state change. This is typically perceived quickly by the trainee, and it is consistently apparent to the trainer in the case of non-communicative young children. Both methods move the person to the state where the exercise of self-regulation strategies—of whatever kind—is most productive.

If ILF neurofeedback is effectively encroaching upon the turf of traditional biofeedback, why do we find ourselves thinking more about biofeedback rather than less? If at the level of theory we are attracted to the commonalities, at the level of practice we are drawn to the complementarities. If the induction of state change is so important in ILF training, then the peripheral measures may be helpful in tracking such changes. Further, they could be helpful in sorting out the training options, as well as in fine-tuning the parameter optimization procedure.

It has also been our experience over the years that nearly every new technique that we have ever tried has added something to our repertoire. Even while ILF training effectively trains autonomic regulation, it still does not entirely duplicate what can be done with Heart Rate Variability training, or so I am convinced. It stands to reason that HRV is to some extent complementary to ILF training, and to the extent that they poke in the same direction, they can reinforce each other. There need be no interference between HRV and ILF training.

At the end of the AAPB Meeting I had a chance to talk with Mark Schwartz over breakfast, and this brought to light the other major point of difference between biofeedback and neurofeedback. As I was relating a case of PTSD that was one of the standouts in our experience, having trained with us for about sixty sessions, Mark came back with, “There you go—60 sessions. Neurofeedback seems so inefficient.” The fact is that in about 25% of PTSD cases, the substantial resolution of symptoms takes only on the order of ten sessions. And even in the above case, there had been 90% recovery of classic PTSD symptoms within 18 sessions. Good function, however, goes beyond mere symptom abatement. We were after higher goals.

Our experience with ILF training is that results are achieved much more quickly than before, which makes it very competitive with biofeedback even on this measure. The fact remains that in neurofeedback we typically train a lot longer than is common in biofeedback, and that remains true with ILF training. We ask people to commit to 20 sessions first of all, and make no promises that that will be the end of it. But is this bad news or good news? In a survey of our cases done by David Kaiser years ago, outcomes were better for those who stayed in training longer. This is likely traceable to the perceived benefit on the part of the customer, since that is what ultimately determines longevity in treatment.

In cases of profound dysregulation such as we often encounter, there is typically an early component of fairly rapid recovery, and that is followed by a transitional period in which the focus gradually shifts from symptom abatement toward optimal functioning. Clients will persist with this agenda according to their own value system. If these same clients had instead visited biofeedback practitioners, chances are that they would have experienced the early recovery phase and then terminated treatment. The more the conversation shifts toward the language of optimum performance, the more EEG feedback is favored by its natural advantages.

In summary, the case is made in favor of getting back to a common conversation involving both biofeedback and neurofeedback, so that each can play its proper role in the maturation of self-regulation technologies. At the organizational level, there was strong sentiment within the Division for moving adroitly toward a joint annual meeting. The planning for that is already underway.

Siegfried Othmer, PhD

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Dr. Sherman then focuses on psychophysiological assessment and intervention for pain disorders, including headache, backache, phantom limb pain, Raynaud’s phenomenon and regional pain disorders.

Order your copy today at www.aapb.org or telephone 800-477-8892
This edition is focused primarily on tDCS training and experience. Several years ago at an ISNR conference, Yuri Kropotov, PhD, presented his studies/work among children with ADHD and children that were mentally retarded, which had very helpful and hopeful results. Finding an instrument in the United States proved to be nearly impossible. The instruments seemed to all be manufactured in other countries and were very, very expensive. Also, getting them into the United States seemed to be very difficult. So the years have passed with the desire to learn more about tDCS being ever present. Finally, having an edition that focused on this powerful training seemed one way to have more information and even find instruments that one could obtain.

The mild electrical shock (like a 9-volt battery level) seems to make the cells more responsive to inputs and Weisend thinks it “accelerates formation of neural pathways during the time that someone practices a skill.” Sally Aldee, writing of her experiences in This Week noted “I only remember feeling like I’d just had an excellent cup of coffee, but without the caffeine jitters. I felt clear-headed and like myself, just sharper. Calmer. Without fear and without doubt.”

The tDCS training seems to reduce the chatter, might help OCD a lot, and provides a calmer operational brain. Used with development of skills, whether motor or cognitive, seems also to have been very beneficial with improvement in the skills taking place in half the time as normal. This is very exciting, just think what this could do for our educational outcome. Children could learn math skills quicker and more thoroughly, with more children graduating with better competence in several areas.

To complete my journey into tDCS, I now have such an instrument and am searching for a clinician or researcher or both to train me on the appropriate use and appropriate selection of the sites.

The articles that focus on tDCS are by Dr. Sokhadze on work with autism; Dr. Riss, Ms. Daehling, and Dr. Ernst on their work with aphasia, and part II of Dr. Siever’s PTSD, which touches on tDCS with instruments that can produce such training. Be sure to read Tom Colluru’s discussion of Avatar. It works so beautifully and results are rather startling.

Also in this edition is Ann Marie Brown’s clinical study of a client with acquired brain injury, since we could not publish due to space limitations in the last two editions.

Hope your summer plans are all finished and you are going to have a wonderful vacation for some part of this beautiful summer.

See you in Orlando for the ISNR conference!

Warmly,

Merlyn Hurd, PhD, BCN Fellow

Roger Riss, PsyD

Creating Quite a Buzz—The Reemergence of tDCS Therapy

Therapeutic use of electricity on excitable tissues is not new (Sparing & Mottaghy, 2008). As early as 43 AD, Serenius Largus, a physician of the Roman emperor Claudius, provided 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. One of Charcot’s medical residents, Georges Duchenne de Bologne (1806-1875) became the first to systematically use electricity in the diagnosis and treatment of disease, and even employed an early form of cardiac electro-shock to revive a patient from carbon monoxide induced coma (for historical perspective, see (Priori, 2003)).

It was the invention of the battery that made DC stimulation or faradization, as it was termed at the time, possible. The basic design of tDCS, using direct current to stimulate the human nervous system, has been around for over 100 years. There were a number of rudimentary experiments completed during the 19th century using this technique that tested animal and human electricity. Luigi Galvani and Alessandro Volta were two such researchers who utilized the technology of transcranial direct current stimulation (tDCS) in their explorations of the relationship between motor cortex and muscle movement (Gross, 2007).

Early researchers also explored clinical applications in psychiatry. As early as 1804, Aldini demonstrated that cortical applied direct current stimulation was successful in improving the mood of melancholy patients. Subsequently, this discovery was largely ignored in the favor of efforts to develop pharmacological agents. When electroshock therapy was developed in the 1930s, and was found to be a relatively effective treatment against depression, tDCS therapy in psychiatry was largely abandoned, until its re-emergence in the last decade (Utz, Dimova, Oppenlander, & Kerkhoff, 2010).

In the 20th century, Russian researchers developed a cortical electrical stimulation technique involving application of a low amplitude alternating current, termed cranial electrotherapy stimulation (CES) or ‘electro sleep therapy’, which was anecdotally utilized by Soviet cosmonauts to promote more efficient rest while in outer space. In the US, this form of stimulation has received FDA approval as a safe alternative to pharmaceuticals for insomnia, pain, and mood disorders (Gilula & Kirsch, 2005).

The past decade has witnessed a rapid resurgence of interest in therapeutic applications of transcranial direct current stimulation (tDCS) and other non-invasive brain stimulation technologies. An important principle of neuroplasticity is that “cells which fire together wire together.” Indeed, on a cellular level, the process of learning is best described by repeated engagement of transient, task specific neural networks. With repetition, the initially transient association between neurons during task performance becomes consolidated, or “hard wired,” a process known as long term potentiation (LTP) (Rioult-Pedotti, Friedman, & Donoghue, 2000).

Advances in neuroscience research have given rise to a new generation of neuroplasticity-based therapies, and reawakened interest in tDCS for its potential to enhance speed of motor, cognitive, and emotional recovery following psychiatric or neurological insult, with 150 peer reviewed papers published within the past 12 months alone.

Elsewhere In the present issue, we describe an emerging shift in cognitive and physical rehabilitation in which tDCS is emerging as a potential tool to boost the neuropsychiatric impact of traditional speech, physical, and occupational therapies. In a similar vein, tDCS holds promise as a potential synergistic intervention to neurofeedback training. In contrast to neurotherapy protocols which rely for their effectiveness on cumulative learning over a number of sessions, neurostimulation therapies such as tDCS induce targeted shifts immediately within the first training session, potentially promoting quicker skill acquisition and more robust response to neurotherapy-based learning effects. Synergistic effects such as these are already being observed in combined tDCS + physical therapy, and tDCS + speech therapy studies. While not yet FDA approved for the treatment of any medical condition, tDCS is a “methodology on the move” and is creating quite a buzz among research groups around the globe. We hope that you enjoy learning more about the cause for all this interest and enthusiasm, in the pages of the current issue. (References on page 36)
Letter from AAPB ED

Conference Attendance up 13%!

This year’s annual conference in Baltimore marked an increase in attendance over last year by 13%! The conference content just keeps getting better. And this year was no exception. The sessions were so good that at one point, a keynote speaker was running over the allotted time and when he acknowledged this, the audience simultaneously encouraged him to keep going saying “we will make time for you!”

This is just one example of the quality of content that was delivered at this year’s AAPB conference. The overall atmosphere for the conference was upbeat with member networking at its best. There were plenty of highlights with the keynote presentations, concurrent sessions, and pre-conference workshops. When the planning for the 2012 conference began, President, Gabriel Tan, PhD, asked the Program Planning Committee (PPC) to seek content that is not a repeat of the same material presented year after year. And, under the leadership of PPC Chair, Ron Rosenthal, PhD, we did just that!

So, what is in store for next year? Riding on the success of this year, the PPC is off and running in preparation for the 2013 meeting scheduled for March 13—16 in Portland, Oregon. Under the leadership of next year’s PPC Chair, Fred Shaffer, PhD, three of the keynote speakers are already confirmed including:

- Leslie Sherlin, PhD: “High Performance Brain Training: Old Idea–New Reality”
- Chet Moritz, PhD: On Movement Disorders (title to be determined)
- Dennis Turk, PhD: “Management of Chronic Pain PATIENTS and Not Just Their Pain”

The theme for the 2013 meeting is already set, “Creating Synergy: Integrating Methods and Modalities.” As the speakers and topics above suggest, the PPC is well on its way to addressing the strategy that is inherent in this theme.

With meeting attendance and membership showing positive trends, AAPB is looking to achieve a banner year for 2012! To keep that momentum going, we encourage you to start planning now to attend AAPB’s 2013 meeting in Portland! Mark your calendar now and plan to be there! You don’t want to miss it. See you there!

David L. Stumph, IOM, CAE

NeuroConnections

Mentoring—What’s it all about anyway?

Fred Shaffer, PhD, BCB, BCIA Chair and Judy Crawford, BCIA Director of Certification

Mentoring is the process of transferring skills and knowledge from a more experienced person to a student wanting to learn something new or enhance their current knowledge base. When learning to employ any new skill, this is the time-tested method for going that step beyond the words in the book and exploring the real-life application of skills. For those of us in the health care community, we are exposed to this method through our university practicum, supervised clinical training while we gained hours for licensure, or even some continuing education workshops.

Many feel that learning the hands-on practical biofeedback or neurofeedback skills is really the foundation for building the next generation of well-trained clinicians. The BCIA blueprints of knowledge clearly delineate the fundamental science, history, and theory required for entry-level competency, and while the requirements for practical skills training are outlined, it is learning the nuance and the subtlety behind what we do that can’t be easily quantified. This is where the skill and the experience of the mentor can make the biggest impact.

The relationship between a student and the right mentor can have lasting and positive implications, not only for the student, but for the mentor as well. As Albert Einstein wisely said: “Teaching should be such that what is offered is perceived as a valuable gift and not as a hard duty.”

What is the difference between mentoring and supervision?

Supervision is the legal oversight of a person’s work. Mentoring is simply teaching how to use a modality with no implied patient responsibility. Please check the laws of your state that govern all professional activities to be sure if there may be any statements that could impact this relationship. For example, in some states the practice standard guidelines specify whom a person may work with professionally.

BCIA mentoring is based on a consultation model rather than a strict supervision model. If the mentor is also providing licensing supervision, this should be clearly outlined. Remember, typically, a licensing supervisor cannot accept payment (gifts) from the trainee. Make sure to check your professional state laws.

Who should provide mentoring?

Those who have gone beyond the entry-level stage in their professional development may see it as their duty and their privilege to reach out and help another gain clinical skills, just as they were mentored and taught when they were beginners. Anyone who believes they have mastered specific skill areas and wishes to see our field advance may feel the calling to help the next generation gain competency.

How do we make a good match between the student and the mentor?

Making a good match is really key to a successful mentoring relationship and it is based on several considerations. Professional preparation may be an important starting place. It may be helpful if the student and the mentor come from similar or related fields—medical vs. psychological for example. Physical therapists know their own specific scope of practice, can relate to how their undergraduate training prepared them, and how they view the client relationship. Another important consideration is the current or anticipated client base. Finding a mentor who has skills with the same disorders and client population or where you’d like to move your practice is essential. Each disorder we treat takes a specific skill set and finding a mentor who can provide you the insights and skills related to those symptoms is an important consideration.

Most people start their mentor search with geography; however, due to enhanced e-communication tools, we can more easily remove those barriers and expand the avail-
Jonathan E. Walker, M.D.

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

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

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

**Neurotherapy Center of Dallas**
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Email us: [admin@neurotherapydallas.com](mailto:admin@neurotherapydallas.com)
Visit our Web site: [www.neurotherapydallas.com](http://www.neurotherapydallas.com)
ability of the mentor pool. Time zone may be a factor, but only because it makes setting up meeting times more convenient. The best place to start is with the Find a Practitioner search function at www.bcia.org. For best results use only the two-letter state abbreviation as your search criterion. You will see a list of the certificants in your state arranged in geographical order. Did you know that if you click on most names, you will get more insight into who they are as a clinician, what they treat, and other information that will help in the selection process?

We suggest that you draft an email introducing yourself professionally and outlining your request. Be specific about your needs, for example, describe your equipment, client-base, didactic training, and skill level. These details will outline the type of mentoring relationship this could be and will give the professional more information to consider. Also think of if you have something to offer. Could you offer free services of any kind in trade for some mentoring time?

Do I have more than one mentor?

Absolutely—and in fact we encourage it. If students can find more than one teacher, their learning experience will be positively affected—even if the perceived value of the two mentors is not the same. We have found that a student may take things from both mentors and combine what they learn to establish the professional presence that makes the most sense for them. Another way to use that second mentor is to spend a few hours with that professional and learn from their expertise in a specific topic who may not be geographically convenient or may be even too expensive for using for all of the contact hours.

Do mentors get paid?

Yes, in most cases mentors are paid and here is why. Mentors are health care professionals who typically expect to receive an hourly rate for their services. If they take a clinical hour out of their work week to spend time with a student, they may expect this time to be repaid. Additionally, they are helping a student build a tool that will enhance all future professional work.

What is the going rate? There is no going rate. This is like asking what do you pay for a haircut. There are many factors such as geography, experience and prestige of the mentor, and specialty area that can impact the fee schedule. Some mentors may do this for free because they feel they wish to give back to the field, and for this, we are grateful.

Another impact on the fee schedule could even be in the structure of the mentoring. For example, if a mentor gives up a clinical hour during the week, they would expect to be paid for the hour of client time they may have lost. If the meeting time may be done over the weekend from home or outside of their clinical work, they may feel that the cost can be lowered a bit.

What other things impact mentoring?

The number one thing that we can suggest is to know who you are, how you fit into the field, and exactly what you are asking. Please consider this scenario—a licensed therapist across town has had exposure to the field over several years so she is not really a beginner. She has her own practice and equipment. Perhaps what she is looking for is to polish her skill set or to provide proof of the type of formal training required for certification. A mentor may look at this situation as fairly easy to accommodate into a busy schedule. However, consider this other scenario—an unlicensed person with no practice, no clients, and no equipment is seeking mentoring. Is it really mentoring they seek or rather an internship? Isn’t she really looking to work in somebody’s office using that professional’s equipment and clients? This is a completely different question. BCIA advises students to know what they are asking. Remember, you will get the answer to the question you’ve asked, so ask the right question.

Do you suggest a mentoring contract?

Yes we do. Good fences make good neighbors and good mentoring contracts make for a rewarding experience for both parties involved.

Continued on page 18
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

QEEG / TOPOGRAPHIC BRAIN MAPS: Cingulate Dysfunction: Anxiety, Rumination, Obsessive Compulsive Disorder

High Mean Frequency Beta: Anxiety, Alcoholism, Insomnia

Standard Deviations

Delta Theta Alpha Beta

-3.0 3.0

22Hz 3.0 0.0

23Hz 3.0

24Hz -3.0

25Hz

STANDARD DEVIATIONS

Delta Theta Alpha Beta

Cingulate Dysfunction: Anxiety, Rumination, Obsessive Compulsive Disorder

22Hz

23Hz

24Hz

25Hz

SINGLE-BAND MAGNITUDE TOPOGRAPHIES

MICROVOLTS

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Tel: (480) 424 7200 Fax: (480) 424 7800
Web: www.add-clinic.com Email: add@add-clinic.com
Established 1982

QEEG / TOPOGRAPHIC BRAIN MAPS: Delta Theta Alpha Beta

Available Services

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

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

01) NX Link - NYU/E. Roy John Normative Database (Eyes Closed)
AI 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 Database - Eyes Closed

03) Neuroguide - R. Thatcher Normative Database
A) Eyes Closed Linked Ears Z-scores // Eyes Closed Laplacian Z-Scores
B) Eyes Open Linked Ears Z-scores // Eyes Open Laplacian Z-Scores

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

05) Thatcher TBI Discriminant Analysis and Severity Index

06) Thatcher Learning Disabilities Discriminant Analysis and Severity Index

07) Clinical Correlations and Neurotherapy Recommendations by Bob Gurnee

08) Conventional Medical EEG - Read by Neurologist

09) EureKa3! - NovaTech EEG LORETA Analysis - Eyes Open Non Database

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

11) Supervision and Training Hourly Rate

12) Extra set of Printed Maps sent priority mail

13) Electronic (sent via FTP or E-mail) and Paper Copies of Maps sent priority mail with package purchase

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

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Total Value: $630

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$195.00

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$70.00

$70.00

$125.00

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$70.00

$100.00

$35.00

$20.00

Varies

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MSW, BCIA:EEG, QEEG Diplomate, Director
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RESEARCH UPDATE

2012 Mini-Grant Program
Application Deadline Extended to July 1, 2012

Two applicants whose projects best demonstrate potential for contributing to the knowledge of basic processes involved in neuromodulation methods and technologies or to understanding of the clinical effects of neurofeedback or other neuromodulation methods with populations/disorders currently under-researched will each be awarded a $2,000 grant. Replication studies will be accepted when adequately justified.

Questions regarding the application process should be directed to the Research Foundation Executive Director, Dr. Cynthia Kerson at executivedirector@isnr-researchfoundation.org.

Check out our new Web site: www.isnr-researchfoundation.org!

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ADD Centre Brodmann Booklet
Thank you to the authors: Michael Thompson, James Thompson and Wu Wenging

Multi-Component Treatment for PTSD
Thank you to the author: John Carmichael

The Art of Artificating
Thank you to the authors: Cory Hammond and Jay Gunkelman

Doing Neurofeedback: An Introduction
Thank you to the authors: Richard Soutar and Robert Longo
Neuromodulation Using Transcranial DC Stimulation (tDCS) and Repetitive Transcranial Magnetic Stimulation (rTMS) as a Translational Neuroscience Approach to Treat Autism

Abstract

Transcranial Direct Current Stimulation (tDCS) is a non-invasive neuromodulation procedure used to increase (anodal tDCS) or decrease (cathodal tDCS) cortical excitability. Recently, tDCS has been increasingly used to investigate cognitive functions in both healthy subjects and psychiatric patients. Our team was first to report positive effects of repetitive Transcranial Magnetic Stimulation (rTMS) in autism and provided rationale to consider it to be theory-driven neurotherapy. Although tDCS produces cortical effects over a longer period of time, it has several practical advantages over rTMS. First, tDCS is less prone to artifacts and is more suitable for controlled study designs. Second, tDCS is not as expensive as rTMS and can be performed with compact equipment. Third, tDCS can be administered while recording electroencephalogram (EEG) and autonomic nervous system activity, thus allowing for concurrent investigation of physiological effects of neuromodulation. Fourth, tDCS may have great potential for cognitive and behavioral enhancement targeting to treat some of the core autism symptoms.

Introduction

Transcranial Direct Current Stimulation (tDCS) is a promising neuromodulation tool for research and treatment of various disorders. Even as far back as the first century, Roman physician Scribonous Largus had noted that delivery of an electric current to the scalp, in the form of a live torpedo fish, could be used as a remedy for headaches (Brunoni et al., 2011). Today we have a considerable body of research regarding the neuromodulatory effects of tDCS, including its application for treatment of symptoms of various neurological disorders, enhancement of performance on certain tasks, as well as data that suggests that it is a safe and relatively inexpensive tool (Utz et al., 2010). Research applications using tDCS can enhance our knowledge of neuroplasticity and neuromodulation, and could lead to promising non-pharmacological interventions for disorders such as autism.

Unlike transcranial stimulation methods such as TMS, tDCS does not directly induce neuronal action potentials. Instead, tDCS works by mediating the flow of ions in and out of the neuronal membrane. By shifting the polarity of the membrane, transcranial DC stimulation alters the resting membrane potential, which results in changes in the spontaneous firing rates of the stimulated neurons. In addition, prolonged stimulation leads to a temporary modification of the synaptic microenvironment, which accounts for the observed after-effects that persist for up to an hour after a 10-minute session of stimulation (Angelakis & Liouta, 2011). The stimulation is administered using a cathode and anode electrodes. Cathodal stimulation has an inhibitory effect on the underlying cortical neurons, while the anode electrode has an excitatory effect. Based on current literature, current densities up to 0.029 mA/cm² can be delivered without causing unpleasant sensations to the subject (Brunoni et al., 2011). In addition, single-blind or double-blind study designs are completely feasible, since the experimental conditions and sham conditions are indistinguishable to the subject, as long as the delivered current is gradually ramped up and down in the beginning and end of the experimental session (Gandiga, Hummel, & Cohen, 2006). Since the location and current density of stimulation can be varied, tDCS can target specific functional areas with various intensities, making it a very versatile investigational tool.

TDCS in Research Aimed to Enhance Autistic-Like Savant Skills

Individuals with autism are known to be more literal and are less prone to commit errors in memory traces retrieval. They perform better in some visual symbol discrimination tasks, and often present savant-like skills (Snyder et al., 2003, 2006; Snyder, 2009). There is some evidence which suggests that autistic-like literal skills are associated with left hemisphere deficit along with right hemisphere compensation. This inspired Chi, Fregni, and Snyder (2010) to investigate whether memory for visual shapes can be temporarily improved by tDCS. Only participants who received left cathodal tDCS (aimed to decrease excitability), in conjunction with right anodal stimulation (aimed to increase excitability) showed improvement in the visual memory task. The improvement in visual memory was similar in those with autism, who are known to be more literal. This demonstration provides insight into potential neural mechanisms underlying savant skills in people with autism, such as deficient left-fronto-temporal activity along with compensatory over-activation of the right temporal areas.

There are still only a limited number of studies which show that tDCS can lead to cognitive enhancement (Cattaneo et al., 2011). Monti et al. (2008) found that cathodal stimulation of the Broca’s area of the left fronto-temporal region led to a 33% improvement in naming accuracy. Studies by Boggio et al. (2009) showed that tDCS (anodal stimulation) of the left temporal lobe resulted in enhanced memory. Effect of brain stimulation can be dependent on topography of neuronal orientation in space. Studies which show that tDCS can lead to cognitive enhancement of cognitive functions by brain stimulation using tDCS in combination with clinical neurophysiological techniques such as quantitative EEG (qEEG) and even-related potentials (ERP).
sentences for syntax training. All procedures were performed both before and after tDCS. Results of this study demonstrated a large effect size of the difference between pre- vs. post-tDCS syntax acquisition scores, indicating positive effects of neuromodulation (Schneider & Hopf, 2011). This is as an excellent pilot study of treatment and assessment of language-related deficits in autism.

**TDCS research in healthy volunteers and in neuropsychiatric clinical research**

Significant effects of tDCS neuromodulation have been reported for motor, visual, somatosensory, attentional, vestibular, and cognitive/emotional function in healthy volunteers, in addition to a range of neurological and psychiatric disorders. Recently, tDCS has been attracting increasing interest as a promising clinically applicable tool for neurorehabilitation due to its potential to modulate cortical excitability and therefore promote neuroplasticity (Stagg et al., 2011). Application of tDCS for clinical purposes is approved in the United Kingdom, several European Union countries, and Canada, but is not yet FDA approved in the USA. Several well-received presentations on the potential of tDCS as a neuromodulatory approach to treating neurological dysfunctions including autism have been recently presented (Angelakis, 2011; Kropotov, 2009).

We are currently conducting a pilot study where tDCS over the prefrontal and fronto-temporal area is used to improve executive functions and enhance language function in children with autism using simultaneous recording of EEG and peripheral nervous system measures. For the assessment of outcomes of this experimental research study, we use behavioral surveys, language function tests (CTOPP and CELF-4), and auditory and visual oddball tests with ERP recording.

**TMS-based neuromodulation in autism to improve inhibition/excitation ratio**

Transcranial Magnetic Stimulation (TMS) allows scientists to stimulate the brain noninvasively in alert, awake patients. Repetitive TMS (rTMS) can be divided into low-frequency rTMS (≤1Hz) and high-frequency rTMS (>1Hz), and they differentially affect cortical excitability. It has been shown that low-frequency (“slow”) rTMS (≤1Hz) increases inhibition of a stimulated cortex, whereas high-frequency rTMS (>1Hz) increases excitability of a stimulated cortex. It has been proposed that the effect of “slow” rTMS results from increases in the activation of inhibitory circuits through long-term depotentiation (Hoffmann & Cavus, 2002).

TMS has been applied to a wide variety of psychiatric (e.g., ADHD, depression) and neurological disorders (e.g. Parkinson’s disease) in adult populations and more recently rTMS has been applied in children (Croarkin et al., 2011). We proposed that rTMS has unique applications as a treatment modality for autism spectrum disorders (ASD) based on our autism neuropathology model (Casanova, 2007; Casanova et al., 2002, 2006). It has been suggested that a wide range of deficits in autism might be understood by an increase in the ratio of cortical excitation to cortical inhibition and increases in local cortical connectivity accompanied by deficiencies in long-range connectivity. One possible explanation for higher-than-normal cortical noise and abnormal neural connectivity in ASD is the recent finding of minicolumnar abnormalities. We hypothesized that contrary to other inhibitory cells (i.e., basket and chandelier), whose projections keep no constant relation to the surface of the cortex, the geometrically exact orientation of double-bouquet cells and their location at the periphery of the minicolumn makes them the appropriate candidate for induction by a magnetic field applied parallel to the cortex. Over a course of treatment “slow” rTMS may restore the balance between cortical excitation and cortical inhibition and lead to improved long-range cortical connectivity.

In a recent study (Baruth et al., 2010), we investigated evoked EEG gamma band activity (35–45 Hz) in subjects with ASD and assessed the effects of 12 sessions of bilateral rTMS applied to the prefrontal cortices in 16 of the ASD participants. Following rTMS, individuals with ASD showed significant improvement in discriminatory gamma activity between relevant and irrelevant visual stimuli, and there was also a significant reduction in irritability and repetitive behavior as a result of rTMS.

We have also been interested in investigating event-related potentials (ERP) abnormalities in ASD. In a previous paper (Sokhadze et al., 2009a) we investigated ERPs in a three-stimuli visual oddball task in ASD (Sokhadze et al., 2010a) and assessed the effects of a course of “slow” rTMS stimulation applied to the left prefrontal cortex on performance in a visual task of selective attention in 13 individuals with ASD. TMS minimized early cortical responses to irrelevant stimuli in this task, and increased responses to relevant stimuli, indicating improved selectivity and better target stimulus discrimination. These results were recently confirmed in 24 subjects with ASD by finding significantly improved ERP indices of attention after 12 sessions of bilateral prefrontal rTMS. We also evaluated the effects of 12 sessions of bilateral “slow” rTMS on error monitoring and correction in autism. The active rTMS group showed significant improvement in error detection and correction compared to a randomized, non-active rTMS group (Sokhadze et al., 2012); this may point to improved executive functioning and behavioral performance in ASD as a result of rTMS.

Overall, our preliminary findings show promising results for rTMS as a treatment modality targeting core symptoms of ASD. Treatment with “slow” rTMS decreased excess gamma activity and amplified ERP responses in ASD patients during visual tasks, and improved the signal differentiation between processing relevant and irrelevant stimuli (Baruth et al., 2010; Sokhadze et al., 2009b; Sokhadze et al., 2010ab). There was also a significant reduction in the percentage of errors in motor responses to target stimuli (Sokhadze et al., 2010ab), and rTMS was associated with a significant improvement in indices of error detection and correction (Sokhadze et al., 2012). Our results suggest that low-frequency rTMS has the potential to become an important therapeutic tool in ASD treatment.

**Conclusions**

Novel neurotherapeutic techniques such as tDCS and rTMS represent non-invasive procedures of neuromodulation used to increase (i.e., anodal tDCS, high frequency TMS) or decrease (i.e., cathodal tDCS, low frequency TMS) cortical excitability. Recently, tDCS
has been increasingly used to investigate cognitive functions in both healthy subjects and psychiatric patients. There have not yet been any controlled studies reporting outcomes of tDCS in autism, while more progress has been made in application of rTMS in treatment of autism. Our research team was first to report positive effects of rTMS in autism and provided rationale to consider it as a theory-driven enotherapy based on Casanova’s “minicolumnar” neuropathology hypothesis of autism. We are just starting research of tDCS applicability in autism. Although tDCS produces cortical effects over a longer period of time, it has several practical advantages over rTMS. Both neuromodulation techniques reviewed in our paper may have substantial potential to advance both basic research and contribute to understanding of neuropathology of autism, and furthermore advance so desperately needed applied neuroscience approaches to treatment of autism spectrum disorders. 

References


19 Channel NeuroGuide Neurofeedback
Seamless Integration of QEEG and EEG Biofeedback

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Symptom Check List
Discriminant Functions
Power
Coherence
Phase
JTFA
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Phase Reset
Statistics
Instantaneous Coherence & Phase Reset
Phase Lock and Phase Shift Duration
3-D LORETA Z Score Biofeedback.

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tDCS: A Stimulating Boost for Aphasia Rehabilitation Outcomes?

Cognitive Plasticity Working Group; Madonna Rehabilitation Hospital

Roger H Riss, PsyD, FACPN, Department of Neuropsychology; Julia Daehling, MS, CCC SLP, Clinical Supervisor Department of Communication Disorders; Ryan Ernst, PsyD, Department of Neuropsychology

Introduction

tDCS, a portable, safe, non-invasive, brain stimulation technique, is capable of modulating the excitability of targeted brain regions by altering neuronal membrane potentials, based on the polarity of the current transmitted through the scalp via sponge electrodes. Anodal stimulation increases cortical excitability in the stimulated brain tissue while cathodal stimulation decreases it.

tDCS has enormous clinical potential for use in stroke recovery because of its ease of use, its noninvasive nature, its safety (does not provoke seizures), its sham mode (important for controlled clinical trials), and the ease with which it can be combined with traditional cognitive, language or motor recovery interventions (e.g., simultaneous speech/occupational/physical therapy) (Schlaug, Renga, & Nair, 2008). Interest in tDCS in aphasia treatment is growing exponentially; with 14 of the 20 tDCS aphasia studies completed to date published within the past 12 months.

Background

For the past two decades, clinical rehabilitation practice has been guided by a consensus of opinion emphasizing the teaching of compensatory strategies and only rarely supporting restorative approaches, citing limited research to support their use. However, the viewpoint that compensatory strategy training alone is an acceptable therapy outcome is now coming under serious challenge.

Functional neuroimaging research has given rise to a tantalizing and provocative new viewpoint on the role of rehabilitation therapy; to wit, that neuroplastic cortical reorganization following brain injury is the norm (Kim, Ko, Parrish, & Kim, 2002; Warren, Crinion, Lambon Ralph, & Wise, 2009), and that a necessary and sufficient condition for any effective rehabilitation intervention is that it facilitates and directs this process. Stroke rehabilitation paradigms are evolving as researchers explore both pharmacological and neurostimulatory technologies to facilitate neuroplastic change in order to improve functional outcomes; concurrently, researchers are exploring algorithms to more accurately predict who will benefit from this new class of interventions (Burns, 2008).

There are a number of ways of performing electrical brain stimulation. In the case of deep brain stimulation, surgeons implant electrodes directly into specific areas of the brain, allowing currents to be sent directly to the relevant cells. This highly invasive procedure is currently being tested on crippling diseases such as Parkinson’s. A less invasive approach, called repetitive transcranial magnetic stimulation (rTMS), uses fluctuating magnetic fields from a large electromagnet to trigger the firing of neurons in specific areas of the brain, or to induce “virtual lesions” which interrupt normal brain activity.

Among neurostimulatory techniques, tDCS appears to hold particular promise as a practical clinical tool for noninvasive therapeutic stimulation of the brain cortex, combining portability, low cost, and ease of use with negligible risk and minimal side effect profile. It is a noninvasive brain stimulation technique that utilizes low amplitude direct currents applied via scalp electrodes to modulate the level of excitability of cortical cellular networks, rather than overriding normal cellular activity. With tDCS, a weak electrical current of 1 or 2 mampere is applied to the head with an electrode (For comparison, ECT, or electroconvulsive therapy, utilizes a current in excess of 900 m Amperes). The electrode is a nonmetallic conductive rubber electrode, covered completely by saline soaked sponges.

Mechanically, the tDCS stimulator is a simple device quite similar to those routinely utilized by physical, occupational and speech therapists to facilitate recovery of muscle use. When applied cortically, electrodes attached to the person’s scalp target an exceedingly small current to specific regions of the brain. At the cellular level, these extremely weak currents are sufficient to shift the voltage barrier needed for a nerve to fire, essentially modulating normal activity. Depending on whether the anode (positive lead) or cathode (negative lead) is placed over the targeted region, the clinician may either subtly activate or inhibit a particular cortical region to facilitate task performance.

Continued on page 21
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DATA PROCESSING
Programmable 64 bit data processing and digital filters, mathematical operations and quick export

ADVANCED EEG
DC-EEG (0-3000Hz), SCP ERP VEP, P300, Zscore, GEEG export, real-time Neuroimaging (NeXus-32)

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1-Main menu: select Start Protocol
2-Select Neurofeedback protocols
3-Select Theta/Beta Training
4-click Next>> or Skip to jump to 10
5-Follow Instructions, click Next>>
6-Check Connectors, click Next>>
7-Check Electrodes, click Next>>
8-Place Electrodes, click Next>>
9-Run Signal Check, click Next>>
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2. Select Neurofeedback protocols
3. Select Theta/Beta Training
4. Click Next>> or Skip to jump to 10
5. Follow Instructions, click Next>>
6. Check Connectors, click Next>>
7. Check Electrodes, click Next>>
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BioTrace for Advanced users

QEEG, ERP, SCP and Multi-Modal Physiology

Expert users and researchers need precise data acquisition, so we store signals with 26 bit resolution and process them with 64 bit floating point precision. Advanced users can create their own virtual data channels, create their own data processing, perform quick statistical analysis of sessions (up to 26 hours of data) and export data in various file formats for easy import into other software. The NeXus-32 software has additional features for the full cap EEG, including neuroimaging and QEEG export. We have included a short list of some of the most features of BioTrace+ below. BioTrace+ software runs on our entire NeXus family of products: NeXus-4, NeXus-10 and NeXus-32. One software, many systems...

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Data Processing
Programmable 64 bit data processing and digital filters, mathematical operations and quick export

Advanced EEG
DC-EEG (0-500mV), SCP ERP VEP, P300, Zscore, QEEG export, real-time Neuroimaging (NeXus-32)

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2-Select Neurofeedback protocols
3-Select Theta/Beta Training
4-click Next>> or Skip to jump to 10
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7-Check Electrodes, click Next>>
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9-Run Signal Check, click Next>>
10-PRESTO: you are ready to start!

BioTrace for the NeXus-32: a wireless QEEG
Biofeedback and Neurofeedback platform

Clinician screen
Client screen
The Stens Corporation, incorporated in 1976, is internationally recognized as the #1 provider of Biofeedback and Neurofeedback training in the United States, Canada, and abroad. Over the past 35 years Stens has trained and introduced more than 14,000 new people into our industry! President and C.E.O., Stephen Stern, was honored with the AAPB Presidential Award in 2005 for his role in fostering the growth of the Biofeedback/Neurofeedback industry over the past 3 1/2 decades.

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A particular advantage of tDCS is the ease with which it can be incorporated into rehabilitation therapies and research designs. The typical 10- to 30-minute stimulation duration lends itself to concurrent use with traditional therapies, to test for any incremental benefit of stimulation on learning and skill acquisition. Additionally, due to its subtle effect, tDCS lends itself to sham-controlled research designs. The level of current during training is so slight that participants in well-controlled studies are typically unable to discern the difference between actual tDCS stimulation, and sham tDCS, where the device is attached to the scalp but turned off (Gandiga, Hummel, & Cohen, 2006).

**Aphasia therapy**

Aphasia is an impairment of language as a result of focal brain damage to the language dominant cerebral hemisphere (Darley, 1982), serving to distinguish it from the language and cognitive-communication problems associated with non-language dominant hemisphere damage, dementia and traumatic brain injury (Orange & Kertesz, 1998). One of the most common consequences of stroke in both the acute and chronic phases, an estimated 35% of individuals with stroke have symptoms of aphasia at the time of discharge from inpatient care (Dickey et al., 2010).

Most, if not all aphasias are accompanied with word finding difficulty. Many approaches have been used to help the patient with word retrieval (McCaffrey, 2012). The approaches for language retrieval therapy take on many forms such as an individual one-on-one session, group therapy, trained volunteers to work with the patients, and at home computer-based programs. The role of the speech-language pathologist is to attempt regain as much of the lost language system as possible. Often it is the case of a combination of restoring the language system and compensating for the specific deficits of function. Family members often have to be trained on new ways of effectively communicating with their loved one.

Over the past decade, neuroplasticity research has revolutionized aphasia therapy, leading to the development of tremendously successful, neuroplasticity based, high intensity interventions such as Constraint Induced Aphasia Therapy (Pulvermuller et al., 2001), and Intensive Language Action Therapy (Pulvermüller & Berthier, 2008).

Neuroscience research indicates that therapy involving mass practice and high frequency training help to utilize remaining linguistic abilities and to regain lost language functions. Language therapy is most effective in treating aphasia when provided intensely; less intense therapy given over a longer period of time does not provide a statistically significant benefit, although some clinical benefit may be achieved (Teasell et al., 2009). A family of techniques collectively called intensive language-action therapy (ILAT) incorporates these recommendations (Pulvermüller & Berthier, 2008). They are based on three principles from neuroscience research. The principles are mass training, communicative relevance, and the patient’s communicative needs. Constraint-induced aphasia therapy (CIAT), which has sometimes been called constraint-induced language therapy (CILT), is one variant of ILAT that encourages patients with Aphasia to practice language in combination with the appropriate actions in “therapeutic language games.” It is a therapy program observed to improve object and action naming, where patients may only respond with verbal output (no gestures, writing, sound effects) (Maher et al., 2006; Meizner, Djundja, Barthel, Elbert, & Rockstroh, 2005; Pulvermüller et al., 2001). The principles of CILT are:

- Forced verbal language use and application of constraint
- Verbalization required
- Compensatory strategies prohibited (constrained)
- Intensive treatment schedule with massed practice
- Shaping verbal responses by beginning with words or short phrases and moving to longer and more complex utterances.

Short intervals of intense practice are preferred over long-term, less frequent training. This was shown in a Pulvermuller et al study, in which patients receiving CILT (3 hours of therapy per day for 2 weeks) significantly improved on all outcome measures, compared with the patients receiving conventional therapy who showed no significant improvement (Pulvermuller, et al., 2001). Researchers have noted that there is association between intensive therapy and improved aphasia outcomes (Bhogal, Teasell, Foley, & Speechley, 2003).

Another form of ILAT is Melodic Intonation therapy (MIT). For more severely afflicted patients, recovery potential of the left hemisphere language cortex may be severely limited; MIT helps these patients by reinforcing compensatory reliance on the non-language dominant right hemisphere, by putting words and phrases to a melody. The intonation at the heart of MIT was originally intended to engage the right hemisphere, given its dominant role in processing spectral information, global features of music, and prosody. The right hemisphere may be better suited for processing slowly modulated signals, while the left hemisphere may be more sensitive to rapidly modulated signals. Therefore, it is possible that the slower rate of articulation and continuous voicing that increases connectedness between syllables and words in singing may reduce dependence on the left hemisphere (Hyde et al., 2009).

Further studies that assessed the efficacy of CIAT for the treatment of aphasia confirmed its beneficial effects in patients with chronic and acute aphasia, and showed that this treatment can be performed efficiently not only by experienced therapists, but also by appropriately instructed and trained lay individuals (Berthier, Pulvermuller, Davila, Casares, & Gutierrez, 2011).

**Combining tDCS with Aphasia therapies**

The introduction of cortical neurostimulation techniques into the therapy session holds promise to take these advances to the next level of learning efficiency.

Emerging research suggests that the pairing of neuroplasticity-based speech therapy interventions with concurrent stimulation of relevant brain networks is likely to maximize therapy outcomes at a level unattained by either of type of intervention alone (Schlaug & Renga, 2008). If results of preliminary studies showing long-lasting benefits continue to be replicated, tDCS is likely to become a routine adjunct to language rehabilitation therapy, both in acute and chronic stroke settings.

Functional imaging studies have observed a compensatory shift in cortical activation patterns away from the lesioned left hemisphere during performance of language tasks immediately following acquired aphasia due to stroke. Observed compensatory
activation patterns during early recovery include a recruitment of undamaged left hemisphere regions distal from the lesion as well as an increased reliance on right hemisphere regions homologous to damaged left hemisphere language areas (Kim et al., 2002).

While scientists disagree whether this increased reliance on the contralesional cortex early in recovery represents an early compensatory pattern or is simply an example of mal-adaptive spontaneous neuroplasticity (Baker, Rorden, & Fridriksson, 2010), all agree that long term reliance on the right hemisphere appears to be an adaptive strategy only for the most severely afflicted patients, whose lesions are so extensive as to severely limit the capacity of the left hemisphere to resume its role in language production. This finding has guided researchers to focus on development of neurostimulation protocols to re-establish a balance between hemispheres via anodal stimulation of language-specific regions of the left hemisphere, or alternatively, via cathodal inhibition of the over-activated contralesional hemisphere, in order to guide and facilitate return to optimal activation patterns associated with efficient language processing (Schlaug, et al., 2008).

While studies using tDCS alone have shown significant short term effects on motor, language and cognitive skills, recent interest has shifted to the combined effect of traditional physical, language or cognitive therapies with simultaneous stimulation of strategically selected, task-specific brain regions for a further synergistic effect, the idea being that combined peripheral and central input can enhance synaptic plasticity, and promote speed, efficiency, and precision in skill re-learning at a level unattained by either of type of intervention alone (Schlaug & Renga, 2008).

To date, studies investigating the synergistic benefits of tDCS + aphasia therapy have been limited to small-N, proof-of-concept pilot designs, although several major NIH funded studies are already underway. Preliminary studies have found that tDCS stimulation targeted to specific regions of the language production network appears to facilitate language acquisition and verbal fluency in both normal healthy individuals (Fertonani, Rosini, Cotelli, Rossini, & Miniussi, 2010; Floel, Rosser, Michka, Knecht, & Breitenstein, 2008; Iyer et al., 2005; Sparing, Dafotakis, Meister, Thirugnanasambandam, & Fink, 2008) and aphasic stroke patients (Baker, et al., 2010; Fridriksson, Richardson, Baker, & Rorden, 2011; Kang, Kim, Sohn, Cohen, & Paik, 2011), with results typically demonstrating that naming accuracy and fluency can be improved by applying anodal-tDCS to the left hemisphere Broca’s region, while aphasic patients, but not healthy normals, appear to benefit from cathodal, or inhibitory, tDCS applied to the right hemisphere language homologue sites to support word production.

In several studies, aphasic patients receiving excitatory anodal stimulation to facilitate activation of left hemisphere regions comprising the language network, concurrent with speech training, demonstrated faster skill acquisition than those who received sham stimulation with speech therapy. Positive impact on naming skills has been reported with excitatory anodal stimulation over both Broca’s (Baker, et al., 2010) and Wernicke’s (Fiori et al., 2011) regions. Effective treatment protocols must be guided by unique patient-specific factors. For patients with more restricted lesions, cathodal inhibition of the contralesional right Wernicke’s homologue during language training appears to promote improvement in naming skills by releasing the perilesional left hemisphere language cortex from reciprocal inhibition effects of an overly active “helper hemisphere,” and promoting shifting of language back to the left hemisphere (Kang, et al., 2011). By contrast, for severely aphasic patients with limited left hemisphere recovery capacity, anodal stimulation of the right hemisphere Wernicke’s homologue results in improved naming skills, likely reflecting that for this more severe group, continued reliance on right hemisphere for language production is the best available outcome (Floel et al., 2011). Given these complexities, there is increasing interest in use of pretreatment structural MRI and fMRI data to map the margins of spared perilesional cortex in order to guide optimal electrode placement and protocol selection (Baker, et al., 2010; Fridriksson, et al., 2011).

Tantalizing early findings indicate that provision of tDCS concurrent with aphasia therapies not only boosts short-term within-session learning, but also appears to launch patients on a distinctively different long-term recovery trajectory than behavioral therapy alone (Fridriksson, 2011; Fridriksson, et al., 2011). If results of preliminary studies showing long lasting benefits continue to be replicated, tDCS is likely to become a routine component of stroke rehabilitation therapy, both in acute and chronic clinic settings.

Additional applications
Emerging research of tDCS technology extends its application to other acute and chronic neurologic conditions. As this non-invasive technique has been shown through various studies to have the utility of normalizing cortical excitability, rebalancing of distributed neural network activity, and induction of neurotransmitter release (Wu, Fregni, Simon, Deblieck, & Pascual-Leone, 2008), one can reasonably imagine a large number of neurologic and psychiatric illnesses that may be ameliorated in some way by non-invasive neurostimulation techniques such as tDCS. A review of current literature reflects groups of studies surrounding Parkinson’s disease, depression, motor ability post stroke, and chronic pain conditions, predominately.

In one such study of subjects with Parkinson’s disease, anodal tDCS applied to the motor and prefrontal cortices was introduced for eight sessions over two and one-half weeks (Benninger et al., 2010). Results indicated improved gait by some measures for a short time and also significantly improved bradykinesia for a duration longer than 3 months. Similarly, another study noted motor improvement of Parkinson’s patients with anodal stimulation of the M1 area of the motor strip, that was correlated with increases in motor-evoked potential amplitude and area. People suffering from Parkinson’s disease may also experience improvement of working memory through anodal stimulation of the left dorsolateral prefrontal cortex (LDLPFC), as was investigated by Boggio (Boggio et al., 2006). In this study, significant improvement of working memory was evidenced by anodal treatment of a 2mA treatment dose whereas 1mA, sham condition, and anodal stimulation of the motor cortex showed no improvement.

A recent meta-analysis was conducted that included tDCS as the focus of treatment for depression. The general findings where that tDCS seems to be an effective intervention for reduction of depressive symptoms, but effect sizes varied widely across the ten studies included in the meta-analysis (Kalou, Sexton, Loo, & Ebmeier, 2012). One of the more promising studies of major depression was that of Boggio et al. (Boggio et al., 2008). This investigation reported significantly larger reductions of depressive symptoms, as measured by the Hamilton Depression Rating Scale (HDRS), that averaged 40.4% after anodal stimulation of the LDLPFC, a 21.3% reduction after stimulation of the occipital cortex as an active treatment control condition, and 10.4% for the sham tDCS condition. Beneficial effects of tDCS in this 10-session, two-week treatment protocol indicated beneficial effects for the LDLPFC group persisted for 1 month after the end of treatment. A similarly conducted study with a group of treatment-resistant depression (TRD) subjects resulted in no significant treatment effects as measured by the HDRS when tDCS was used as a stand-alone treatment, although subjective mood ratings showed an increase in positive emotions after real tDCS, compared with sham tDCS (Palm et al., 2011). However, with another TRD population in a different study, tDCS was used as an adjunctive treatment to pharmacological intervention with previously poor responders. Results did confirm fairly robust treatment effects (Dell’osso et al., 2011).

Pain control has also gained attention as an area of application for tDCS. The primary motor cortex and dorsolateral prefrontal cortex have been the most frequently cited areas of stimulation across most current research. Substantial treatment effects for pain relief have been found through some investigations of central pain due to spinal cord injury as well as fibromyalgia. In two studies conducted by
the same lead investigator, tDCS application led to an experienced pain reduction that averaged up to 58% (Fregni, Boggio, et al., 2006; Fregni, Gimenes, et al., 2006). The treatment effect noted surpasses that of other similar studies utilizing rTMS. In regard to the psychological perception of pain, one recent study employed a new twist to tDCS, which they call high definition tDCS or HD-tDCS. The difference in technology was to use <12 mm diameter electrodes with the intent of increasing the spatial fociality of tDCS. Results of this study found HD-tDCS to be well tolerated; it also significantly decreased heat and cold sensory thresholds, decreased thermal wind-up pain, and produced a marginal analgesic effect for cold pain thresholds.

Another area that has received approximately as much research attention to aphasia treatment with tDCS has been hemiparesis following stroke. Study designs have included tDCS alone, in combination with traditional occupational therapy, with constraint-induced movement therapy (CIMT), as well as in concert with peripheral stimulation, among other methods. In a study of tDCS with occupational therapy, tDCS+OT resulted in significant improvement of range-of-motion in multiple joints of the paretic upper extremity. The effects lasted at least one week post-stimulation. Improvement in motor outcome scores was correlated with a decrease in fMRI activation in the contralesional motor region exposed to cathodal stimulation (Nair, Renga, Lindenberg, Zhu, & Schlaug, 2011). When paired with CIMT, another study demonstrated tDCS+CIMT gains were larger in the active tDCS group than sham control. Neurophysiologic measurements showed a reduction of corticospinal excitability in the unaffected hemisphere, and increased excitability in the affected hemisphere. This restoration of normal state asymmetry was apparent only in the active tDCS/CIMT group. A main conclusion of the study was that CIMT alone appears effective in modulating local excitability, but not in removing the imbalance in transcallosal inhibition (Bolognini et al., 2011).

**Considerations for safety and patient comfort**

While tDCS is safe, non-invasive, and painless, a mild transient tingling sensation during the first 30 seconds of stimulation is experienced by about 70% of participants. A phosphene, or brief flash of light may be experienced if an electrode is placed near to the eye (Nitsche et al., 2008). Less common symptoms, such as headache, are reported as frequently in the sham treatment condition as when the device is actually turned on (Fertonani, et al., 2010). In contrast to rTMS, tDCS does not directly induce cellular firing and seizures have never been reported. Moreover, as an additional safety precaution, patients with history of seizures, metal skull plates or skull breach, and cardiac pacemakers are typically excluded in tDCS research studies. (Bikson, Datta, & Elwassif, 2009; Iyer et al., 2005; Liebetanz et al., 2009; Priori, 2003) (Nitsche et al., 2008).

While higher current settings can lead to skin irritation under the electrode sponge in some individuals (Poreisz, Boros, Antal, & Paulus, 2007), significant patient discomfort has not been reported in studies utilizing a 1 to 1.5 mA stimulation level. Minor discomfort can be reduced or prevented by skillful clinical technique. Skin irritation during stimulation is reduced by thoroughly preparing the electrodes with saline solution and the skin with electrode cream. Subjects can be desensitized to tingling-associated discomfort by slowly “ramping up” the current during the first 10 to 60 seconds until the desired current is reached, or by momentarily reducing the current setting until the patient has become habituated to the sensation.

Clinical efficacy of tDCS is still considered investigational and its use in the rehabilitation setting is currently limited to IRB approved research (Arul-Anandam, Loo, & Sachdev, 2009). (References available on http://isnr.org/neurofeedback-info/neuroconnections-newsletters.cfm)
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Audio-Visual Entrainment as a Treatment Modality for Posttraumatic Stress Disorder—Part II

Dave Siever

Abstract

Posttraumatic stress disorder (PTSD) is the aftermath of trauma. Trauma spans a diverse spectrum of unfortunate life experiences such as sexual abuse, assault, car accidents, war, and natural disasters. PTSD occurs when the afflicted can no longer mentally cope with the situation. Following trauma, permanent changes occur within the brain that increases “racy-headedness,” guardedness, anxiety, depression, insomnia, plus memory and cognitive impairments. The behavioral aftermath of PTSD also typically involves increased aggression and drug and alcohol abuse. Audio-visual entrainment (AVE) has been shown to reduce anxiety and insomnia and improve coping for police officers and military. AVE has also been shown to reduce depression and anxiety among veterans with chronic fatigue syndrome and fibromyalgia.

Introduction

The aftermath of PTSD leaves the afflicted with serotonin deficiency, a racy head, and either dangerous behavior or depression. Audio-visual Entrainment (AVE) has been shown to stop a racy head and increase serotonin, thus boosting the relaxation response and rebalancing brain wave activity, which therefore reduces aggression and depression.

Audio-Visual Entrainment as a Treatment Modality for PTSD

All sensory information, except for smell, must pass through the thalamus in order to gain access to other brain regions. When lights are pulsed into the eyes or tones pulsed into the ears, the nerve pathways from the eyes and ears carry the evoked potentials into the thalamus. When a repetitive stimulus of the proper frequency and sufficient strength to excite the thalamus is present, their frequency signature is shown in the EEG. From there, the entrained electrical activity within the thalamus is amplified and distributed throughout other limbic areas and the cerebral cortices via the cortical-thalamic loop. This is a signaling loop between the cerebral cortex and the thalamus that generates the alpha rhythm at roughly 10 Hz during neuronal rest (Demos, 2005). This effect of modulating the cortical-thalamic loop with light and sound is known as audio-visual entrainment (AVE). In essence, AVE is the continuous electrical response of the brain in relation to the frequency of the stimuli plus the mathematical representation (harmonics) of the stimulus wave shape.

AVE has also been shown to reduce depression and anxiety among veterans with chronic fatigue syndrome and fibromyalgia.

The Digital Audio Visual Integration Device (DAVID) AVE devices present pulsed light to the user via a pair of sunglasses (TruVu Omniscreen™ Eyeset (Siever, 2005)) with an array of flashing LEDs and pulsed tones through a pair of headphones. Because most maladies have an abnormal brain wave signature, the DAVID AVE device can help treat a host of maladies, including anxiety, depression and insomnia, impact of trauma, a racy mind, attentional disorders, fibromyalgia and cognitive decline, and risk of falling in seniors.

Photic Entrainment was discovered in 1934 by Adrian & Matthews and occurs best near one’s own natural alpha frequency from 9 to 11 Hz (Toman, 1941). The results of a study by Kinney et al, (1973), shown in Figure 1, shows strong and pure entrainment at 12 Hz. The harmonics (small wavelets) seen in the EEG are a reflection of the harmonics produced in the EEG from the Xenon square-wave, strobe-light stimuli.

Entrainment primarily shows itself in the frontal, central, and parietal regions. (Siever, 1998). Given that PTSD most commonly causes enhanced beta and suppressed alpha activity coincident with high arousal (Jokić-Begić’, 2003), AVE can rapidly re-
disorders. A 16 Hz, second harmonic is also present (the circled image), which is typical of semi-sine wave (part sine-wave, part square-wave) stimulation.

AVE at 18.5 Hz has also been shown to produce dramatic increases in EEG amplitude at the vertex of the head (Frederick, Lubar, Rasey, Brim, & Blackburn, 1999). It was found that:

- Eyes-closed 18.5 Hz photic entrainment increased 18.5 Hz EEG activity by 49%.
- Eyes-open auditory entrainment increased 18.5 Hz EEG activity by 27%.
- Eyes-closed auditory entrainment increased 18.5 Hz EEG activity by 21%.
- Eyes-closed AVE increased 18.5 Hz EEG activity by 38.3%.

**Normalizing EEG Activity in Depression**

Studies suggest that a significant number of those with PTSD develop depression, characterized by frontal alpha asymmetry with more left frontal alpha activity as compared with right frontal alpha activity (Gordon, et al, 2010; Rabe, et al., 2008).

A. To help treat depression from an electro-neuro perspective and re-balance the frontal lobes we need to re-excite the left frontal lobe (the happy side) and suppress the right frontal lobe (the fear-based side). Two conditions must be met: We must inhibit the excessive left-frontal alpha (thus “waking” it up).

B. We must simultaneously boost right frontal alpha (calming it down). Therefore, we need a means whereby we can affect both frontal lobes independently of one another.

It has been shown (above) that AVE clearly increases alpha and beta activity, but to treat depression, we also need a way that can suppress alpha in the left frontal lobe. Visual entrainment has been found to inhibit brain wave activity at the ½ frequency of stimulation, thus satisfying condition A. Figure 3 shows an ADHD child with aberrantly high theta, which 14 Hz visual entrainment was used to suppress at the 30-minute mark. (Collura & Siever, 2009). Notice how rapidly the excessive theta disappears. In the case of depression, we can stimulate with 20 Hz and inhibit the 10 Hz alpha activity.

Independent hemispheric stimulation is accomplished by utilizing the optic chiasm (Siever, 1995), thus satisfying condition B. Stimulating the right fields of both eyes with a different frequency than the left fields, as shown in Figure 6, can accomplish this. Figure 4 depicts stimulus “A” at 12 Hz and stimulus “B” at 4 Hz. Notice the corresponding frequency evident in the opposite hemisphere of the brain. For treating depression, 18–21 Hz stimulation in the right fields would inhibit the left-frontal alpha from 9–10 Hz, thus boosting activity. Stimulating at 10 Hz in the left fields will boost right-frontal alpha, thus calming activity.

Figure 5 shows the QEEG (also referred to as a brain map) of a happy person as compared to the Sterman-Kaiser Imaging Labe (SKIL) normative database. This person constantly exhibits approach behavior towards socializing and what she considers to be fun activities. Notice that the alpha activity is stronger in the right frontal lobe, the EEG signature typically associated with happiness.

Figure 6 shows a fairly typical brain map of a person with depression and anxiety from trauma as shown on the SKIL database. The scale is 2.2 standard deviations (SD) and the pink area in the alpha view is actually 2.6 SD. Activity above 2 SD is considered a clinical abnormality. Notice that alpha activity is higher on the left side coincident with a personality trait based on a focus of withdrawal and avoidance from self-perceived negative stimuli. Also, the generalized red colored region is an indicator of generalized cognitive fatigue. The Beta 2 activity is just approaching 2.2 SD (an indication of mild anxiety). Non-clinical persons have greater right frontal alpha associated with an attraction toward positive stimuli (Demos, 2005).

Approximately 10 minutes following a 30-minute AVE session designed to reduce the symptoms of depression, both alpha and beta activity is normalized as shown below in Figure 7. Notice that the frontal alpha activity and the Beta 2 activity have been reduced to roughly 1.2 SD above the norm (non-clinical). The participant was also well aware of his elevated mood and energy.
Body/Mind Effects of Audio-Visual Entrainment

We conceptualize AVE as achieving its effects through several mechanisms at once (Siever, 2000). These include:

1. dissociation/hypnotic induction
2. increased neurotransmitter production
3. increased cerebral blood flow
4. normalized EEG activity

Dissociation

Dissociation, as a tool in psychotherapy, helps in diminishing the emotional component of disruptive memories. Dissociation, when referring to AVE, is a disconnection of self from thoughts and somatic awareness, as experienced during deep meditation (Figure 8). AVE-induced dissociation is rapid, requires only 4 to 10 minutes in most cases, and provides an excellent means for clearing a tormented, racy mind of destructive, fearful thoughts and allowing the person to relax and restabilize (Siever, 2000).

Visual entrainment alone, in the lower alpha frequency range (7 to 10 Hz), has been shown to easily induce hypnosis (a form of dissociation). It has been shown that nearly 80% of subjects enter into a hypnotic trance within six minutes during alpha photic entrainment (Kroger & Schneider, 1959), as shown in Figure 9.

Inducing dissociation using AVE delivered by the DAVID1 was found to be more effective than dot staring or stimulus deprivation (Leonard, et al., 1999). AVE using the DAVID Paradise demonstrated to be effective in clinically dissociating people with dissociative anxiety while simultaneously calming them down somatically and reducing their heart rate (Leonard, et al, 2000). As a result, AVE may be used directly to stop the distressing mental chatter and as an effective desensitization tool for reducing dissociative anxiety that is sometimes seen in the PTSD population.

Limbic Stabilization

As mentioned, the amygdala initiates the activation of the fight-or-flight response, which activates the hypothalamus, which in turn controls all autonomic functioning and is responsible for the tensed-up feeling in the body (chest breathing, shortness of breath, racing heart, cold, clammy hands, tense muscles, etc.) that is experienced during a fear response. Anyone who has consumed too much coffee will be familiar with these feelings.

Properly applied AVE produces a calming effect on limbic structures, such as the amygdala and hypothalamus, in which muscles relax (Thomas & Siever, 1989), electrodermal activity settles down, peripheral blood flow stabilizes (hand temperature normalizes to 86 to 90 F), breathing becomes diaphragmatic and slow, and heart rate slows and becomes uniform (Siever 2000). As a result, AVE can re-induce a relaxed state of mind and calm disposition, thus providing some badly needed time away from the distressing thoughts.

Figures 10 and 11 show the calming effect of AVE on the somatic functions of forearm EMG (electromyography) and finger temperature (Hawes, 2000). Heart rate and heart-rate variability (HRV) are sensitive measures of stress (Stein & Kleiger, 1999). Figure 12 shows graphs of the emWave HRV analysis system by HeartMath. It shows dramatic improvements in both heart rate and HRV in a woman who developed PTSD after discovering that her husband molested two young girls. Within 10 minutes, her heart rate dropped by 22 bpm, and she showed dramatic reductions in both sympathetic and parasympathetic activity (notice the blue mountains).

Using AVE to Balance Neurotransmitters

As mentioned previously, people with lingering PTSD and clinical depression are low in serotonin, dopamine, and norepinephrine.
Figure 13 shows that 30 minutes of white-light AVE at 10 Hz increased serotonin levels by approximately 23%, with endorphin and norepinephrine levels increased by 18% (Shealy et al., 1989), leading to increased hopefulness, self-esteem, mental sharpness, improved sleep, reduced pain, and reduced anxiety.

Cerebral Blood Flow and Metabolism

SPECT and FMRI imaging of CBF show that hypoperfusion of CBF is associated with many forms of psychiatric disorders. Of particular concern are conditions involving hypoperfusion of CBF in frontal lobes. Frontal lobe issues include anxiety, depression, attentional and behavior disorders, and impaired cognitive function (Amen, 1998). Adequate CBF is essential for good mental health and function. AVE increases brain glucose metabolism and CBF (Sappy-Marinier et al., 1992). Figure 14 is a study by Fox and Raichle (1985) showing marked increases in CBF, with a 28% peak increase at 7.8 Hz in the striate cortex (a primary visual processing area). Overall whole brain oxygen consumption increased by 5%. Accomplished Zen meditators show a peak frequency of 7.8 Hz during meditation (Cade, 1987).

In addition, AVE has also been shown to increase CBF throughout various other brain regions including frontal areas (Mentis, et al., 1997; Sappy-Marinier, et al., 1992). A whole-head PET analysis of visual entrainment at 0, 1, 2, 4, 7, and 14 Hz on 19 healthy, elderly (mean age, 64 years) subjects (Mentis, et al., 1997) found that regional cerebral blood flow (rCBF) was activated differentially with the:

1. Left anterior cingulate showing maximal increases in rCBF at 4 Hz.
2. Right anterior cingulate showing decreases in rCBF with frequency.
3. Left middle temporal gyrus showing increases in rCBF at 1 Hz.
4. Striate cortex showing maximal rCBF at 7.8 Hz.
5. Lateral and inferior visual association areas showing increases in rCBF with frequency.

Studies

In 1995, David Trudeau, a physician with the VA Hospital in Minneapolis conducted a study on 15 war vets, all suffering from PTSD. The volunteer subjects received 60 daily sessions of AVE at 18 Hz. Pre- and post-intervention, QEEGs, Beck Depression Inventory (BDI), McGill Pain Questionnaire (MPQ), Test of Variables of Attention (T.O.V.A.), and DSM-IV Attention Deficit and Hyperactivity Disorders (ADHD) symptom checklist were done.

As of summer 1999, ten subjects had completed the study. Following 60 daily sessions of 15 minutes of AVE at 18 Hz, there was a significant decrease in BDI scores from an average of 17 to 9 (p<0.05) and DSM-IV impulsivity-hyperactivity criteria from 3 out of 9 to 0 out of 9 (p<0.01). Consistent with the decrease in self-assessed impulsivity is a trend toward decreased impulsivity on the T.O.V.A. Anecdotally, subjects reported onset of dreaming as well as improved sleep and higher energy levels. Focus may be improved following AVG, and depression symptoms may be improved. Clearly more study is required, and further trials should include sleep assessment.

John Carmichael’s PTSD Work with the Royal Canadian Mounted Police (2006) Dr. Carmichael is the approved and designated clinical psychologist to the Royal Canadian Mounted Police in British Columbia, Canada. Currently, most of his private practice in clinical psychology is with police officers who most typically present with depression in which accumulated traumatic incidents have played a significant role, with PTSD, or with both depression and PTSD. Given that the police have a tendency to wait until the last minute for treatment, their symptoms are very marked in both number and intensity and have been ongoing for a considerable time.

However, since discovering our DAVID technology, Dr. Carmichael now includes audio-visual entrainment for all of his police clients with depression and/or PTSD once there is psychophysiological confirmation that they have mastered diaphragmatic breathing, that they can establish a respiratory sinus arrhythmia (RSA) pattern, and that the entrainment creates desirable changes.

Most police officers continue to use the DAVID AVE devices on a daily basis. It is clear that well over 90% of his police clients find the DAVID helpful. Among the most common findings are:

- A rapid decrease in both autonomic nervous system hyper-arousal/hyper-reactivity and muscle tension (I show clients the changes during their first session with DAVID in my office).
- A longer duration of these positive effects the more frequently they use the DAVID.

- A rapid increase in mental calmness and corresponding decrease in “monkey mind” (thoughts all over the place).
- Rapid improvements in sleep (reduced latency to sleep onset, decreased night waking, and increased sense of restfulness come morning) when they use it at regular bedtime and again if they wake during the night and are unable to fall back asleep within 15 minutes.
- What appears to be self-initiated changes in both behaviour and cognitions even before any formal introduction of cognitive behavioral therapy.
Conclusion

Chronic rumination, hypoperfusion of CBF, loss of neurotransmitters, altered brain wave activity, and adrenal fatigue all contribute to PTSD and the continuation of PTSD. These effects also play a part in anxiety, bodily ailments of all kinds, aggression toward family and civilians at large, depression, substance abuse, and loss of work productivity. Interventions to help those with PTSD are poor at best and can have significant, unwanted side effects.

The DAVID AVE dissociates those experiencing PTSD from destructive distressing rumination, increases blood flow, normalizes brain wave and neurotransmitter production, calms the limbic system, restores the adrenals, and produces somatic relaxation. The subjective benefits of AVE are reduced anxiety, improved sleep, improved mood, increased energy, improved relationships with family and civilians, reduced physical problems, improved productivity and reduced dependence on medications or self-medicating with alcohol and recreational drugs.

There are hundreds of anecdotal cases of childhood and adult trauma, including abused women, police, and emergency personnel, confirming the benefits of AVE as a treatment methodology. AVE has been shown to reduce depression and impulsiveness while improving sleep in war vets with either chronic fatigue syndrome or fibromyalgia syndrome (Trudeau, 1999). AVE also has a proven history in treating posttraumatic stress-related disorders for the Royal Canadian Mounted Police (RCMP) in Kamloops, British Columbia, where 90% of the officers respond with improved sleep onset and quality of sleep and with reduced daytime anxiety.

Dave Siever of Mind Alive, Inc. has lectured and provided workshops with leading psychological organizations including the Association of Applied Psychophysiology and Biofeedback, the International Society of Neurofeedback and Research, the College of Syntonic Optometry, American College for the Advancement of Medicine, Walden University, the University of Alberta, Open University-England, A Chance to Grow Charter School, STENS Biofeedback Training Programs, and other venues. Dave Siever has been designing and studying AVE since 1984 when he originally developed the DAVID1 to help performing-arts students overcome stage fright.

References

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*Continuing education applications on file with SmithBucklin for APA, AMA, MFT and LCSWW
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BrainAvatar is the next generation of BrainMaster software, supported by the Atlantis and Discovery EEG devices. It is the successor to Atlantis 3.7i, and Discovery 1.5.9, and is designated the 4.0 release. It includes all existing features and can run all existing protocols, including peripheral biofeedback, but include several innovative new capabilities, including live 3-D brain imaging for assessment and biofeedback, using the sLORETA algorithm (Pascual-Marqui, 2002).

The important new capabilities of BrainAvatar include:

• Programmable tabbed screens, 8 tabs per screen.
• Easier use of folders and settings files; ability to run from a desktop shortcut.
• Special second screen with 8 tabs for EEG review or client.
• 16 frequency bands instead of the original 8.
• New contoured and 3-D-look display panels.
• A 24-channel EEG simulator for testing, research, and sham feedback.
• Fast 2-D live topographic maps (“flat maps”) (raw or z-scores).
• Fast 3-D live topographic maps on realistic head (raw or z-scores).
• Instantaneous live sLORETA projector with real-time 3-D imaging.
• Z-Builder (creates z-score templates, allows users to build reference databases).
• EEG Review and Edit screens, integrated with LLP and Z-Builder.

BrainAvatar contains all existing displays, games, etc. from our previous software. In addition, the LLP display provides a powerful, new feedback display. Rather than a graphic, sound, movie, or other display, the client watches his or her own brain activity, instantaneously. When the Region of Interest or single voxel change, the client sees it instantly, within 30 milliseconds. This provides unprecedented resolution and acuity in the feedback display. The client could see which voxels are active, their precise location, and when they activate. This provides new mind-brain connection. Similar to an fMRI in its imaging ability, this method is more than 1000 times faster. It reflects true brain electrical activity, scientifically localized in real time.

These features combine to provide an unprecedented level of flexibility and ease of use. Each of the eight tabbed screens is fully programmable and can contain any displays or controls, so changing screens is now a single mouse click.

The 16 frequency bands allow the use of specific frequency ranges, and are adjustable with a resolution of 0.0001 Hz. Even slow-cortical potentials or infra-low EEG (ILF) signals can be imaged and trained. The EEG simulator is useful for testing, teaching, demonstration, and sham feedback applications. However, the most significant new features are the sLORETA-based Live LORETA Projector (LLP) and the innovative Z-Builder z-score reference creation tools.

BrainAvatar also includes a complete set of live “flat” brain maps, as well as surface maps rendered on a 3-D head. These can be raw or z-scored maps, and can be change maps, so that a client could see what is changing in time, or what has changed since a last session.

The LLP Live LORETA Projector provides instantaneous, real-time sLORETA-based brain activity images, at a rate of 32 frames per second. This provides a brain functional imaging system that operates at brain speeds, and has high-resolution imaging capability.
BrainAvatar uses the sLORETA ("standardized LORETA") mathematics and theory, but is an entirely original implementation by BrainMaster. No software from the Key Institute is used in our system, although it is possible to use the Key Institute’s LORETA and sLORETA software packages using BrainAvatar data, for research purposes. The Live Loreta Projector (LLP) provides over 30 3-D sLORETA images per second, and images can be positioned, rotated, or auto-rotated as needed for assessment or neurofeedback.

We chose to use sLORETA as the basis for BrainAvatar, rather than LORETA. Despite the fact that sLORETA requires more computations, it has higher resolution and its guaranteed zero-error localization are critical for neurofeedback applications. LORETA has a non-zero localization error, which compromises its accuracy and usefulness for neurofeedback. For any applications, particularly for live imaging and neurofeedback, sLORETA is technically superior to LORETA.

The sLORETA algorithm is based on the work of Dr. Roberto Pasqual-Marqui (2002), and has been widely validated for its accuracy. It provides a resolution of 5 mm per voxel, and has demonstrated use in a variety of studies. The most important aspect of the LLP is its speed and real-time 3-D imaging capability. The ability to visualize brain activity at the voxel-level, in real time and in multiple frequency bands, is a new capability that has not been available in any other systems. This introduces a new look and feel to neurofeedback, taking feedback from the world of simple displays, games, and videos, into witnessing one’s own new brain activity.

**Differences between LORETA and sLORETA**

The following are the major differences between LORETA and sLORETA:

- sLORETA is the successor to LORETA, introduced several years later, by Dr. Pasqual-Marqui (2002). (the yet more advanced eLORETA is now available, as well). For our purposes, sLORETA is more than sufficient, whereas LORETA would have been not quite adequate.
- sLORETA has 6,239 voxels, compared to LORETA’s 2,000+ voxels.
- sLORETA uses 5-millimeter voxels, compared to LORETA’s 7-millimeter voxel size.
- sLORETA does not include the amygdala, while LORETA includes the amygdala, but it is not reliable.
sLORETA has a guaranteed zero localization error, while LORETA has a non-zero localization error.

sLORETA, like LORETA, contains ROIs and Brodmann Areas.

sLORETA has more voxels per Region of Interest, hence provides higher spatial resolution.

sLORETA is based on the same basic theory as LORETA, but reduces its sensitivity to noise. It has been extensively validated against MRI and other direct imaging methods, particularly with regard to its zero-localization error.

In the BrainAvatar, each voxel is converted into a current source density, and three spatial values (vectors) that describe the dipole. These spatial components are also relevant to connectivity. Each voxel represents an “analog” number, the local level of activity. In BrainAvatar, all projected signals are time-domain signals. This provides superior speed and accuracy, compared to FFT-based approaches, which introduce delays and possible distortion, due to the epoch size limitations. FFT-based methods also show only averaged activity, not instantaneous values. BrainAvatar computes the sLORETA projection using high-speed time-domain methods, and accurately shows the momentary changes in EEG signals. The combination of BrainMaster’s high-speed digital filters with our unique projection technology provides the ability to compute hundreds of whole-brain sLORETA projections per second, and image and train on the data. Trained data can be from voxels or Regions of Interest (ROIs). All values are available through the flexible Event Wizard interface, so that an entire sLORETA training protocol can be inserted with a single, very simple event. An example of ROI training using the occipital area is shown in Figure 8.

The LLP system is also interfaced with the built-in JFTA and quadrature digital filter system, using up to 16 different frequency bands. In BrainAvatar, frequency-based training does not depend on FFT or related transform techniques. These can introduce delays, and require an “epoch” in order to process the frequency data with adequate resolution. Rather, all sLORETA projection in this system is done in the time-domain, on live filtered data. This means that, when imaging an alpha wave, for example, each and every cycle of every wave is processed and imaged, one sample at a time. This preserves phase information as well, and means that the sLORETA projector has access to individual wave events as quickly as possible.

There are many ways to use this data, as this is a toolkit, integrated with all of our existing software, including the Event Wizard. Figure 9 shows a design used by Dr. Neils Schnepel, training down 12-20 Hz on Brodmann Area 4. The pre/post QEEG maps show the effects of a single 20-minute session:

**BrainAvatar sLORETA ROI and Voxel Training**

- Based upon 88 ROIs
- Can train any band, any ROI
- Resolves and combines every voxel in ROI
- Each voxel or ROI provides a CONTINUOUS variable
- Event Wizard Interface
- Fixed or dynamic thresholds
The NeuroConnections SUMMER 2012 page contains text about the sLORETA LLP interface training software, which is user-programmable and provides a wide range of options. It allows for the following:

- Combine any number of ROIs
- Simultaneous display of ROI on screen
- 30 msec maximum delay from EEG event to screen or sound
- The individual source density for any voxel or voxels can be read out and used directly.
- The combined source density amplitude for all voxels in an ROI can be measured.
- Dipole moments of individual voxels or ROIs can be measured and imaged.
- The number of voxels in an ROI that meet a z-score criterion can be counted and trained.
- The number of voxels in an ROI that meet any other criterion can be counted or trained.

LLP feedback does not depend on a simple on/off response for a voxel or ROI; it provides continuous, proportional data. In addition, many types of on/off features can be defined. With the LLP, it is possible to do multivariate proportional feedback, such as Percent Z-OK (PZOK) (Collura, 2008a, 2008b; Collura et al., 2009; Collura et al., 2010; Festa et al., 2009) on ROI. The practitioner can look at theta in the posterior cingulate, for example, and get a training variable that represents the percent of voxels in that ROI that meet a criterion or evaluate and train total and average activity of the entire ROI on a representative basis. If a clinician wants to select a single voxel, that can be measured and trained as well, using the Event Wizard.

Although live z-scores can be imaged and trained using various reference sources, BrainAvatar does not depend on z-scores to image brain activity. The LLP provides raw brain activation data, showing what is happening, not what is not happening. As noted by Leslie Sherlin (2009, p. 94), when one understands EEG and brain processes, it is not necessary to use normative z-scores in order to view and assess LORETA data. Raw data are an important dimension of visualizing brain activity when imaged in this way. BrainAvatar provides raw EEG-based brain electrical imaging, as well as the ability to visualize changes or differences when compared to other references. Those references could be normal but do not have to be normal. They could even be better than normal.

LLP neurofeedback is qualitatively different from any previous forms of neurofeedback. This is the first implementation that gives the trainee a complete, instantaneous, 3-dimensional view of his or her own brain activity.
Figure 12: “Mini” Live sLORETA Projection using F3, F4, C3, and C4. This reveals the degree of asymmetry and anterior-posterior energy, showing regionalization of activity.

In addition to what has now become the standard in normative live z-score training, this system now adds a new concept, that of template-based training. Template-based training is based upon the client’s or someone else’s actual QEEG profile, which can be used to create training protocols tailored to each specific client. While this is still live z-score imaging and training, the reference is no longer necessarily normal. It can be any type of individual, in any state, including the client’s own reference state, or a different individual, for peak-performance or optimal functioning training.

Other applications are recording clients’ brain activity under different conditions such as relaxation, reading, and speaking, or observing the changes during interventions, and their reactions to tasks.

The data regarding voxel dipole moments and vector components is an important new dimension in neurofeedback. These parameters are relevant to the pattern of pyramidal cell dipolarization in each voxel, as well as connectivity factors. Different vector components reflect different connectivities, hence provide a new way to assess and train brain connectivity, using sLORETA voxel data.

When used with less than 19 channels, the LLP provides information that is related to regionalization, if not complete localization. For example, Figure 12 shows a projection achieved using only 4 sensors, F3, F4, P3, and P4. Although the central and peripheral areas of the brain are not represented, this image nonetheless makes it possible to image the amount and type of activity in the four major lobes, which are covered by the sensors.

**Possible protocols could be:**

- Train the anterior cingulate gyrus to increase its beta activity.
- Train down theta and uptrain beta in the anterior cingulate.
- Train “C3 beta/C4 SMR” using the sensorimotor cortex, not just the EEG sites.
- Train down theta in the anterior cingulate and train up beta in the posterior cingulate.
- Train the dorsolateral frontal lobes to have more beta than the ventromedial frontal lobes.
- Reward alpha activity from the frontal area, if it occurs after a posterior alpha burst.
- Implement an asymmetry protocol between any two ROIs; that is, train (ROI1 - ROI2)/(ROI1 + ROI2). Rather than depending on F3-Cz and F4-Cz as leads, you could use the relevant dorsolateral frontal lobe regions and train them directly.
sLORETA ROIs used in LLP

Frontal Lobe
Limbic Lobe
Occipital Lobe
Parietal Lobe
Sub-lobar
Temporal Lobe
Angular Gyrus
Anterior Cingulate
Cingulate Gyrus

Cuneus
Extra-Nuclear
Fusiform Gyrus
 Inferior Frontal Gyrus
 Inferior Occipital Gyrus
 Inferior Parietal Lobule
 Inferior Temporal Gyrus

Orbital Gyrus
Paracentral Lobule
Parahippocampal Gyrus
Postcentral Gyrus
Posterior Cingulate Gyrus
Precentral Gyrus
Precuneus
Rectal Gyrus

Sub-Gyrual
Subcallosal Gyrus
Superior Frontal Gyrus
Superior Occipital Gyrus
Superior Parietal Lobule
Superior Temporal Gyrus

Gyrus
Supramarginal Gyrus
Transverse Temporal Gyrus
Uncus

Brodman areas 1-11, 13, 17-25, 27-47

References

BrainAvatar, Live sLORETA Projector, LLP, and Z-Builder are trademarks of BrainMaster Technologies, Inc. US, Canadian, and foreign patents and patents pending.

References for Letter from AAPB Co-Editor

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Acquired Brain Injury: A Case Study

Ann Marie Brown

This is a case study of a 19 Year old boy, Jake, whose parents sought neurofeedback treatment after an acquired brain injury as a result of snorting Ritalin. After a drinking binge and snorting Ritalin, he wandered around the streets by his college and did not know where he was. He knew enough to contact his mother from his cell phone. She arranged for an aunt to take him to the local ER and directed him over the phone back to his apartment. He was brought to the emergency room for an evaluation and was hospitalized in the psychiatric unit of that hospital. He was diagnosed with drug-induced psychotic episode. It was advised that this boy remain in that hospital for treatment but the parents moved him to the hospital near his home town. He remained in this hospital for 10 days. He was prescribed the following medications: Abilify, 15 mg once per day and Benztropine, 1 mg twice per day. Upon discharge, the patient began to see a therapist to address his stressors and the consequences of his choices of drugs and alcohol. He was also set up with an outpatient psychiatrist. The outpatient psychiatrist prescribed Paxil to deal with his anxiety and depressive feelings. He suffered a negative effect and it was discontinued after a week. He was then prescribed Welbutrin but that also had the same negative side effect of confusion and unresponsiveness.

Jake’s history was unremarkable. He reached developmental milestones without delays. He was of average intelligence, athletic, and played tennis, softball, and soccer in high school. His grades were good. He was attending college full time. He has a family history positive for depression on the paternal side, and for thyroid disorder, diabetes, arthritis, and cancer on the maternal side.

At the time of intake, Jake was experiencing great difficulty in expressive and receptive speech, had interrupted sleep throughout the night, and had a flat affect, difficulty with motor planning and organizing thoughts. He had difficulty trying to get through a day along with severe anxiety riding in a car and processing quick or fast movements around him. He also suffered from daily headaches. In the office it was evident that Jake had great difficulty comprehending simple one-step directions. He also had a great deal of trouble answering a question, and needed a lot of time to formulate his words. He had no eye contact and moved with a lot of rigidity.

Because Jake’s level of functioning before his incident with the drugs and alcohol was high, and because of his difficulty with motor planning, speech issues, affect, and sleep, I decided to proceed with the Low Energy Neurofeedback System (LENS) map to ascertain where there were difficulties in the brain after this incident. He had difficulty with anxiety, sitting still for any period of time, and closing his eyes for long. Sam’s map showed a great amount of suppression overall. He also had a high level of amplitude of slowed activity in the frontal lobe. Because of these factors, I felt that the LENS was a good approach to use.

I used the LENS treatment-by-treatment report form to monitor symptoms throughout treatment. He started with his five top symptoms which included inability to get through a day, interrupted sleep, speech issues, motor planning, and headaches. All symptoms began with a rating of 10 being the worst. He felt symptom changes after his map, which brought all symptom ratings down dramatically. When Jake began his LENS treatment, he was 23 days post psychotic episode. He was taking 15 mg Abilify and 1 mg benztropine 2 times per day.

I followed the total amplitude site sort. I used an offset of +20 with 2 seconds of stimulation and treated five sites each session.

After his first LENS treatment session, Jake was very tired but slept through the night and late the next day. That following day he also had an increase of frequency and duration of headaches, but also had an increase in his ability to find words and communicate with others.

By the end of the following week, Jake became more irritable. His psychiatrist decreased the Benztropine to 1 mg per day. He then had a reaction to coffee, which produced wired feelings for days afterwards. His anxiety also continued to be an issue.
Jake continued to have issues with anxiety. He started using HeartMath® and was able to practice this between sessions. But by Jake’s eighth session, he began to feel tired after sessions. I changed his stimulation to one second and remained at five sites. His parents reported to me that this week they had noticed increased reading ability, comprehension, energy, and sleep quality. He had a decrease in headaches, agitation, and jitteriness. His speech was reported as “back to normal.” He was able to communicate to me accurately and able to respond to conversation without issues. He did continue to report difficulties with organizing and finishing what he had started.

By week 15, Jake was able to take on a full time job. He finished tests that needed to be completed at his college, and did well. The new job fatigued him easily but he was motivated to continue to work. He began to take Biostrath, a natural stress and fatigue formula that contains yeast and an herbal blend to help with focusing, attending, and organizing. He noticed this was very helpful. He also noticed he had more difficulty in the afternoon than in the morning. It seemed his brain continued to fatigue as the day went on. Jake’s self-awareness and reporting were very accurate according to his parents.

By session 23, Jake had no more headaches. His sleep and energy were all reported good. His speech and motor planning seemed back to normal. He planned to return to college in another month.

Jake continued to stop in for sessions when he was home from college. He had six monthly follow up sessions and did not require the same amount of stimulation to keep him on track. He received treatment at two sites at +20 1 second of stimulation. He was able to ask for sites he felt he needed most and that was usually FP, FP1, or FP 2 for treatment.

Figures 2–6 above are a tabulation of each session’s reported symptom level with a 1-10 scale used, in which 10 was the worst level. As can be seen, he registered 10 on all the symptoms on the first day, and 23 sessions later all were zero except organizing, which was rated as one.

This case suggests the value of the LENS method for treating acquired brain injury due to drug toxicity and its relative shortness of treatment for this very debilitating disorder. The resolution of the symptoms, which enabled him to graduate from college and become gainfully employed, illustrates that this is a therapy which clinicians versed in the LENS treatment should consider using.

1. HeartMath is a breathing training program that has a coaching screen and can be set at the best breathing rate for the client. The program uses a thermal sensor or ear clip and measures blood flow with the information displayed on the computer.
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