

NEURO CONNECTIONS



Spring 2011 **Newsletter**

A joint newsletter from the  ISNR
& the  apb Neurofeedback Division

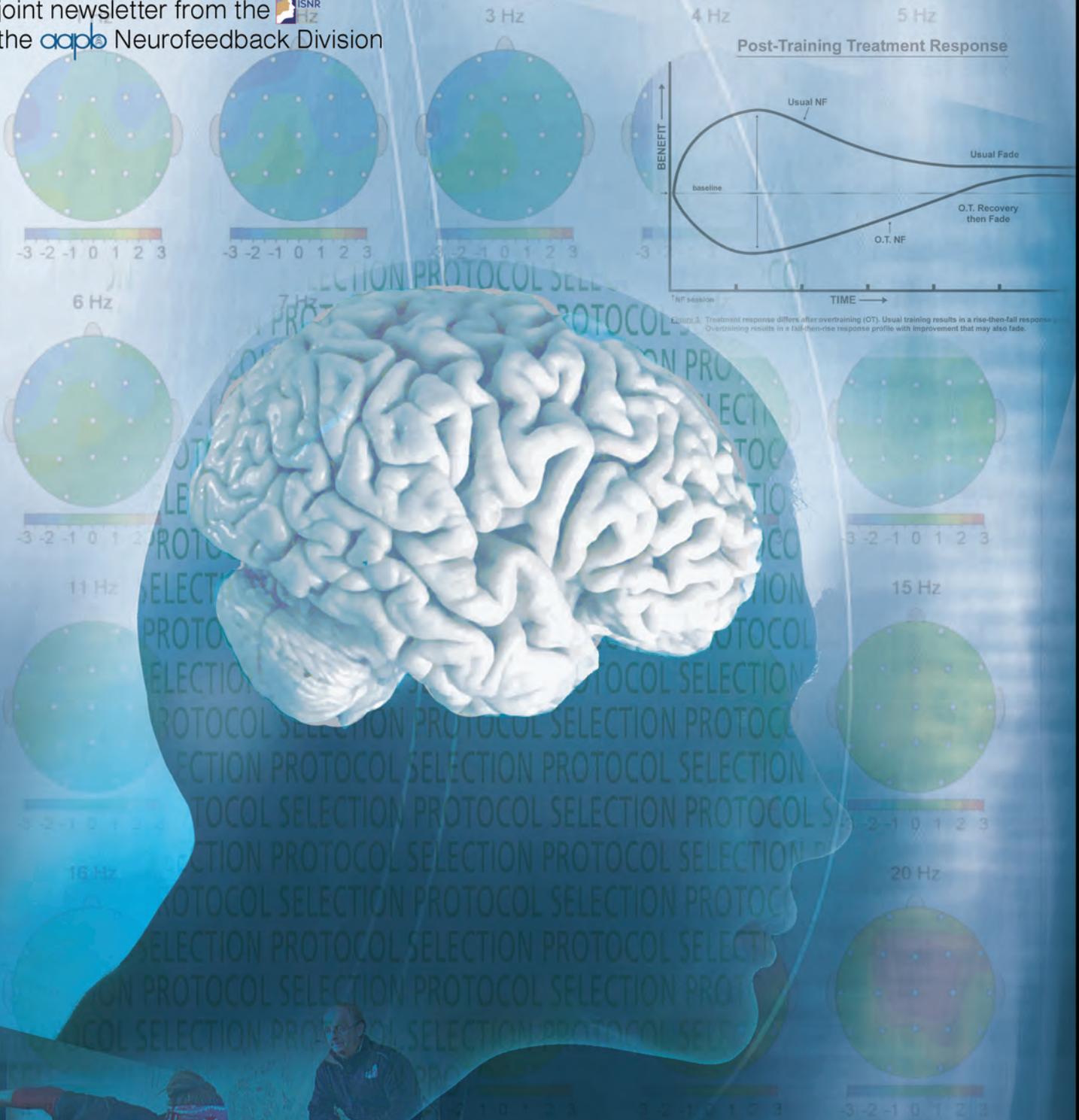


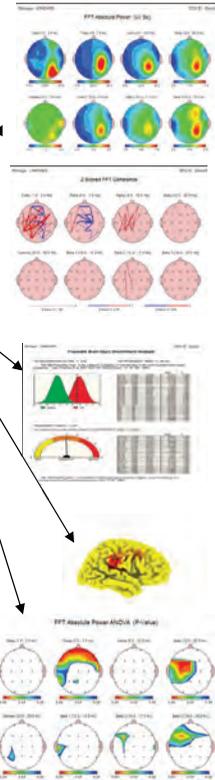
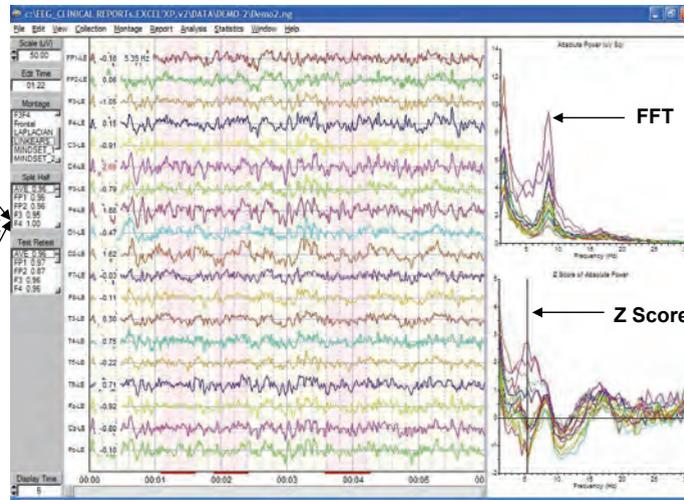
FIGURE 3. Treatment response differs after overtraining (OT). Usual training results in a rise-then-fall response. Overtraining results in a fall-then-rise response profile with improvement that may also fade.



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LETTER FROM ISNR PRESIDENT



Leslie Sherlin, PhD

This second letter as president means we are already a fourth of the way through the year. When I aspired to this position I had such huge dreams for making a lasting positive impact on the organization and already time is slipping away. With this in mind let me revisit my opening letter of our goals and give you a status update.

My vision for the organization and what I believed to be our major push was education about what it is that our organization and members do. My intention was that education should be focused in three groups and areas, our own profession, our colleagues in other professions and the public. We have significant efforts already moving forward in these areas. For details on the specific agenda reference the Winter 2010 issue of NeuroConnections.

First in order to educate our own membership and professionals in our field we of course continue to promote our conference. This year's conference Call for Papers and schedule outline is now posted on our website. We have an integrated focus this year to not only include neurofeedback and other neuromodulation techniques but are specifically asking for submissions that also discuss other biofeedback modalities and how they are utilized in conjunction with neurofeedback. We have already seen a strong interest in our group for submitting and attending talks that discuss these integrative neurofeedback and biofeedback techniques.

Another significant endeavor, undertaken by our Standards Committee this

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LETTER FROM AAPB NFB DIV PRESIDENT



David Kaiser, PhD

As a college student I worked in halfway houses and frequented state hospitals and group homes in Massachusetts, Connecticut, and Vermont as part of my job with the state's conservatorship program, assisting those with schizophrenia and other individuals who could not take care of themselves. I dealt daily with young adults suffering from schizophrenia as well as older adults, including one memorable institutionalized man who hid his mother in the freezer lest his social worker discovered her (natural) death, which of course they did. In all of these formative college experiences I discerned no clear pattern of what constitutes mental illness, except a difference in focus from most people – that, and a tendency to smoke. This difference in focus or awareness seemed very natural to these men and women, even though it separated them from others, but I couldn't help but think that they might fit in somewhere, given the right place and time.

When animals are removed from their natural environment, they often grow sick and die, and this is true for people as well. As a species we are adapted to the Pleistocene era, a world of mastodons, giant ground sloths, shamans, and tribal customs. The Pleistocene is where our kind had its most recent speciation, our latest branching. John Bowlby called this the 'Environment of Evolutionary Adaptedness' the world for which our genes were engineered. We are genetically mismatched to the challenges of modern life, though we

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LETTER FROM ISNR CO-EDITOR



Merlyn Hurd, PhD

DEAR MEMBERS,

The spring edition of NeuroConnections is focused on assessment and protocol selection. What a formidable task to try to contain the various methods into one edition.

The Brownback, Mason and Associates over many years have developed a very easy to use assessment that is computer based and has a follow up assessment every three weeks (you can define how often) for the duration of the treatments. Reading Tom's article is highly recommended to see how carefully they assess the brain's operation, the symptoms of the client and design the protocols to match those findings. Although some clients will say the assessment via computer is self monitoring and may be biased, I can speak from experience of using their methods that if the clients will stick with it they begin to see the improvements in themselves and the changes in their lives.

Looking at a completely different angle in neurofeedback Dr. Tom Mathews helps us look at the issue of overtraining. Most of the time the discussions around the table are regarding the under training of clients. Such as clients leaving before the most benefits could be obtained. So it is a thoughtful nudge to all of us that Dr. Mathews is making to remind us that overtraining can and does happen. Enjoy his article and maybe even re-look at some of your client's progress.

There is a very informative article culled from the small group meeting at ISNR regarding addiction. Addiction is

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.

often treated by every practitioner, without always identifying the issue as addiction. Often alcohol addiction is viewed as not an addiction and rather as “maybe I drink a little too much.” Many other addictions have the same caveat. I remember a client who after 9/11 shopped extensively, as she put it, “because the mayor said we needed to.” Guess what her addiction turned out to be? Enjoy the insights in the article.

The Othmers have given us the article “Clinical Decision Making and Protocol Selection in Neurofeedback” which is the fruit of their many years of practice and research. The use of decision trees and fitting the information from fine history and symptom taking is well worth your time to peruse.

At the same time Dr. Cory Hammond reminds us that pretreatment assessment is paramount in our field. He looks at the issues of assessment and what can be the outcome when the pre assessment is complete and informative.

Finally, the publisher has given us permission to reprint an article regarding Peak Performance, written by Beauchamp and Beauchamp. Whether you are working with clients who are striving for peak performance on the playing field or in the boardroom, this article will give you new insights into the methods that are available to assist clients to achieve their highest goals.

Have a wonderful spring and enjoy the day!

Merlyn Hurd, PhD, BCN Fellow 

LETTER FROM AAPB CO-EDITOR



Roger Riss, PhD

Welcome to the spring 2011 edition of *NeuroConnections*. Our theme for the issue is *clinical decision making and protocol selection in neurofeedback*. Given both the complexity of our field, and the early stage in its evolution, it is not surprising that a diversity of training approaches continues to thrive, each offering unique contributions and perspectives to the whole of the field. Despite this diversity, practitio-

and neuropsychologically-based clinical inferences. Sue and Siegfried Othmer offer a lively discussion of the neuroregulatory model which informs their approach to practice and has been formative to so many practitioners in the field. Tom Matthews rounds out the discussion with a timely and important contribution - a systematic clinical model to predict, prevent, and manage risk associated with inadvertent overtraining in clinically vulnerable patients.

As this issue reaches you, AAPB's 42nd annual meeting will be well underway. This year's program chair, Wes Sime, has assembled a stellar program around

**GIVEN BOTH THE COMPLEXITY OF OUR FIELD,
AND THE EARLY STAGE IN ITS EVOLUTION, IT IS
NOT SURPRISING THAT A DIVERSITY OF TRAINING
APPROACHES CONTINUES TO THRIVE**

ners within the field share a common commitment to *clinical decision making and protocol selection* which is guided by clinically sound methodology and anchored in scientifically based practice.

David Kaiser opens the discussion with a general decision-making model which transcends any single school of neurotherapy practice. Tom and Linda Brownback present a systematic overview of clinical decision making guided by both QEEG

the theme: *Paths to Resiliency*. No phrase better captures the core of our work in biofeedback, or better describes the spirit and resiliency of this year's host city, New Orleans. Please take a look at the Website for the AAPB Conference information in New Orleans. I look forward to seeing you there! http://www.resourcenter.net/images/aapb/files/2011/annmtg/2011Prelim_FINAL.pdf

Roger Riss, PhD 

LETTER FROM ISNR ED



Cynthia Kerson, PhD

This year, quickly heading into its second quarter, is crammed with projects. Leslie spoke of most of them in his letter. And, this issue is so packed that I was told to keep my letter short!

There are many individuals who would like to use neurofeedback training but simply can't afford it. Yet, these same patients are instructed to get MRIs, CAT scans, X-rays and all sorts of other tests that are often inadequate, misread, expensive and unnecessary. Many of these tests cost more than a neurofeedback treatment plan and/or QEEG, but because they're reimbursed, the cost is not real to the consumer and therefor not fully considered.

Thus, I want to bring your attention to the Parity Project. As you may remember, in 2008 the *Wellstone Domenici Mental Health Parity Act* was enacted. In it, health care insurers and third party payers are instructed to reimburse for mental health and addictions care using the same standards they use for medical care reimbursement. This is progress for mental health consumers in that they will no longer be limited to restrictive reimbursements.

The Parity Project Committee, which is Richard E. Davis, Randy Lyle, Anita Myers, Ed Pigott and me, is working on a packet that ISNR members can access that provides step-by-step instructions for the unfortunately lengthy denial procedures. We are looking for a core group of practitioners to help with creating the packet

by allowing us to walk through their denial progressions with them, ironing out kinks and establishing a foundation for the procedure packet. We figure it will take a few denial cycles to argue powerfully for parity with medical care reimbursement. And the more winning arguments we have, the better grounding we have for parity.

I think this is an important project that will benefit ISNR members and create a stronger consumer base for our field. If the decision to partake in neurofeedback training does not get weighted down by the personal cost it then becomes equal to the many currently inadequate standard treatments for several of the disorders that neurofeedback training is successful with.

Best,

Cynthia Kerson, PhD, BCN, BCB 

LETTER FROM AAPB ED

AAPB LOOKS TO ITS FUTURE



David L. Stumph

Future thinking is one of the most important jobs that a board and its executive team has in serving its constituents. In difficult economic times, it is especially important to understand the threats and challenges that are inherent as a result of the downturn and to set strategies that will guide the organization and its members to a more positive future.

Much has changed over the past few years. And much of that change has been driven by the downturn in the economy. Members typically hold on to their money longer before renewing their membership, registering for a conference, or making other important decisions that affect payment of funds. Members are also looking for more continuing education options that might not involve travel or less expensive, more localized educational opportunities. While online networking will never replace an in-person exchange of ideas and expertise, it is becoming more and more a part of the day to day human experience.

As AAPB looks to its future, all of these factors must be taken into account. In doing so, the executive committee took

MUCH HAS CHANGED OVER THE PAST FEW YEARS.

time to meet following the ISNR meeting last fall in the Denver area. The meeting was highly productive and involved a look at the challenges facing not only AAPB but its member practitioners, educators, and the entire biofeedback and neurofeedback community. The primary focus in this process had to do with finding strategies to develop that will provide value to AAPB members in a way that will also benefit the association so that we are all working together going forward.

In establishing the plan, the previous strategic plan was revisited and reviewed

to determine to what degree the prior initiatives had been achieved. We were very pleased in conducting this analysis to see that most had been accomplished, such items as the establishment of the online Clinician's Toolkit, a revamp of the website, expansion of continuing education options to include multiple disciplines, and expansion of the educational content to include more focus on new technology and more of a focus on the clinical practice of bio and neurofeedback.

Here is a brief summary of the strategic planning outcomes:

New Vision Statement:

Enhancing the innate ability to heal one-self

New Mission Statement:

AAPB is committed to providing information and support in the areas of mind-body science and biofeedback, serving healthcare professionals, the media and the public.

There were four themes defined, consistent with the themes from the previous plan but given new objectives and strategies. For purposes of this report, we will spare you the listing of all the strategies but provide the themes and overall objective as follows:

The four themes of the plan are:

- o Science
- o Education
- o Practice Support
- o Technology

The goals for each of the themes are:

- o **Science:**
 - Increase funding to support research grants
 - Enhance the Foundation's presence on the AAPB website in support of their fundraising efforts
 - Improve publicity for scientific outcomes
 - Improve the marketing of AAPB scientific publications
 - Work closely with vendors in support of research objectives
- o **Education:**
 - Create a speaker's bureau as a means of reaching out to outside professional groups and the public
 - Reach out to international health care and psychophysiology communities

- Continue to and enhance support for BCIA certification
- Explore non-traditional, low cost educational options such as webinars, regional programming, and the use of technology for the delivery of education
- Explore the possibility of creating protocols for treatment of disorders
- o **Practice Support:**
 - Improve and update the online Clinician's Toolkit
 - Explore ways to enhance patient and public awareness of the benefits of bio and neurofeedback
 - Improve member education related to AAPB's members benefits
 - Focus on student members and emerging practitioners, the future of AAPB
- o **Technology:**

- Continue to improve the AAPB website including navigation and content
- Improve the use of social networks as a means of member networking and online marketing and enhancing public awareness of bio and neurofeedback
- Continue to incorporate new technologies such as gaming into our educational content
- Explore ways to disseminate information about the availability and use of bio and neurofeedback devices.

While this list provides the primary focus of AAPB's plan, the numerous strategic initiatives that were also developed are designed to move the organization forward in a way that meets these themes and goals. As we look to AAPB's future, we will be reaching out to our members to volunteer for service on task forces and committees assigned to address these goals.

While the economy has had its effect, the future offers exciting times. New opportunities are on the horizon that can be capitalized on to the benefit of our members. The plan that we have developed is one that will lead us into the future with a focus on our members and their core values.

David L. Stumph, IOM, CAE 

ISNR PRESIDENT
CONTINUED FROM PAGE 4

year, is a paper on practice standards in our field. In the current issue of our flagship *The Journal of Neurotherapy*, enclosed in this mailing, is the ISNR Position Paper “Standards of Practice for Neurofeedback and Neurotherapy” authored by Hammond, D. C., Bodenhamer-Davis, G., Gluck, G., Stokes, D., Hunt Harper, S., Trudeau, D., MacDonald, M., Lunt, J., & Kirk, L.. This paper is an excellent step in our field rising to the task of providing basic provisions for the professional utilization of neurofeedback and neurotherapy techniques. We see our field growing rapidly with an influx of new providers offering services in which this position paper will establish a minimum standard. It will further our goal of educating our own professionals. Congratulations and thank you to these authors and this committee for this contribution. Additionally in the executive director’s letter of this issue you will see other efforts from the board and committee members on the topic of Parity for Reimbursement.

In our efforts to educate our colleagues we have representatives on the recently formed Collaborative Neurofeedback Project. The project is underway with a large contingency of members from across the ADHD spectrum of researchers and clinicians. This group meets regularly and is planning a efficacy/effectiveness study to be started this year. Here is the perfect illustration of how ISNR members who have sufficient expertise in neurofeedback methods are participating to help researchers in the ADHD clinical and research fields make the best methodological considerations to objectively and accurately measure and interpret outcomes.

On point of educating the public as well as providing a service to our members the PR committee and Web site subcommittee are continuing to develop content and strategize about implementation. The organization is now requesting and seeking proposals from Web design and implementation companies/individuals to take our Web presence to the next level. If you are currently satisfied with your Web designer or have recommendations please send those names to the ISNR office at office@isnr.org so that an official RFP can be sent. We expect this project to be a lengthy one but hope to have the task initiated this year.

Finally to address one of the themes of this issue, Cynthia posed a question “To Q or not to Q?” As expected there was some

discussion on various listservs on the topic. I have not seen any responses submitted to the editor but look forward to the full array of comments by our members. The question first called for further discussion on definitions. On one listserv I saw a more precise definition clarified that I’ll paraphrase and quote here, there are a very large number of providers who utilize 19 channel recordings and software for analysis of such, but even 2 and 4 channel evaluations are “also QEEG, i.e., non-QEEG is defined as “Eye Ball” visual examination of the EEG traces and no use of a computer to compute spectra or any analysis. Even [neurofeedback] is QEEG” (Robert Thatcher, personal communication, February 10, 2011). I know of a very limited number of circumstances that would prevent a provider from doing a most basic QEEG evaluation even if only examining one electrode site prior to conducting a session. However, even in these limited circumstances, once the neurofeedback session is being conducted, the premise of quantifying the EEG and determining parameters for feedback are performing QEEG and most providers are examining the spectral output during sessions to help guide thresholds, future intervention and learning response curves. So the question that actually remains and was asked by many people on various media – when do we not “Q”?

I wish you all a productive and prosperous spring. I hope you take the time to visit the ISNR website to read about the 2011 conference and submit to our open call for papers.

Best regards.

Leslie Sherlin, PhD 

AAPB NFB DIV PRESIDENT
CONTINUED FROM PAGE 4

do the best we can with what we have, and this was clearly so for those people I assisted. The lifetime prevalence of mental illness in the industrial world is 1 in 2 —half of us will suffer a major mental disorder before we leave here. In other words, our world no longer fits us. We are tooled by nature to live in an earlier time and a simpler place; but here we are, moving faster than we ever imagined across the landscape and contacting friends and family who are thousands of miles away. We are not adapted for Chicago, or upstate New York, or any town or city. We belong in a tribe, oyster fishing off the coast of South Africa a hundred thousand years ago, when and where our brain reached the pinnacle of fitness, our last retooling of genes. AAPB and the Neurofeedback Division, and groups like them, are modern-day versions of tribes, allowing us to spend time with fellow tribesmen and women once a year or so, and my genes are grateful for this ongoing experience.

This is the last letter of my presidency of the NF Division. During my brief tenure we created a NF Division listserv and selected Jay Gunkelman and Rex Cannon as speakers for the two AAPB NF Division dinners. Jay did not disappoint, and Rex is coming up to bat in New Orleans this March, which I am looking forward to. I am succeeded as President by physicist-turned-entrepreneur Siegfried Othmer PhD, a pioneer of this field.

David Kaiser, PhD 



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DECISION MAKING AND PROTOCOL SELECTION FROM THE PERSPECTIVE OF THE BROWNBACK MASON AND ASSOCIATES NEUROFEEDBACK SYSTEM (BMANS)—PART 1 OF 2



Thomas S. Brownback, MEd

I would like to begin by thanking Roger Riss for inviting us to present two companion features on the topic of clinical decision making and protocol selection in this NeuroConnections issue and also for his kind words about The Brownback Mason and Associates Neurofeedback System (BMANS) stating that “an issue on this topic really would not be complete without attention to the important work you folks have contributed in this area.”

The BMANS System, in its broadest sense, is comprised of four major therapies and their systematic integration. One: psychodynamic psychotherapy for the individual (which is typically done within the neurotherapy session and is often done in response to the insights which neurotherapy training brings to the client’s awareness), marriage therapy, family therapy and/or parenting: if the individual, marriage or family system is dysfunctional, this will often make neurotherapeutic training success impossible, or at least will significantly impede it.

Two: cognitive behavioral psychotherapy: it is our position that healthy brain chemistry plays a powerful role in neurotherapy success. Many years ago, in order to impact the biochemistry of the brain, we created the BMANS Bio-Psycho-Socio-Theological Cognitive Behavioral Framework or *The Basics* for short

Three: heart rate variability (HRV) training: we believe that neurotherapy is one of the most powerful tools for healthy specific reregulation of the brain, and that HRV training is one of the most powerful tools for healthy systemic reregulation of the entire body, which in turn supports brain function. The most frequent guidance that I give to clients during neurotherapy training is to focus on slow, deep diaphragmatic breathing. This type of breathing is the foundation for success in HRV training and significantly supports success in neurotherapy training.

Four: neurotherapy training: the BMANS System, in a more narrow sense, is comprised of eight manuals or steps which fit under the heading of neurotherapy training. These eight steps have been fleshed out in the BMANS Manual Series, which is the foundation of our neurotherapy training and our four-day BMANS workshops. Clinical decision-making and protocol selection is the beginning of this foundation and is covered in the content of the first four manuals.

Over the last thirty years we have been on a journey to achieve the highest clinical success rate possible. Our goal has been to analyze the information which we continue to gain from both our successes and failures, extract the knowledge from this information in order to do neurotherapy at a higher level and continue to use this knowledge to build an ever more powerful neurotherapy system. Like most clinicians, our journey began with listening to what our clients said were their presenting problems and then we utilized recognized training protocols to treat those neuro-pathologies. While that approach certainly worked with a number of clients, we did encounter two significant barriers to achieving

The BMANS Cognitive Behavioral Framework (The Basics)

	Units of Measurement	Goals								avg	score
BIOLOGICAL											
A. NUTRITION											
1.	Fruit (days/week servings/day)	7-2 Std. 7-3 Bns.									
2.	Vegetables (days/week servings/day)	7-4 Std. 7-3 Bns.									
3.	Whole Grains (days/week servings/day)	7-3 Std. 7-4 Bns.									
4.	Protein (days/week servings/day)	7-1.5 Std.									
5.	Dairy (days/week servings/day)	7-1.5 Std.									
6.	Multivitamin (days/week servings/day)	7-1 Std.									
B. EXERCISE											
1.	Aerobic (days/week minutes/day)	2-20 Std. 2-30 Bns.									
2.	Anaerobic (days/week minutes/day)	2-20 Std. 2-30 Bns.									
C. SLEEP											
D. RELAXATION											
1.	Breathing/Cueing (days/week minutes/day)	4-10 Std. 4-15 Bns.									
2.	Body Scans (days/week times/day)	7-5									
3.	Biofeedback (days/week minutes/day)	4-10 Std. 4-15 Bns.									
PSYCHOLOGICAL											
A. Journaling											
B. Read/Listen for Growth											
C. Self-Affirmations											
D. Addictions											
1.	(days/week number/day)	7-0 Std.									
2.	(days/week number/day)	7-0 Std.									
3.	(days/week number/day)	7-0 Std.									
SOCIAL											
A. Conflict Resolution											
1.	Resolutions needed (number of times/day)										
2.	Resolut. attempted (number of times/day)	all									
B. Sharing											
C. Fun											
D. Affirming others											
SPIRITUAL											
A. Worship/Corporate											
B. Bible Time											
C. Prayer Time											
D. Christian Music											
										TOTAL	

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Figure 1: The Basics is made up of 16 healthy behaviors which clients track on a daily basis. Once clients have learned this process of tracking and recording their health behaviors, it only requires about 2 to 3 minutes at the beginning of their neurotherapy session to report their scores for that week.

our goal of the highest level of success: one: what a client reported as the presenting problem was not always the primary neuropathology that needed to be treated for the client to experience success. John Jones came to our office and said that his son, Johnny, had Attention Deficit Disorder (ADD). We used a standard ADD protocol for training. (The term, “standard protocol” generally applies to the idea of training at a specific placement, using a totally or partially fixed frequency band, to always train up or always train down, using a particular metric such as microvoltage or coherence, always using the eyes open or always using the eyes closed training condition in order to ameliorate a particular neuropathology. For ex-

ample a typical protocol that has been used for anxiety reduction is to train at PZ, using an (8 to 12) Hz bandwidth, to increase the microvoltage with eyes closed.) After three or four training sessions it began to emerge that Johnny was also depressed and anxious, and that these problems were both larger issues of concern in Johnny's life than the ADD. So not only were these two additional neuropathologies causing Johnny more emotional distress than the ADD, they were exacerbating his difficulty with focusing. From this experience and many others like it, we realized that we needed a method for having all clients and their significant others evaluate a broad range of neuropathologies at the beginning of the neurotherapy process. This led to the creation of our comprehensive neurodiagnostic checklist, which will be discussed more fully later.

The second significant barrier to achieving our goal was the experience that we had with Alice Adams. With Alice, we had a correct match between what she said was the problem (ADD) and the actual neu-

ropathology. However, the protocol that was currently the standard in the field did not work for her. We used the standard protocol of training, which was the microvoltage of theta down and beta up with eyes open at Fz, but Alice not only did not get better, she got worse. We were left questioning: was it the placement? the frequency band the direction of training (up or down) etc.? After years of struggling and searching we realized that many of our questions could be answered and that our failures with Alice and other clients could have been prevented by using a QEEG.

However, as I began my quest to understand the role that the QEEG could play in successful neurofeedback training, I came to realize that to obtain the highest level of training success, it was crucially important to have a full grasp of three primary areas of knowledge: one, the neuroanatomy and neurophysiology (or the jobs and functions of the brain) at each of the International 10 20 System placements, or constellation of placements; two, how information moves through the brain from placement to place-

ment; three, the definitions of the characteristics which are associated with each brain wave frequency (theta, alpha, etc.). This information became the content of our Manual One or step one of the BMANS System.

Building on this knowledge of neurophysiology or healthy brain function, at each International 10 20 System placement or constellation of placements, we have the foundation for the next level of the BMANS System, which is neuropathology. For clinical decision-making and protocol selection, it is critically important to understand the underlying rationale for this link between healthy neurophysiology and neuropathology; i.e., between healthy brain function and brain dysfunction. To support healthy brain function at any given 10 20 system placement, that placement must have a normal amount of microvoltage at each frequency and a normal amount of connection to all other placements--determined by a normal amount of coherence and phase

Continued on page 10

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at each frequency. When the microvoltages at any frequency and/or the connections at any frequency for each 10 20 system placement are not normal, this often leads to neuropathology.

For example, to understand which 10 20 system placements are most likely implicated in ADD, Inattentive Type, one must understand which areas in the brain and their correlate 10 20 placements are associated with the ability to attend or focus. The neurophysiology of the prefrontal cortex (Fp1, Fp2 and surrounding placements F7, Fz, F8) has as one of its functions the ability to sustain focused attention. Therefore, when one of the presenting problems

of a client is the inability to attend, our model would direct the clinician to look at the prefrontal cortex and the surrounding area for abnormal QEEG metrics.

Another example is to understand which placements are most likely implicated in a reading problem. First, one must understand which 10 20 system placements are associated with the areas of the brain that are implicated in the ability to process visual receptive language. In this case, it would be the left hemisphere (Fp1, F3, F7, C3, T3, P3, T5, and O1) that has as one of its functions the ability to work with language. The posterior regions of the brain (the parietal, temporal and occipital lobes) have as one of their functions data processing from the external world. For example, the

occipital lobe (O1, O2) processes visual information. Therefore, if the client is struggling with reading, the BMANS model would direct the clinician to look at the left posterior cortex (especially focusing on O1, O2, P3, T5) for abnormal QEEG metrics. This perspective, which concerns how to determine the best scalp placement(s) for each neuropathology from an abnormal microvoltage, peak frequency, asymmetry and coherence metric point of view, is the content of the BMANS Manual Two or step two of our system. (At this point in time there are 42 neuropathologies in BMANS Manual Two.

Remember Johnny? Well, we went on to discover that to achieve the highest level of training success we needed to pair

Figures 2, 3, 4 and 5 are examples of the CNC-1020 heads with the percentage of probability for D.R., a 33 year-old male with the presenting problems of ADD, impulse control problems and hyperactivity abnormalities.

Figures 6, 7, 8, and 9 are examples of the psychoeducational testing heads with percentiles of probability for D.R.

Figure 2:

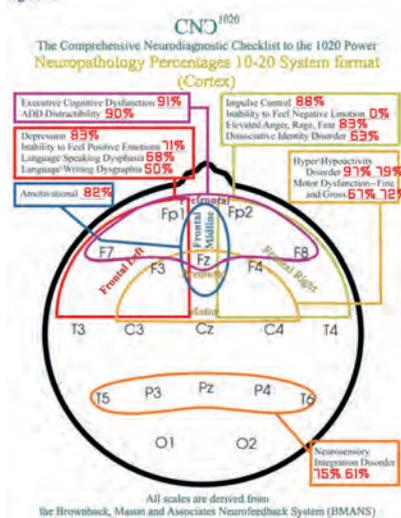


Figure 3:

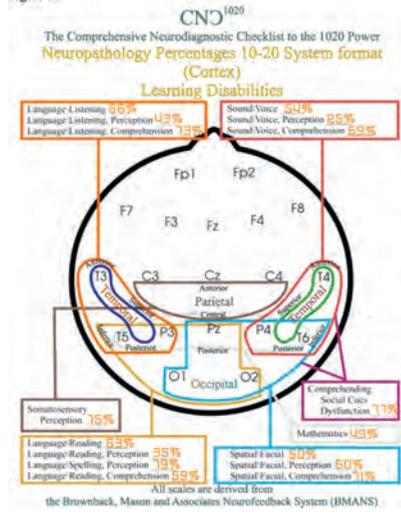


Figure 6:

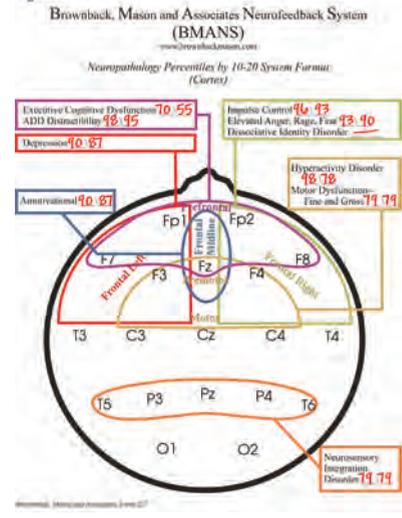


Figure 5:

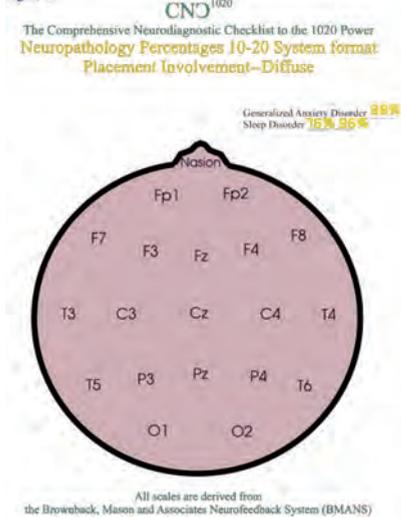


Figure 4:

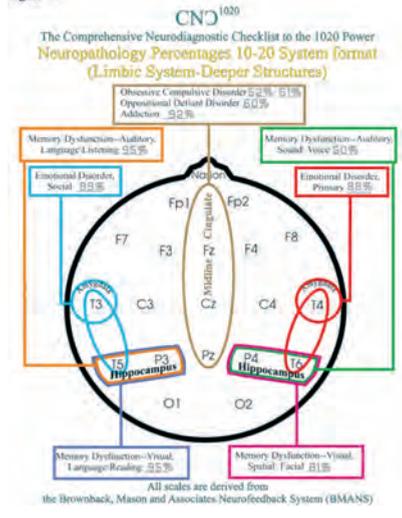
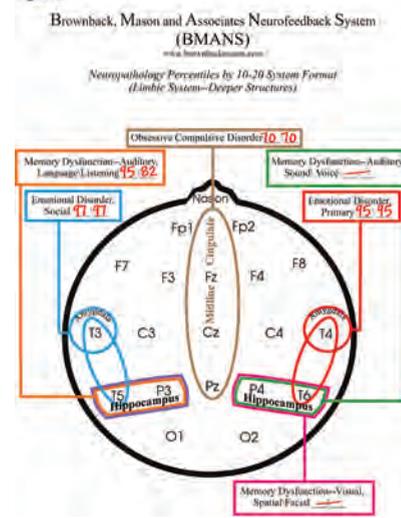


Figure 8:



the QEEG with a comprehensive reporting of symptomology that systematically evaluates other reasons for a presenting problem, evaluates confounding influences on the problem and gives information that guides in the interpretation of the QEEG. Over time we developed The Comprehensive Neurodiagnostic Checklist (CNC-1020), which utilizes 300 items drawn primarily from The Diagnostic and Statistical Manual (AMA 1994) to flesh out the 42 neuropathologies from the BMANS step 2. These 300 items have been transformed into a very specific format that allows us to determine, from the subjective point of view of our client (if he or she is old enough) and up to three other raters, to what degree our client has any of the aforementioned 42 neuropathologies.

The CNC-1020 not only provides a means of understanding to what degree our client has any of these neuropathologies, but it also directs the neurotherapist to the most likely International 10 20 System placement or constellation of placements for training, as the results are displayed across four International 10 20 System heads. The first head looks primarily at frontal cortical neuropathologies; the second looks primarily at posterior cortical neuropathologies; the third, at deeper structures, including the cingulate, hippocampus and amygdala from the limbic system; and the fourth is used to consider frequency abnormalities more than particular placement abnormalities.

In our pursuit of the highest success rate, what we encountered was that approximately 20% of our clients had a tendency to under-report neuropathology on the CNC-1020. We have utilized two methods for dealing with this problem. One, we use the Marlowe Crown Social Desirability Scale. (Many of the items on the Marlowe Crown are shared with the Lie scale on the Minnesota Multiphasic Personality Inventory--MMPI.) This test helps us know the likelihood of under-reporting the severity of symptomology on the CNC-1020. Two, we have incorporated a number of psychoeducational tests to provide a more objective method of rating approximately 30 of our 42 neuropathologies. The results of this battery of tests are displayed across the same four 10 20 System heads which are used for CNC-1020.

In Step four of the BMANS System the results of the CNC-1020 and the psychoeducational testing, as displayed on the 10 20 System heads, serve as an overlay for interpreting the QEEG. We choose the six highest-rated neuropathologies on the CNC-1020 that are confirmed by elevations of those same neuropathologies on the psychoeducational testing heads. Focusing on the 10 20 System placements associated with these six neuropathologies, we then analyze the QEEG (we use several referenced databases, as well as non-database displays) looking for abnormalities in the microvoltage, peak frequency, asymmetries and coherence metrics at those same placements.

Our analysis has two fundamental goals. The first is to compare the placement(s) that are associated with each of the six highest rated neuropathologies that the subjective (CNC-1020) and the objective (psychoeducational testing) agree on, to the same placements on the QEEG for abnormal z-score correspondence. It still fascinates me, after all these years, that 95%

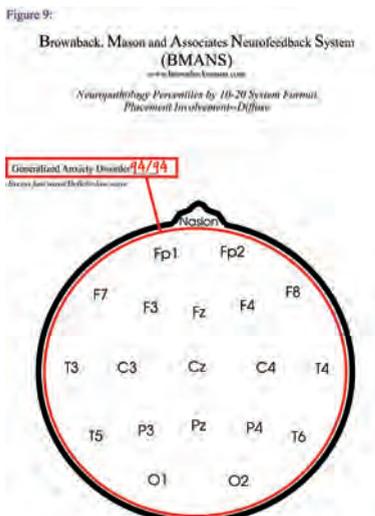
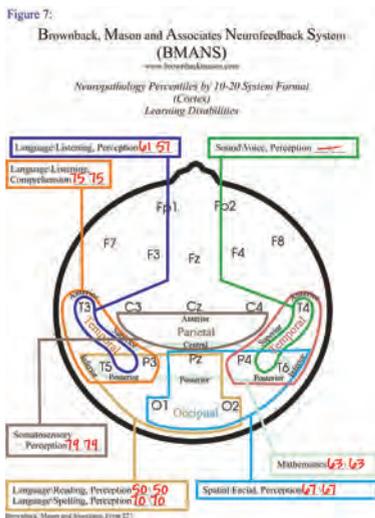
of the time there is correspondence amongst the CNC 1020 heads, psychoeducational testing heads and the QEEG topographs. For example, compare D.R.'s scores on the first CNC-1020 head (Figure 2) and the first psychoeducational testing head (Figure 6) to his linked ears, eyes-open brain map (Figures 10 & 11) for his presenting problems of ADD, impulse control problems, and hyperactivity abnormalities.

The second goal is to use the QEEG results to create totally customized training protocols for each client. Each of the top six neuropathologies is systematically evaluated. In order to obtain the highest level of training success, the first thing we do is to determine which placement(s) associated with a particular neuropathology has the most deviant z-score values. For example, we most often find the most deviant z-scores at Fp2 and Fp1 on the QEEG of a person whose CNC-1020 and psychoeducational testing indicate the presence of ADD, inattentive type. However, we have also seen the most deviant z-score placement(s) at F8, Fz or F7, and at other times, we have even found the most deviant findings at T6 or P4, which Bob Gurnee refers to as the "posterior attentional circuit."

This search for the most deviant z-scores includes considering both the eyes open and the eyes closed conditions at the placements associated with attentional problems. Often we find similar abnormal z-scores for both. However, with a number of clients, we have observed the abnormal z-score readings in one condition and not the other. Therefore, to get the best training results, we need to know in what condition the most deviant z-scores occur.

The next consideration is frequency: of great surprise to me was to find in the literature, for example Chabot, et al (1996) work on ADD, that the abnormal frequencies could be anywhere along the frequency spectrum. He and others have referred to the theta (4-8) subtype, the alpha (8-12) subtype and even the beta (12-25) subtype. For the most powerful training effect, I have come to recognize that it is not truly about specific bandwidths based on subtypes. The bandwidth determined by abnormal z-scores of 1.5 standard deviations or more can be across any frequency band, for example, 6-11, 10-15, or even very broad bands, such as 3-13 or 10-30. Therefore, we create training bands for individual protocols to be precisely whatever bandwidth involves greater than +/- 1.5 SD z-scores at that placement.

Continued on page 13



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To my even greater surprise concerning frequency, I have come to understand that it is not always about training microvoltages down, but often the abnormal z-scores, even of slow waves, need to be trained up. (To my embarrassment, it wasn't until approximately five years ago that I stopped ignoring my own battle cry of "Let the Q guide the training!" and began training to increase delta when delta had abnormally low z-scores.)

I spent much of my earlier years only training microvoltages up or down. However, analysis of the QEEG may reveal that the location of the neuropathology may also have, or only have, deviant z-score(s) in one or more of the coherence bands. In those situations creating coherence protocols will replace those designed for magnitude training.

**IN OUR PURSUIT OF THE
HIGHEST SUCCESS RATE, WHAT
WE ENCOUNTERED WAS THAT
APPROXIMATELY 20% OF OUR CLIENTS
HAD A TENDENCY TO UNDER-REPORT
NEUROPATHOLOGY ON THE
CNC-1020.**

Up to this point all of our discussion concerning training to normalcy has utilized the z-scores generated by the Fast Fourier Transform (FFT) from the brain map. However, with the advent of Live Z-Score Training (LZT) (BrainMaster, Ohio, USA) the z-scores generated by the Joint Time Frequency Analysis (JTFA) (Collura, 2009) provide us with an additional z-score metric. Manual Five of the BMANS System utilizes three Z-Score designations: one: Z-Score Not Applicable (ZSNA) for clinicians who do not have Live Z-Scores Training. Two: Z-Score Passive Training (ZSPT) is for protocols in which the audio and visual feedback is driven by training the microvoltage or coherence to increase or decrease with the Live Z-scores running in the "background. Three: Z-Score Active Training (ZSAT) is for protocols in which the Live Z-Scores provide the audio and visual feedback. If the QEEG shows significant deviant z-scores for magnitude or coherence at a placement(s) related to the primary presenting problem, then the client's initial training protocol will be magnitude or coherence training with ZSPT. I have found that most clients quickly relate to this clear and direct connection to their presenting problem, thereby increasing the likelihood to experience initial success. Once they have an understanding of the interplay between the audio and visual feedback and their subjective experience, I will move to ZSAT protocols, especially in situations in which the neuropathology is revealed on the QEEG to involve 4 placements with deviant z-scores. (There is much to say about Live Z-Score Training, but this is beyond the scope of our present topic.)

The BMANS System for neurotherapy training is comprised of two major parts. Part one, as stated earlier, includes steps one to four and focuses on clinical decision-making and protocol selection. Part two, includes steps five to eight and focuses on training and tracking the progress of training. The intersection of these two major parts takes place in our form 254

Each PSP file or training file contains 10 neurotherapy training components. To organize the files, the client's three initials

Figure 10:

LINKEARS
Eyes Open

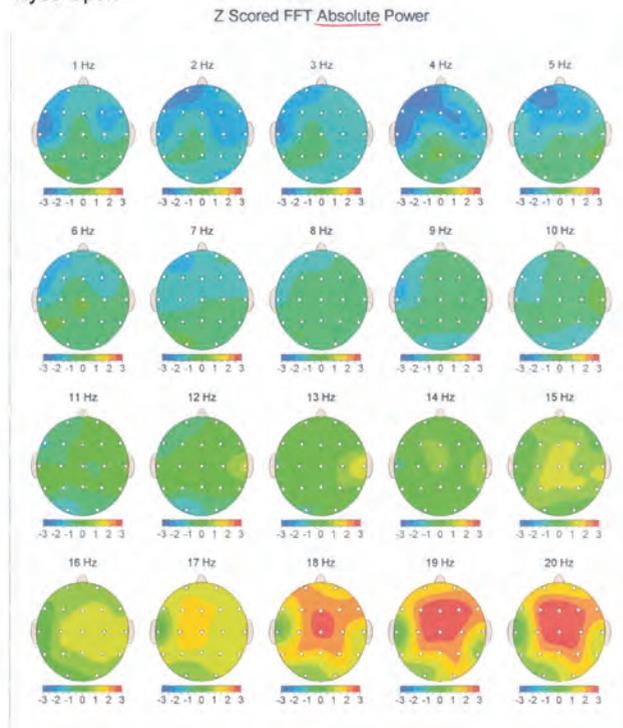
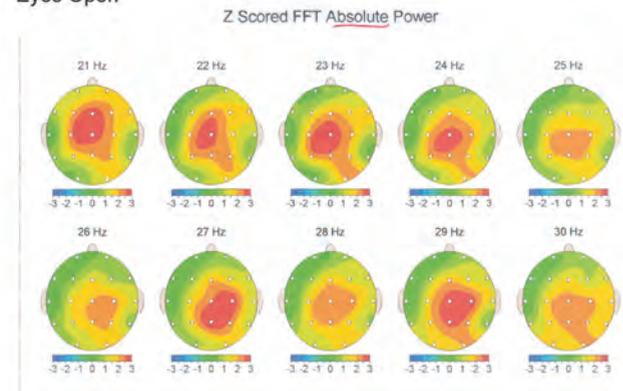


Figure 11:

LINKEARS
Eyes Open



Figures 10 and 11: The abnormal z-scores in the prefrontal at 18 to 21 Hz. correspond to the placements which we would expect for ADD; the abnormal z-scores in the right frontal at 18 to 23 Hz. for FP2 and 18 to 30 Hz. for F4 and F8 correspond to the placements for impulse control problems; the abnormal z-scores in the premotor and motor strip at 18 to 30 Hz. correspond to the placements for hyperactivity abnormalities.

(component one), the client's age (component two) and whether or not Z-Score training will be used and whether that Z-Score training will be passive or active (component 10) are used. The remaining seven components come from the analysis that was accomplished in step four. The CNC-1020 and the psychoeducational testing determine which neuropathology will be trained (component three), and they also guide the decision-making for the analysis of the QEEG as to which placement(s) will be trained (component five).

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CLINICAL DECISION MAKING AND PROTOCOL SELECTION IN NEUROFEEDBACK

Siegfried Othmer, PhD and Susan F. Othmer, BA



Bringing neurofeedback into a mental health practice means acquiring a working model by means of which all the clinical phenomenology can be reframed in a psychophysiological perspective. Clinical decision-making then emerges largely out of that framework. As the training proceeds, clinical observations are interpreted in terms of that framework and lead to fine-tuning of the clinical strategy. There are two feedback loops here. One involves the client and the feedback signal. The other involves the client and the clinician. The importance of the latter has increased over time as the techniques have strengthened in their impact. Within-session changes in physiological state have to be attended to promptly to steer the training in a propitious direction. This responsiveness, which is observed fairly typically, means that the burden of clinical decision-making has shifted from being a rather freighted decision at the outset to a continual, iterative process that is itself feedback-guided toward the desired objectives. This places the principal burden of competent guidance upon the clinician--more so perhaps than with any other approach.

One proceeds from the basic orientation that the brain must satisfy all of the criteria of a feedback control system. We think in terms of hierarchies here. Firstly, the CNS must assure its own unconditional stability. Allied with this concern is the ability to contain behavioral disinhibition. Second, it must manage set-points of activation of different functional domains. Thirdly, it must arrange for the smooth integration of these functional domains to meet the challenges of life. Clinical targeting then follows this same hierarchy. Promoting brain stability is the first objective. Training for better management of states of activation is the second. Functional integration to manage localized deficits is the third.

Our regulatory networks are also seen as hierarchically organized, with the brainstem as the head of the hierarchy in terms of the organization of timing in the orchestration of cerebral processes. Hence the first objective in terms of state regula-

tion is the domain of arousal regulation and of the sleep/wake cycle. We no longer try to characterize people in terms of high and low-arousal tendencies. That is no longer relevant to clinical decision-making. But understanding how people function in the arousal domain yields predictors about how they will react to the training. If these expectations are not met, then one either adjusts one's approach or one's understanding of the client.

Arousal regulation is primary in our considerations for a number of reasons, but a key factor is that it is a good observable for us. We can see readily how people react in the arousal domain. This is key because if we are able to move the person to a well-regulated state in the moment, then we can be sure that this will also yield the desired outcome over the longer term. The two are highly correlated. It even holds true that when we optimize reinforcement parameters for arousal regulation we are at the same time optimizing conditions for other regulatory functions that are not so readily observable. Further, if we have any other cues regarding the person's self-regulatory status, these also serve as indices for guiding the training appropriately. We take advantage of a kind of unitary quality to self-regulatory competence with respect to core regulatory functions.

The second priority is affect regulation, because of the intimate relationship between the limbic system and the brainstem. This also ties us in preferentially to right hemisphere function, so already at this point laterality issues are paramount. Failures associated with the right hemisphere are potentially the most catastrophic for people, and hence demand our early attentions. The right hemisphere is also the earliest to develop, so our training hierarchy aligns with that of early childhood developmental stages.

Addressing left hemisphere function is only the third priority, and that also brings in the left pre-frontal region. This ordering of priorities is somewhat ironic, given that most of us got started in this field with a focus on ADHD. Further, responsi-

bility for a lot of self-regulatory deficits has been assigned to the frontal lobe. But matters are not as they seem. It has been clear to us for a good many years that emotional dysregulation lies at the core of much that is labeled ADHD. The high overlap of ADHD with oppositionality and Conduct Disorder attests to this. It is the emotions that govern our attentions, and hence deserve priority in our hierarchy.

As the above is beginning to make clear, we organize our thinking in terms of principal regulatory axes: the top-down axis (with the brainstem at the top!), the left-right division, and the front-back axis. Each of these constrains the framework in specific ways. Within these divisions, we place the priority on training bottom-up control versus top-down, and we attend to the trophotropic division before we attend to the ergotropic. This is in line with the new findings regarding our resting state networks, namely that the organization of our resting states is a good predictor of our functional competences.

This is also in line with our biofeedback heritage, in which it was recognized early on that the quality of autonomic nervous system regulation was the key to good function more generally. In the early days of neurofeedback it was certainly the view that EEG training would simply complement peripheral biofeedback where the latter did not offer good access---executive function, specific cognitive function, working memory, visual and auditory processing, etc. Now it turns out that EEG feedback may be an elegant and highly efficient means of achieving autonomic nervous system regulation as well, in which case it needs to be placed early in the hierarchy of clinical concerns.

Historically, our migration from the standard SMR-beta training that we taught for many years to what is now predominantly training in the infra-low region of EEG frequencies took over ten years, over which time our clinical competences were enhanced particularly with regard to emotional regulation, autonomic regulation, and interoception. Along the way, clinical prior-

ities needed to be re-ordered. Our approach has by now diverged sufficiently from prior practice, as well as from common practice within the field, that it is called the Othmer Method. The distinctive features are that it utilizes bipolar montages in the principal constituents of the training program, and that reward frequencies are individualized. A total of five bipolar montages constitute the complete set from which a starting protocol is constructed.

The set of five protocols allow us to challenge the regulatory role of each quadrant of cortical real estate, with the fifth protocol devoted to inter-hemispheric training to promote cerebral stability. Our anchor sites are T3 and T4. These sites monitor the multi-modal association area of the anterior temporal lobe, and interact with the limbic system and nearby insula. The T3-T4 placement is used standardly in application to instabilities across the board, including vertigo, panic, asthma, migraine, seizures, and bipolar excursions. The other standard placements are T4-P4, used for physical calming; T4-Fp2, used for calming emotional reactivity and rage; T3-Fp1, used for mental calming and improved executive control; and finally T3-P3 for awareness of detail and related sensory processing issues. The latter category rarely rises to the level of a priority, so as a practical matter five starting protocols reduce to four.

In practice, the clinical hierarchy is implemented as follows. The primary target, as already stated above, is cerebral stability. Given the high prevalence of instabilities in our clinical population, T3-T4 is the dominant starting protocol. This general rule may be trumped in the case of developmental delay, early trauma, or other severe disorder traceable to early childhood development. In these cases the high arousal to which these nervous systems are driven becomes the paramount consideration. It can itself be a contributor to cerebral instability, so even in the event that instability is the primary concern (as in autism with seizure risk), the best approach may nevertheless be to calm the right hemisphere as a first priority. The starting protocol in these cases is T4-P4. If neither instabilities nor developmental issues are prominent, then matters are treated as more ordinary disorders of dysregulation with a starting protocol that combines right parietal with left frontal training. In such cases T3-T4 remains the starting protocol as the best means for finding the optimum training parameters. The details are given in The Protocol Guide (Ref).

In terms of reinforcement frequency

we have found our way over the years to the infra-low regions of EEG frequencies (i.e., < 0.1 Hz). Here we are simply tracking the slow cortical potential (SCP) in its temporal migration. This cannot be represented well with a narrow spectral filter. But at such low frequencies one cannot have both a narrow-band filter and real-time information. So we are actually seeing this signal within a broad signal bandwidth, and it does not appear at all sinusoidal. On the other hand, we have found clients to be highly sensitive to the particulars of the passband, and in this respect the training does resemble traditional frequency-based training. The adjustment of the band edge is the means by which the clinician optimizes the response for each individual during each session.

Unfortunately this technical approach violates all of the rules we have come to associate with traditional neurofeedback. First of all there is no event here that could be rewarded in the traditional mode. There is not even a goal that could be defined, so there is no threshold. There is no better and there is no worse. We are looking at a differential signal with broadly cyclical properties, after all, and a rising signal cannot be considered more virtuous than a falling one. (If we had exchanged electrode placements we would have the reverse polarity.)

The process is therefore one in which the brain is challenged toward a better self-regulatory status without our being able to steer or direct the process in any way at the level of the EEG. We simply provide the brain with information on its own slow cortical potential, and it reacts to that information. No instructions need to be given. We have to conclude that the challenge is built into the process. The brain recognizes that it is the author of the signal on the screen, and it naturally assumes responsibility for that signal. An error function inevitably prevails between the signal on the screen and the interpretation the brain gives to that signal in terms of its own ongoing activity. The minimization of that error function ongoingly constitutes the relevant challenge to the brain.

Now this view of physiological feedback may be strange to us in the neurofeedback community, but in fact peripheral biofeedback has been living with this reality since the beginning. We would describe Heart Rate Variability training in the same way, with the additional factor that in the latter case the instruction may be given, for what it is worth, to increase the excursions in heart rate. A similar bias is implicit in

many of our feedback games, in that larger signals are still favored as a carryover from the earlier days of SMR-beta training. Subject to that bias, our clients would be motivated to enhance the signal amplitude, which translates into greater excursions of the underlying variable, the SCP. So it could be said that we are promoting increased dynamics in the SCP. But there are other feedback modes which harbor no such bias, and we have clients who cannot follow instructions. We must conclude that the challenge is imbedded in the process itself, and is not imposed by the particulars of our mechanization.

We have developed a training method that combines some of the characteristics of traditional frequency-based training with features of traditional SCP-type training and of conventional biofeedback. The combination appears to be an improvement on each of its antecedents, for purposes of state regulation, with respect to breadth of clinical footprint, accessibility by various target populations, scope for skillful guidance by the clinician, rapidity of clinical response, and engagement of the client in the process.

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Siegfried and Susan F. Othmer were drawn to neurofeedback in 1985 to help with the epilepsy of their son Brian. With professional backgrounds in physics and in EEG research, Othmers designed the first Neuro-Cybernetics system in 1985 and the second, known as the EEGer, in 2000. In 2006 they designed the Cygnet system to extend neurofeedback to the infra-low frequency region. Over the last twenty years they have taught more than 5,000 neurofeedback professionals in some nine countries. Othmers founded EEG Spectrum in 1988 as a practitioner network for clinical service delivery. They have published on neurofeedback for ADHD, mental retardation, addictions treatment, chronic pain, and PTSD. Currently Siegfried Othmer serves as Chief Scientist at the EEG Institute in Los Angeles, where Susan Othmer is Clinical Director. Together with their son Kurt they created EEGInfo as a service organization for neurofeedback professionals in 2002. Siegfried Othmer is President of the Brian Othmer Foundation, under whose auspices neurofeedback services are being delivered at no charge to veterans for PTSD through an ad hoc nationwide practitioner network. Currently, Siegfried Othmer serves as President-elect of the Neurofeedback Division of



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QEEG: LIABILITY AND CONSUMER PROTECTION

D. Corydon Hammond, PhD, ECNIS, QEEGD

ABSTRACT

While the use of QEEG assessment is not always required to produce clinical improvement, it nonetheless facilitates the individualization of treatment. There is documented and increasing evidence that inappropriately conducted neurofeedback may produce iatrogenic effects. Therefore, pre-treatment evaluation utilizing a QEEG which is analyzed by a clinician who is certified in EEG and QEEG appears to provide protection not only for patients, but also liability protection for practitioners through demonstrating that they are basing treatment on a scientifically objective and thorough assessment rather than guesswork.

It has been pointed out previously (Hammond, Walker, Hoffman, Lubar, Trudeau, Gurnee, & Horvat, 2004) that there are numerous published papers illustrating that positive clinical outcomes can occur from neurofeedback treatment where the pre-treatment assessment has consisted of monopolar or sequential data that has been gathered at a limited number of EEG sites, without more thorough QEEG assessment.

On the other hand, there is also strong mounting evidence (Hammond & Kirk, 2008; Hammond, Stockdale, Hoffman, Ayers, & Nash, 2001; Lubar, Shabsin, Natelson, Holder, Whitsett, Pamplin, et al., 1981; Lubar and Shouse, 1976, 1977; Whitsett, Lubar, Holder, Pamplin, and Shabsin, 1982) that when not properly done, neurofeedback can result in adverse reactions. As Hammond and Kirk (2008) indicated: "Adverse effects that have been reported by clinicians include: increased anxiety and agitation, panic attacks, manic-like behavior, headaches, nausea, fatigue, sleep disturbance, anger and irritability, crying and emotional lability, incontinence, enuresis, an increase in depression, decline in cognitive functioning (decreased concentration, mental fog-giness), increase in obsessional rumination and OCD symptoms, increase in somatic symptoms (including tics and twitches), vocal tics, seizures, slurred speech, loss of previous symptomatic improvements, and temporary disorientation or dissociation that could put someone at risk for an accident or injury."

Since the publication of that paper, the author has been contacted by a considerable number of consumers of neurofeedback services who have reported further experiences of iatrogenic effects, some of which have endured for a year and a half or more following neurofeedback treatment. In this regard, the author is aware of at least two lawsuits that are in process. The largest proportion of the iatrogenic effects that have been identified have involved treatment that was not preceded by a QEEG, and which often focused on significantly reinforcing various frequencies, often in search of a magical "sweet spot."

Thus it is this author's conclusion that more thorough pre-treat-



ment assessments seem to provide protection for clients through facilitating individualization of treatment (Hammond, 2010). Additionally, more thorough pre-treatment assessment, which includes a QEEG, will undoubtedly prove valuable in liability protection for clinicians, allowing documentation that treatment was being guided by scientifically objective evidence and not simply being done by seat-of-the-pants guesswork. When QEEG assessments are conducted, however, it is imperative that the person analyzing the data be someone with documented qualifications for doing so, for as the old statistics class saying goes, "garbage in, garbage out." If practitioners have to defend themselves in malpractice actions, simply trying to say that they had gone through a two day workshop on QEEG and were relying on far from perfect automatic artifacting features would undoubtedly prove inadequate.

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Dear NeuroConnections readers,

You may notice that in this issue of the newsletter, Thought Technology is not advertising in our regularly-held centerfold position. We are disheartened to think that this may reflect on our investment in the ISNR and AAPB communities or our commitment to serving and supporting the field of which we have been a part for over 35 years. The choice of advertising location was not our own, but rather it was due to a decision to grant the slot to another advertiser. In exchange for this, the *NeuroConnections* editors have graciously offered us this brief note to you the readers as well as an extended advertising layout and an article in each of the 2011 issues of the newsletter. You will find our current ad on the inside back cover; we hope that you appreciate what we have presented.

In 2012, we intend to be back in the centerfold position, advertising as always that we are dedicated to serving you and your clinical needs for many more years to come.

Thank you for your attention and understanding. Enjoy the newsletter.

Sincerely,

Dr. Hal Myers, President, Thought Technology

OVER TRAINING AND NEUROFEEDBACK TREATMENT PLANNING

Tom Matthews, PhD

A novel model of neurofeedback treatment failure is offered in the context of strategies to plan and monitor treatment.

TREATMENT FAILURE AND NEUROFEEDBACK OVER TRAINING: A FATIGUE MODEL

You have seen it. You do a careful assessment, select a neurofeedback protocol based on your findings in combination with client needs, the first few sessions look good, but then you see mystifying side effects. The client is frightened and the clinician is not just discredited but also may feel a strong doubt about his or her own capability. *What happened?* Inadequately managed, this can mean treatment failure, but managed well it can create a bond of trust and help patients respect the power of neurofeedback.

I run a practice specialized in medically, legally and psychologically complex cases. Due to the nature of my practice, many of my patients' cases may eventually be litigated in a courtroom setting. Therefore careful attention to management of risk, and development of an explicit, well-documented clinical decision-making process, is of course particularly important. Learning to effectively respond to these concerns has been an evolutionary process, in which my patients have often served as my best teachers.

Among lessons my patients have taught me, perhaps one of the most critical has been that, at least for some treatment-sensitive individuals, there can be "too

much of a good thing." The first over-training case that I had was an exceptionally bright, willow-thin woman who entered treatment in mental and emotional shambles after extensive self-training with EEG equipment purchased online. In retrospect I would say she had already over-trained, using every protocol I knew at that time, so we used very brief training periods per session and treatment was eventually successful. Fortunately by the time the most harrowing case came along I felt confident to not over-train patients. After over 20 hospital admissions and failed trials of basically every psychotropic medication, a male client arrived to my practice for neurofeedback treatment. The qEEG I acquired correlated well with his history of repeated TBI and cluster of cognitive/ emotional/ behavioral issues. Early frontal training to reduce impulsivity and labile mood was successful, so we increased the number of neurofeedback sessions.

Just hours after a session he called. This client had a history of self-slashing. He was alone, agitated and frightened because it appeared the new treatment – which had been demonstrating the first great hope of recovery – was now mysteriously followed by agitation. I had a lonely moment myself. Good standard-practice crisis management was required, but somehow I managed a calm explanation of what was happening and what we would do about it. This was vital, and we averted any of the several possible bad outcomes. We switched to supportive counseling. After distress declined we switched training to strengthen a compensating site, then later we successfully resumed the frontal training. This was an intelligent

and highly motivated patient with good reality contact who fully understood and participated in treatment planning. In cases of serious disability it is very important to have a close social support system or a multi-discipline treatment team in place and participating in the treatment process.

Now consider what I think of as a parallel example. Suppose you lift weights at the gym and overwork an arm muscle. The produced lactic acid accumulates and irritates the muscle tissue, contributing to temporarily reduced agility and possibly a global malaise – for days you can't even lift a towel! The problem is fatigue - the over-worked muscle exceeded its ability to perfuse and thus sustain activity.

NEUROFEEDBACK FATIGUE IN THE VULNERABLE PATIENT:

Like muscle tissue, our brain tissue also depends on blood flow for nutrition, oxygen, and waste transfer. I propose that it has a similar fatigue pattern presenting as neurofeedback fatigue. And as described below, neurofeedback fatigue in a vulnerable patient can produce transitory adverse effects associated with neurofeedback overtraining.

My clinical observations indicate that certain patients are more likely to have adverse neurofeedback treatment response, and in my recent research, a fatigue model predicts those adverse effects. Simply put, certain patients are more vulnerable to neurofeedback over-training, and when that occurs they display transitory adverse treatment response that has global and lo-



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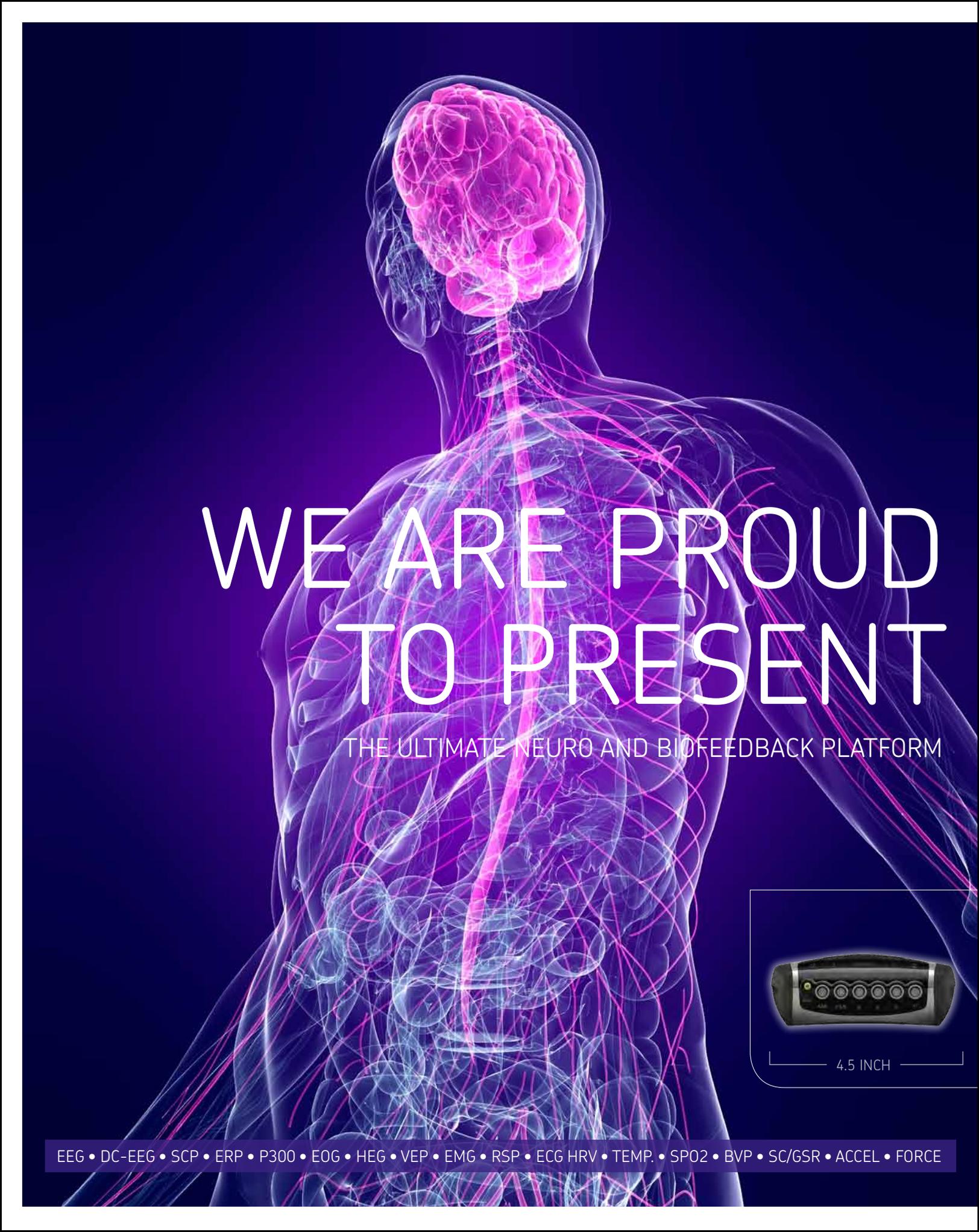
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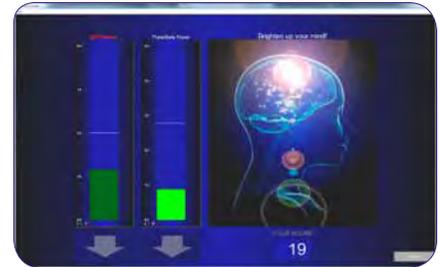
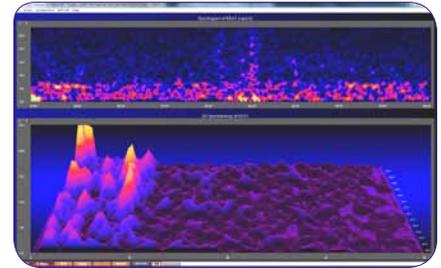
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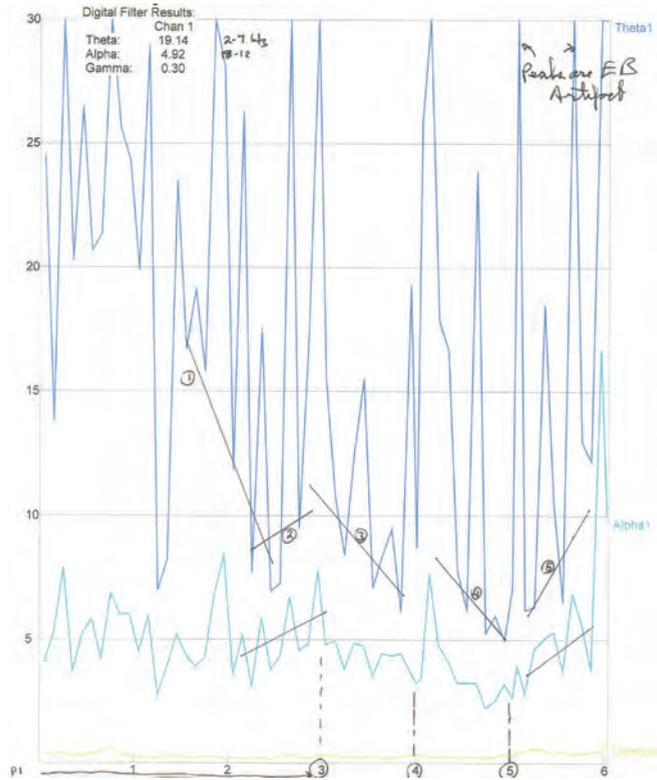
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Figure 1



caution-specific features. Some indications are marked fatigue after treatment, or reports of diverse signs of cognitive overload without situational demand to account for it. Inquiry may reveal they are worse in the behavioral domain you expected to improve. The effects can last for days or up to a couple of weeks.

That is to say, neurofeedback follows a dose-response model, modified by diagnosis-related risk factors. This requires that neurofeedback ‘dosage’ (amount and duration of session per week) be carefully titrated.

FATIGUE IN- AND ACROSS-SESSIONS

Let’s continue the example of muscular weight lifting in order to learn about neurofeedback overtraining. As you lift you may work at a moderate rate, lifting a weight no more often or rapidly than you can sustain, or you might over-work your muscle(s) in one session or cumulatively across several sessions. Your capacity to lift depends on your level of conditioning and general health; a person in rehabilitation has different needs than an athlete. Even the capable lifter will reach repetition limits within in a set, how many sets in a session, and how often the sessions occur.

If we observe a single neurofeedback session closely we find the patient shows

segments of task success or retreat - a pattern in which we can see the emergence of fatigue, which, if unchecked, may produce neurofeedback over-training. The success/retreat pattern is common among our predominantly brain-injured population, so it does not per se predict over-training. That said, one may use such observation to avoid excessive fatigue leading to overtraining in the highly-vulnerable patient. Take for example the session in Figure 1. Sampling was 250 sps averaged to 6 sec epochs. The first minute was artifact-contaminated. We should also ignore the eye-blink

artifact, and focus on the trend in the low-amplitude best-performance data-points as shown by the rough lines added to the figure. You can observe segments of 30 to 60 seconds in which inhibition of 2-7 Hz progressively improved (segments 1, 3 & 4), and segments in which it retreated (2-7 Hz increased in segments 2 & 5). Thus, the patient shows segments of task success followed by retreat - a pattern in which we can see the emergence of fatigue analogous to that experienced by a lifter who does repeated sets. In fact, patients frequently report subjective fatigue on inquiry, and failure to report is not reliable. I urge clinicians to examine within-session data for emergence of fatigue in highly vulnerable patients, based upon these findings, rather than relying on patient self-report alone. The session in Figure 1 did not produce

overtraining, seemingly because we stopped when fatigue was apparent. If training continues without modification and fatigue is unchecked in a vulnerable patient, this may produce signs of neurofeedback overtraining after the session.

Just as fatigue effects can readily be observed within a single training session, the same outcome can result from massed work across sessions. In Figure 2 one may see a series of sessions that produced overtraining signs in a severely vulnerable patient. Note that we carefully titrated dosage (the work-rest load of the session - upward across sessions, then held stable as we shortened recovery between sessions, but then there was a session after just three days that inadvertently produced overtraining. This meta-pattern has been observed repeatedly cross a number of patients, but it is difficult to predict. It is disconcerting because the protocol was perceived as working well at the dose employed. In some cases a contributing factor, such as a change in medication, is the culprit rather than massed work across sessions, but whatever the cause, subsequent management of the neurofeedback overtraining effect is the same. In contrast, there are some cases where neurofeedback progress reduces a patient’s tolerance of a chronic psychotropic medication, producing known medication side effects. Your rapport with the treating physician is going to be important in getting the medication reduced.

PREDICT AND AVOID NEUROFEEDBACK OVERTRAINING: IDENTIFY VULNERABLE PATIENTS

Diagnosis-related predictors: Based on a 2007 informal review of SMR or coherence training¹ and on a 2010 retrospective study of 4-channel z-score all-variable training², this author has previously reported that patients with a history of brain injury or blood-glucose instability are more likely

Continued on page 22

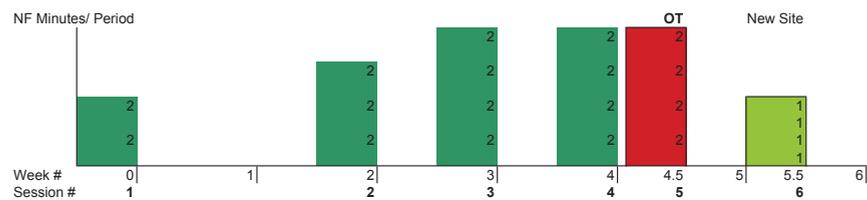


Figure 2. Cumulative over-training effects in a vulnerable patient. Training protocol was 2-7 hz inhibit at (name of site). Due to proximity of training site to craniotomy margin, we identified patient as at-risk for overtraining and limited training to 6-8 minutes per session in two minute increments. During week 4, training session frequency was increased from 1x to 2x weekly. The resulting overtraining effect in session 5 was attributed to decreased recovery time between sessions despite unchanged within-session work load. During next session, therapist adapted to overtraining effects by shifting to a new training site, decreasing duration of training runs, and increasing rest interval duration between runs. Patient reported that this was well tolerated, and symptoms associated with overtraining fully remitted.

Figure 2

HOW TO ADDRESS NEUROFEEDBACK TREATMENT PROBLEMS

Arguably overtraining is not an error but a transient and somewhat difficult to predict consequence of patient individual differences. However, over time the provider may discover that treatment problems of various sorts do occur during neurofeedback. A protocol may simply offer no clear improvement, it may be poorly tolerated, or it may have unexpected undesired results. In these cases it is helpful to rely on the interview process discussed in this article to determine what direction treatment should follow.

There also are times when the complexities of providing neurofeedback result in a procedural error. Most have mild consequence if any. Training may run too long, occur at the wrong location, employ inaccurate protocol definition, or there may be some other technical problem such as delayed detection of artifact, inaccurate display or computer malfunction. It is prudent to have a response plan in place, as it is likely a procedural error will happen in your practice. You can use a similar plan to approach overtraining if it occurs.

One viable response plan for treatment problems is this: 1) promptly acknowledge with low distress; 2) correct as soon as detected; 3) generally replace with the correct training, but avoid overtraining; 4) explain after training that there may be no, or mild, effect and any effect is expected to be transient; 5) request phone contact at 24-48 hours and offer palliative supportive care; 6) document in the chart (Enter a simple statement discussing the patient's condition, treatment plans, and expected outcomes, made immediately after the discussion; be specific and avoid speculation); 7) evaluate at the next session. At the following session treat with a reversal or compensatory protocol, or training at another scalp site, for a period of several sessions until all undesired effects are clearly absent. Inquire very closely for additional contributing causes of any undesirable effects, but avoid any minimization of the power of neurofeedback. Judging the response of your patient, consider offering supportive counseling, or a 1-2 week pause in training with continuing phone contact.

Also, it is prudent to explain before training starts, as part of informed consent, that training is complex; if something does not go as planned the effect is generally mild and transient; much like the first session of any protocol it tends to fade on its own.

You, your practice and your patients will be best served by having a disclosure plan in place, so that the focus is on planning, communicating, and conducting training. Review any guidelines applicable to your license, profession and locale, and seek legal counsel when formulating a disclosure policy. No one can promise immunity from lawsuits. Apparently a patient's decision to litigate is often associated with a perceived lack of caring, so avoid that. You might contact your liability insurer for help in devising your disclosure plan.

OVER TRAINING CONTINUED FROM PAGE 21

to experience neurofeedback overtraining events. The more recent and severe the pathology appears may be associated with greater likelihood of overtraining. The significance of brain injury or blood-glucose instability appears to be consistent with the fatigue model described above. In fact the two predictors may be linked. Published meta-analytic reviews show that diagnosed circulatory disorder is a complication for a third of patients with moderate or severe TBI, and that diagnosed endocrine dysfunction post-TBI occurs in a third of cases and is probably under-diagnosed.

How can the clinician recognize and anticipate overtraining risk factors in patients? Just ask. And explore persistently, obtain and review medical records, interview significant others, and follow-up on hints in your data. Consider referring the patient back to his or her physician for screening if you are unsure, but remember you are interested in even the more-subtle signs of glucose instability, and the remote or sub-clinical brain injury, as well as more frank pathology. For example, I have seen glucose-related overtraining in the following situations: one TBI client required glucose-stabilization during his ICU stay, then glucose signs remitted; another was prone to overtraining until his Diabetes Mellitus was controlled medically; a third denied hypoglycemic signs but his spouse indicated he was irritable when hungry.

Detecting brain injury: In my experience, recent severe brain injury will always require conservative treatment. However, be alert for unrecognized, possibly old injury. Ask about classic brain-insult scenarios such as falls, vehicle crashes, other blows to the head, anoxia, electric shock, or toxic exposure that did—or did not—have medical follow-up, and then inquire about concussion signs. Closely explore timelines of brain-related events and functional difficulties including onset of mental or other CNS-related issues. Sometimes you will not know until well into treatment. One patient made considerable progress with memory recovery and one day suddenly remembered his onset event. Years ago he ran into an electric cattle-fence in the rain, was knocked out, and when he awoke he assumed he was just intoxicated. Finally, the family could understand the mysterious drop in their child's functioning.

If you do work with a significant brain injury case—and you should carefully con-

sider whether you are competent to do so—then obtain the relevant medical and psychological history. Read ambulance notes and hospital records of the ER, surgery, CTs, MRIs, neurology, and discharge note to see the lineup of injury dynamics, medical findings, qEEG and functional issues.

MANAGE OVERTRAINING RISK: ASSESS AND DISCUSS IT WITH YOUR CLIENT!

Consider including risk of overtraining in your general neurofeedback informed consent to set the stage for the assessment and treatment design. Point out that you can minimize but not prevent all overtraining events, and that overtraining signs generally are mild and transient. Encourage your patient to contribute to the treatment process with self-report and support awareness of overtraining signs. In treatment design there is a natural trade-off: the challenge is to balance safe dose with most benefit per session. If your assessment reveals risk factors, it may be effective to start with low training dosage and increase the training time and intensity incrementally.

RECOGNIZE AND TREAT NEUROFEEDBACK OVERTRAINING

Having assessed and discussed this with your patient, he or she can help you recognize signs and calmly manage any overtraining issues that arise. In every session be alert to and inquire for any global or location-specific signs of fatigue or overload. You generally will not recognize overtraining until after the fact, usually in a phone call or in the next session. When those are present, evaluate the symptom time-course. In Figure 3 note how treatment response differs after overtraining. Usual training results in a rise-then-fall response profile that becomes less pronounced with more sessions of the same protocol and shows gradual cumulative improvement. Overtraining results in a fall-then-rise response profile, which nonetheless may produce later improvement. Pointing this out to the patient is reassuring and demonstrates the spontaneous recovery expected. Within the next session it is desirable to correct the training principally by inserting long pauses, reducing total training, or moving to a new training location. If signs persist consider using a protocol selected to compensate for the specific overtraining presentation. Track the benefits as well as the overtraining signs, and consider whether to return later to the overtrained protocol at lower dose.

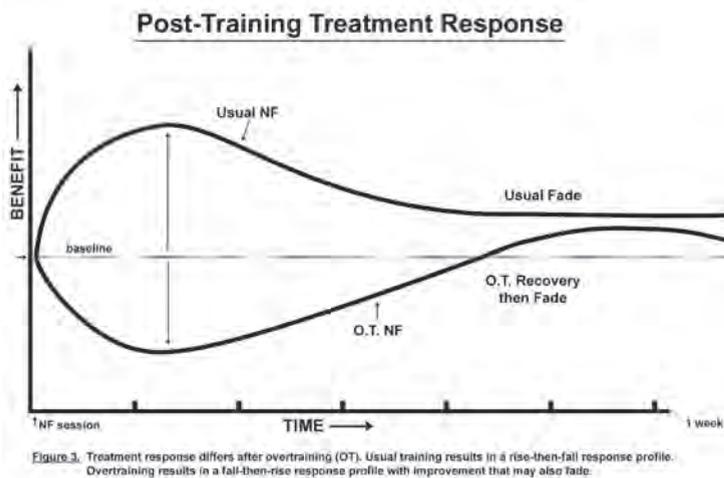


Figure 3. Treatment response differs after overtraining (OT). Usual training results in a rise-then-fall response profile. Overtraining results in a fall-then-rise response profile with improvement that may also fade.

Figure 3

HOW TO TRACK PROGRESS:

I find patients commonly report no change at the session end, but I ask and I also chart my observational impression. The key treatment-guiding information comes during the following session from patient and, especially, family report. After any new protocol I request the patient make a report by phone at 24-48 hours. Session inquiry

model is that if I cannot rule out training as the cause I entertain that hypothesis, in relation to a reasonable link to CNS function and to protocol. That is, if the patient reports flu symptoms I expect they have flu, not symptoms of overtraining. Other considerations are endocrine issues such as PMS or TBI-induced diabetes, for example. The provider having a clear understanding of the patient's medical issues is immensely

first consists of open-ended questions and then focuses on clinical benefits or overtraining effects. The task is to identify the source of any effect, whether due to training, medication change, situation, psychosomatic process, or other medical status. My

helpful. I advocate that both provider and patients use reliable online resources such as Mayo Clinic's website.

I do not use a specific rating system or metric although doing so may be desirable. Often the key finding of the session interview only emerges from careful neuropsychologically-guided follow-up questioning. For example, in exploring a complaint such as trouble reading, it is important to clarify the status of the numerous contributing systems and functions. Have they had visual screening? When does the problem occur? Is this actually a matter of attention, impulse, drowsiness, anxiety, pain, education, medication, motivation, or some other factor? The inquiry may lead to training over regions for vision, primary language, recall, visual-motor control, visual-search, attention, executive control, or—as in a recent case where it turned out the problem occurs only with reading aloud—training over Broca's area.

In another severe TBI case the patient still had trouble seeing, reaching and grasping after occupational therapy for visual accommodation and tracking. There

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SESSION INTERVIEW AND SYSTEMATIC RECORD KEEPING AS THE FOUNDATION FOR EFFECTIVE TREATMENT DECISION MAKING

Simply put, record what you inquire, what you perceive, what you do, and how they respond.

Here is a viable session outline: 1. Read your last note. 2. Inquire for adverse effect; if not offered spontaneously, inquire with open-ended questions for any relevant observations by the patient or by others; listen for and deeply inquire about target effects expected of the protocol and location, but avoid prompting; evaluate credibility of reports. 3. Now teach what to look for, and explain simply for maximum understanding. 4. Select today's protocol. 5. Conduct training. 6. Observe and inquire about response. 7. Record the note; during the session record at minimum the inquiry results, the planned and actual treatment and response, all in detail.

During the session insist on enough calm so that you can do all this accurately. I conduct 50 minute sessions, of which roughly half is devoted to the training per se. Be prepared to receive your patient and their social surround as a whole, including the frequent crises and resistance to change from the patient or the system; failure to do so will result too often in failed treatment.

The inquiry about response to last session is vital for treatment success. Strongly encourage objective and loving input by family members, and request comments by those who see the patient seldom. Family members see change before the patient does. But the caring observer who sees the patient only once in a while will still notice change when it has become too gradual for family to recognize, and yet it continues to be clinically meaningful in reaching maximum benefit. This is not a chat, but it should be friendly. It is your scientifically guided search for data about treatment response, based on fine-grain cognitive analysis of symptoms, and your knowledge of localization of function as well as usual neurofeedback treatment dynamics and effects, including any adverse effects.

OVER TRAINING CONTINUED FROM PAGE 23

was mechanical damage to the dominant arm, fingertip agnosia, and occipital slowing, but after training to the occipital, session inquiry clarified that the patient experienced motor-control and visual search deficits. We trained the frontal eye field (for accommodation), the contralateral hand-motor/ sensory and then the right parietal visual-search regions, each giving marginally more benefit.

Thus the inquiry follows lines of questioning to exclude the various systems, and seeks correlation with the qEEG findings. This is a very engaging process for your patient and family, garnering strong buy-in to the neurofeedback process and progressively more recognition of any treatment progress. It does take considerable time in the session, but it targets training precisely.

NEUROFEEDBACK PLANNING IN Z-SCORE TRAINING

In theory it seems that training simultaneously all the key qEEG findings at (and among) a number of sites would be profoundly useful and efficient. Our clinical experience with 30+ patients over the past several years indicates that training several sites concurrently offers huge great advantage. The procedure can produce rapid changes of diverse functions related directly to the underlying brain site trained. However, planning such training is complex. You must plan and implement the treatment at several sites simultaneously, requiring that you evaluate numerous measures and their relationship with both CNS function and patient needs. In some cases just focusing on the locations with the most abnormal brain wave profiles may be desirable, and in other cases you need to utilize knowledge of brain-localization of complex cognitive systems.

Multi-site all-variable z-score training also requires consistent attention to mitigation of overtraining risks. Our research² indicates that it does produce overtraining effects, and clinical experience suggests that it is more likely to do so than with simpler protocols, or at least that the best training durations are shorter. This treatment approach holds great promise for TBI and other complex presentations, but treatment success may be greatly enhanced by using the overtraining prevention and treatment approach discussed in this article.

IATROGENIC RISK FACTORS IN Z-SCORE TRAINING

Arguably any overtraining is not an error but a transient, consequence of individual patient differences that can be somewhat difficult to predict. There is opportunity for problems in z-score training. When using multi-channel all-variable z-score training the procedures are more complex and the training is higher impact, so there is more likelihood that the provider makes any of several procedural errors, resulting in greater consequence. Consider that one is simultaneously training coherence, phase, asymmetry, power and perhaps other variables.

If the wrong training, whether location or protocol, is applied, much more than frequency may have been impacted. One may have sessions in which just a few minutes of training the wrong location or protocol, followed by correct treatment, can result in several days of mild functional difficulty and then spontaneous recovery. It is rare, but in an unstable patient who was incorrectly trained for a full session may relapse to serious issues and require immediate corrective neurofeedback. In this instance, a previously successful protocol most closely targeting the dysregulation, or a simple peak-performance protocol may be indicated.

In addition, my personal observation suggests that those with skull breach are more prone to difficulties with neurofeedback training. Thus, z-score training at effected (scarred?) locations is contra-indicated. Due to the way EEG is shifted by skull breach, comparison to a normal intact skull EEG database is not valid, and z-score training over a breach may result in unexpected adverse outcome.

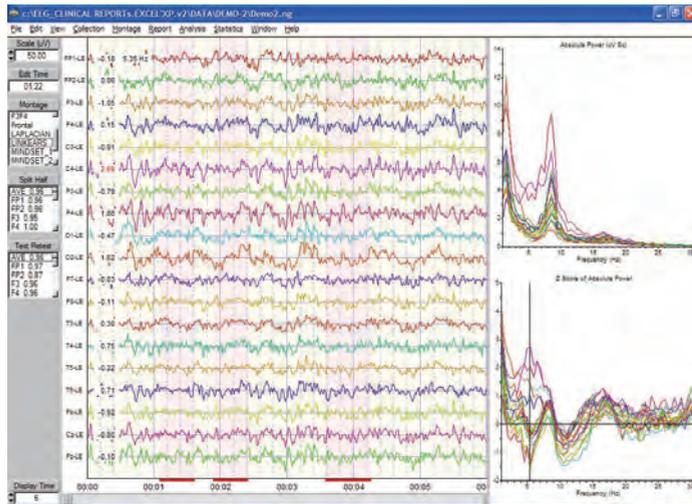
So to summarize, neurofeedback displays fatigue within and across sessions, which particularly in TBI and blood-glucose disordered patients, can produce adverse overtraining effects. These effects and treatment planning can be managed using careful session inquiry. There are particular issues to consider in z-score training. 

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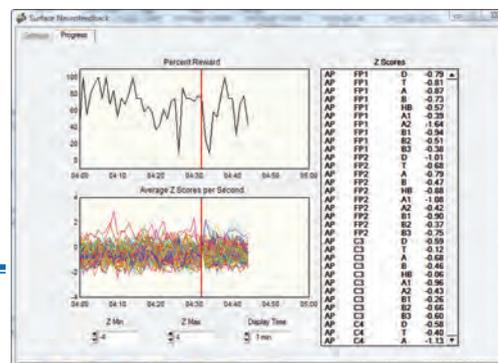
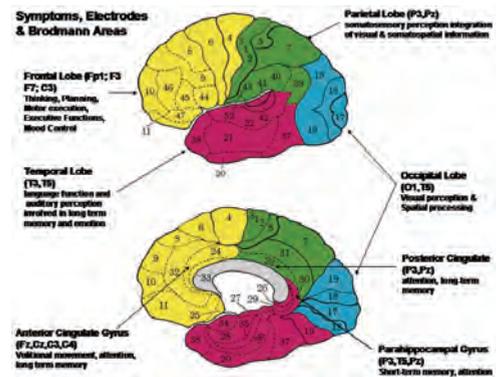
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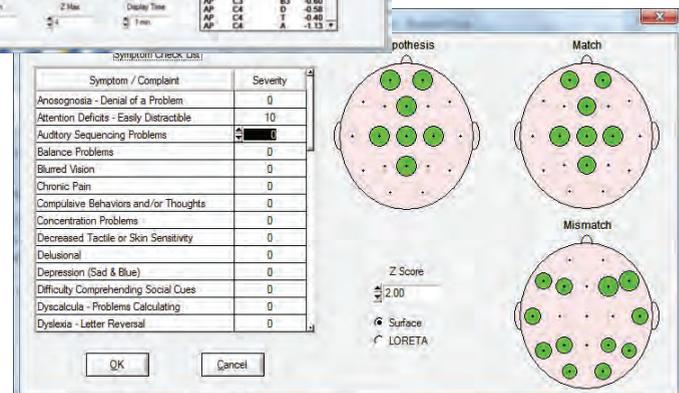
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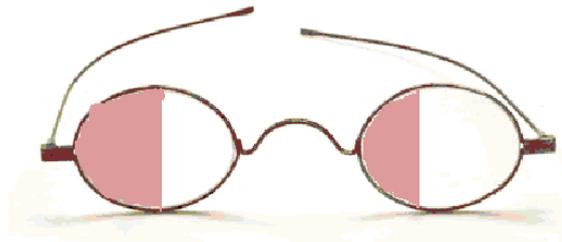
Any intelligent fool can make things bigger, more complex, and more violent. It takes a touch of genius—and a lot of courage—to move in the opposite direction.

—Albert Einstein

There is simply too much to know. Neurotherapists work at a place where psychology, neuroscience, and medicine converge, with data derived from electromagnetism and statistics. Furthermore parenting and culture weigh in on nearly every case, as does coaching, common sense, and technical prowess. When we evaluate a patient's EEG, we can quickly become overwhelmed by the amount of information available to us on the patient, the number of functional abnormalities in any person, even healthy persons, compared to group-based norms. We are all individuals, so what did we expect? We are scanning each brain in its lair of light, under its protective shell, at home, far from the maddening crowd. Did we expect it to be simple?

A normative EEG assessment identifies aspects of brain activity that are statistically abnormal from most people, and we assume these findings reflect areas of concern in the brain, too much activity, too little, too much shared, too little shared.

So let's say we've run a Q eyes closed and eyes open rest and reading and math, a range of challenges for most people, and now we are faced with dozens of significant differences compared to a healthy group. I recommended triplicating recording conditions to reduce statistical noise, to minimize random effects. Only findings that repeat are meaningful. Findings that come and go are problems the patient can handle on their own. They need the most help on those problems that won't go away. In fact triplication across two conditions like EC and EO is what I prefer, as it means opening the eyes doesn't change the basic brain energies. And still, with such a high hurdle to clear, we might find ourselves with dozens of EEG findings, especially if we analyze the EEG thoroughly. When we evaluate single-Hz bands along with conventional binning (clinical bands of delta, theta, alpha, beta, gamma) for 19 sites of



EEG activity between 1 and 40 Hz, this results in 45 bands x 19 sites x 3 activity measures (absolute activity, relative activity, variability of one or both) and 45 bands x 171 site pairs x 4 connectivity measures, and if we throw in montages and source analysis, we quickly approach infinity, or at least it feels like we do. A reasonable survey of human psychophysiology can result in 100 or 150 deviations at an alpha level of 0.05, and dozens at 0.01, and that's for a single recording condition like eyes closed. One reason we duplicate or triplicate conditions is to deal with the number of statistical comparisons we are making. I tend to ignore findings unless they triplicate in both EC and EO, requiring 6 repetitions of the same problem before I'm comfortable to stake a finding as reliable.

So what to make of 100 findings? How do I organize them? How do I rank them? I want to help this person so what should I do first? All findings are equal but some findings are more equal than others -- how do I decide where to start? Which findings will lead me to a place where a small change will amplify, to cascade him or her back to healthier ways? We need a model, and this is where sciences kick in.

Religion is belief in order without change where science is belief in order with change, which is evolution in a nutshell. Evolution is our central principle, change with benefits. So considering brain evolution and brain development during an individual's lifetime should guide our thinking.

Brain disorders are primitive responses to events, primal strategies for fulfilling one's needs and desires. Primitive thinking fails to integrate the full range of humanity into our thought, our extensive experience with others. Brain injury – functional or physical – may be conceived in an evolutionary context. Injury was thought to expose earlier stages of our processing, pulling the tarp away from the wall to reveal the scaffolding beneath. Each brain event incorporates our entire evolution in a matter of milliseconds. We scale the fish-to-human evolutionary ladder in our head every second of our days -- except when we failed

to do so. Brain injury exposes early stages of brain evolution or development, and in this fashion EEG spectral coefficients are prioritized on the basis of ontogenetic and phylogenetic age, where youthful traits (e.g., delta, low connectivity) are compared to mature properties such as specialization (e.g., frontal sites, left sided).

How do we identify the behaviors of archaic systems to those more evolved in EEG activity?

Converting evolution to EEG coefficients, ah, there's the rub.

Before we provide an interpretative guide, a rule of thumb for estimating severity, we must be confident in our results. To have confidence that we are not working with chance events, we simply replicate conditions at different times, preferably sandwiched between different challenges to see if the same abnormality emerges at each instance. Replicate, triplicate, tetraplicate, and ignore anything that modulates, comes and goes, as unreliable. Findings that appear in EC and EO recordings as well as tasks are the most reliable, brain habits unaltered by stimulation or task demands.

Here are EEG-severity correspondences, in descending importance:

1. Infrequent > frequent events (delta > theta > alpha > beta > gamma)
2. Emotional > Social > Intellectual functions (e.g., Cingulate > Orbitofrontal > Dorsolateral frontal)
3. Primary > Secondary > Associative functions (e.g., Area 17 > Area 18 > Area 39)
3. Organized abilities > single-minded abilities (Frontal > Temporal > Parietal > Occipital)
3. Unchallenging > challenging (Eyes closed > Eyes open > Easy task > Hard task)
4. Shared > unshared events (connectivity coefficients > amplitude coefficients)

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MINDFULL

CONTINUED FROM PAGE 27

5. Unpredictability > Incidence (variability > mean)
6. Number > Timing (magnitude > phase)
7. Homotopic > ipsilateral > heterotopic
8. Greater > lower statistical deviance

This ranking system is a mix of anatomical and electroencephalographic properties based on microgeny. Many articles can be written to confirm or devalue each ranking, each suggested indicator of severity. This is an interpretational schematic only, not the final blueprint where we specify and delineate which brain areas are responsible for which actions and which EEG coefficients best reflects each function.

This EEG coefficient ranking system is independent of clinical symptoms. As with any set of principles, context is everything and a neurotherapist must place any rankings into a clinical context based on experience and knowledge. 

ADDICTIONS SMALL GROUP DISCUSSION



Twenty five people attended the small group discussion on Addictions this year which was moderated by Genie and Richard E. Davis. The research for using neurofeedback for addictions was reviewed and several approaches to neurofeedback for addictions were discussed. The discussion centered on modifications or changes to the traditional Peniston Protocol (alpha-theta training) approach through the use of Quantitative EEG. Information presented included frontal and central vertex training to decrease excessive slow and fast frequency activity, especially the fast frequency activity at CZ that is often prominent in alcoholics. Also mentioned was the excessive slow alpha activity at PZ usually seen with drug abuse. This led to talk about symptomology such as anxiety and attention/focus issues and how treating those may impact the cravings and addiction issues. The discussion then moved to how alpha-theta training may be introduced after utilizing the QEEG to address eyes open training issues which usually results in fewer alpha-theta sessions than in traditional alpha-theta treatment. It was suggested that alpha-theta training using the Peniston Protocol was still very effective and could still be used as the course of treatment if access to QEEG was not utilized or available.

This was a very good discussion that could have gone on much longer if time had permitted.

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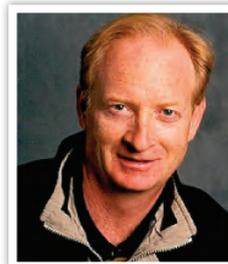


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USING BIOFEEDBACK FOR SPORT PSYCHOLOGY AND BETTER ATHLETIC TRAINING

Pierre Beauchamp, PhD, and Marla K. Beauchamp, MSCT, PhD(c)



The following article, reprinted with permission from *Advance for Physical Therapy and Rehab Medicine* (Beauchamp & Beauchamp, 2010), introduces foundational ideas of biofeedback and neurofeedback applications in a comprehensive peak performance program. For some of you, this may seem rudimentary, for others a good fundamental understanding. Providing this reprint to *NeuroConnections* readers allows us to see what the outside communities are accepting as an introduction to our field. (CRK, Ed)

As a mental performance consultant, I (first author) have witnessed firsthand the evolution of sport psychology services through working with various athletic teams and organizations including the Canadian Olympic Association, the Aerial and Mogul Ski Teams in the Salt Lake 2002 Olympic Games, Speed Skating and Ski-Cross Canada in preparation for the 2010 Vancouver Olympic Games, and most recently with Para-Cycling Canada in preparation for the London 2012 Games.

Consequently, I have had the opportunity to familiarize Olympic athletes with a range of sport psychology programs, and specifically, to introduce biofeedback training that facilitates the self-regulation and mindfulness of athletes that ultimately allows them to perform on demand and under pressure. The primary aim of this article is to introduce the field of applied psycho-

physiology to the greater sports medicine community, using my work with Speedskating Canada as an example.

ENHANCING MINDFULNESS

The rationale for biofeedback interventions in the athletic population is based on the psycho-physiological principle that states that every physiological change is accompanied by a corresponding change in the mental and emotional state. Conversely, the opposite is also true—change in thoughts or emotions will have a corresponding effect on the individual's physiology (Green, Green, & Walters, 1970).

Consequently, biofeedback can be a powerful tool for self-regulation and for enhancing mindfulness among athletes to better manage stress and pressure in preparation for sport performance (Zaichkowsky & Fuchs, 1988). Olympic athletes, in particular, do not receive second chances; therefore, the ability to self-regulate in a desired direction is a critical skill for this population (Schwartz, 1979).

SOME BACKGROUND

Much of the early work in biofeedback was limited to the medical field (Moss, 1998). However, quite a significant amount of biofeedback research was conducted in sport psychology during the 1980s and 1990s. Most studies found positive effects of biofeedback interventions on sport per-

formance and stress management (Falk & Bar-Eli, 1995). Today, the biofeedback approach reflects a transactional view of sport performance (Tennenbaum & Bar-Eli, 1995). Specifically, sport performance (behavior) of athletes within a transactional system considers the environment (e.g., situation-athletes, coaches, professional support, family) and the interrelationships between the physiological, mental and emotional components of sport behavior.

Thus, information from a variety of sources must be assimilated to develop what is known as an "athlete profile" (Blumenstein, Bar-Eli & Tenenbaum, 1997). The sources of information come from the various sport medicine team members (e.g., sport medicine doctors, physiotherapists, nutritionists, physiologists, sport psychologists, equipment technicians, strength and conditioning coaches, high-performance directors), who are referred to as the Integrated Support Team (IST).

Generally, the IST will meet on a monthly basis to review each athlete and make recommendations to the coaches and high performance director. Consequently, the group profile is also important, such that if the group profile demonstrates a significant lack of stress management skills, performance under pressure may be compromised.

BIOFEEDBACK ASSESSMENT AND TRAINING

For Speedskating Canada, the biofeedback training program was conducted at the end of year one as part of a three-year comprehensive sport psychology program leading up to the 2010 Vancouver Olympic Games. Biofeedback assessment and training were introduced after other sport psychology interventions had been completed as part of an extensive program that included mental skills education with a cognitive behavioural approach, as well as mindfulness training through a mental skills log book completed daily by the athletes.

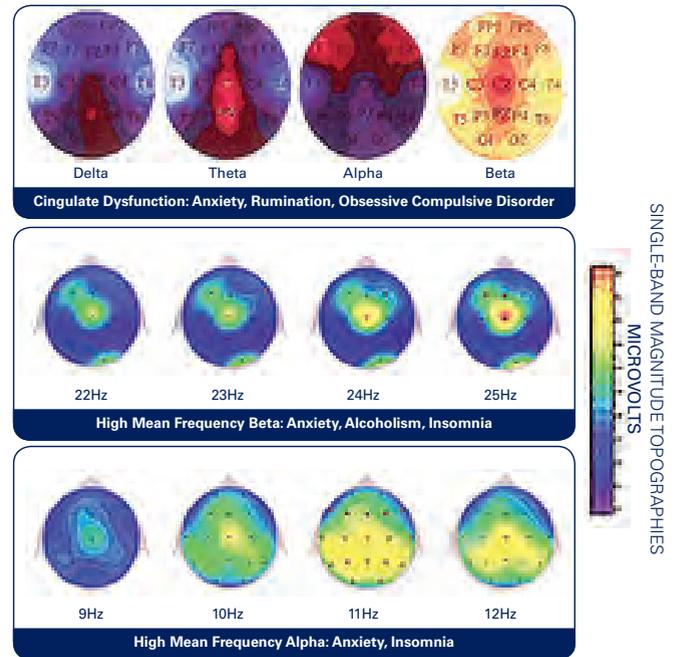
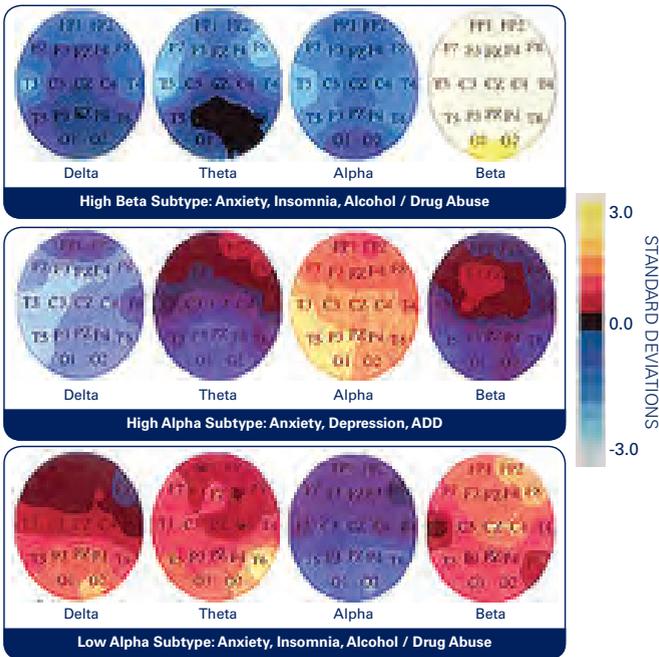
Several psychometric tests were also used to monitor and guide the direction of



Canadian speed skater and Gold Medalist Olivier Jean working on reaction time with Dr. Pierre Beauchamp

Continued on page 28

QEEG / TOPOGRAPHIC BRAIN MAPS: Generalized Anxiety Disorder Subtypes



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Established 1982

ATHLETIC TRAINING CONTINUED FROM PAGE 27

the interventions, such the Ottawa Mental Skills Assessment (OMSAT), Rest and Recovery Profile (RESTQ-S), Competitive State Anxiety Scale (CSAI-2), and the Test of Attentional and Interpersonal Style (TAIS). These data were gathered to develop individual athlete profiles and team profiles, which guided the IST and coaches in intervention decision-making. In addition, this information served as feedback for athletes, which guided their individual mental performance consultations.

The biofeedback stress assessment consisted of both psychophysiological and EEG tests to evaluate individual responses to stress under 14 conditions (e.g., a Stroop test). The following parameters were measured using software: heart rate and heart rate variability (HRV), respiration rate (RR), muscle activity using EMG, skin temperature (ST), skin conductance (SC), and brain wave activity frequency (EEG).

Training sessions were conducted each week both in the physiology and EEG programs. A competency-based approach prevailed until each athlete developed automaticity with each skill area (HRV, EMG, SC, ST, and alpha EEG training). The physiological component of the biofeedback training program involved teaching HRV to the athletes by using a 5 to 6 count to anchor their diaphragmatic breathing. Individualizing the program meant that athletes could continue in each training module until they developed the competency required before moving forward in the program. A stress test device was used for home education for those that required extra training. On average, six to 10 sessions per athlete were conducted in this phase.

The psycho-physiological training program consisted of teaching athletes alpha training such that they could relax mentally by reducing negative self-talk (Beta 2-3) and simultaneously rewarding Beta 1 and alpha, in both eyes-open and eyes-closed conditions. Once in this state, athletes were asked how they got into this state, and to give it a term to which they could return to in their next training session (i.e., centering, quiet mind). Sport-specific visualizations were also added in this alpha state to enhance confidence.

Finally, athletes were asked to use these skills in training through use of their daily log book, to consolidate them within simulated competitions. The next phase was to apply these skills in the World Cup

competition. Self-monitoring and evaluation completed the process, which ended the skill acquisition stage of year two.

BIOFEEDBACK REACTION-TIME PROGRAM

In addition to the core biofeedback intervention, reaction-time biofeedback (provided by Thought Technology Ltd in Montreal, Canada) was utilized off-ice to more effectively prepare 500-meter sprinters with their pre-start routines. This training closely followed the learning of competencies in biofeedback and EEG training. Consequently, the scope and sequence of the reaction-time program was integrated seamlessly with the biofeedback training program.

The aim of the reaction-time intervention was to better prepare athletes for the 500m sprint events at the 2010 Vancouver Olympic Games. In short-track speedskating, having the quickest reaction time combined with a good start allows you the significant advantage of claiming the inside position on the first turn, thus forcing your competitors to skate wide or follow you around the 500m oval track.

Biofeedback reaction time in combination with an individualized pre-start routine, start technique and start confidence all play an important role toward speedskating sprint success.

The reaction time equipment was engineered to improve the athlete's alertness in terms of arousal regulation (activation), vigilance and expert signal sensitivity (Cox & Hawkins, 1976) – that is, the goal was to optimize the reaction time between hearing a gun/tone at the start of a race with the initiation of the first foot movement for-

ward. Hundreds of starts could be trained this way in a training cycle without wasting much physical energy (under sub-maximal muscle tension).

VANCOUVER 2010 OLYMPIC GAMES

The goal of this multifaceted sports psychology program, with biofeedback as an integral module, was to prepare each individual skater to perform their personal best performance under pressure and on demand at the Olympic Games. The Canadian Short-Track Speedskating Team achieved its goals, both from a sprint perspective and from a team perspective.

First, from a sprint perspective, the men's team brought home one gold and one bronze medal, while the women's team earned one silver medal and a fourth place in the 500m sprints. Finally, from a team perspective, the men's team won the team relay gold, while the women brought home the silver in the team relay for a total of five Olympic medals.

FUTURE IMPLICATIONS

The role of sport psychology in a multidisciplinary context is increasingly recognized as an important component of the sports medicine team. Just as clinical athlete support is critical in dealing with injuries, the sport science support team also plays an integral role in guiding the athlete toward preparation and/or re-entry to the athletic playing field.

Multidisciplinary sports medicine centers that cater to a variety of athletes' needs will play an increasing role in guiding athletes toward injury prevention,

Continued on page 32



ATHLETIC TRAINING CONTINUED FROM PAGE 27

sport-specific training and performance enhancement.

The future appears promising with the development of multifaceted sport medicine facilities that-in addition to clinical support-will incorporate psychological skills training and strategies for performance enhancement, which may include the utilization of biofeedback, reaction-time training, vision training, sport-specific decision training, virtual reality simulators and sport performance analytics.

In particular, the use of similar methods by many of our Canadian Olympic teams in preparation for the Summer 2012 Olympic Games in London, England is encouraging. Stress control GSR training, HRV for recovery in parallel with imagery and focused attention through neurofeedback are all integral parts of the athletes' training regimen. The biofeedback and new reaction time equipment (ProComp Infiniti from Thought Technology, Montreal, Canada) is also being used to prepare sprinters, swimmers, tennis return of serve, etc. and several other sports for faster reaction times. The spread of technology in sports performance applications is exciting and

bodes well for the future, as professional teams across the world begin to realize the benefits of using the equipment and methods those of us in the biofeedback community have been intimately familiar with for many years.

As experienced biofeedback practitioners, sports psychologists and mental performance experts continue to collaborate, and companies like Dynamic Edge in Ottawa, Ontario, Octothorpe Software in Vancouver, BC and Thought Technology and Cognisense Athletics, in Montreal, Canada continue to work closely with professional organizations like the Ottawa Senators, Vancouver Canucks and Pittsburgh Penguins of the National Hockey League and Chelsea FC, Manchester United of the British Premiership (soccer), the boundaries of biofeedback for peak performance are expanding at an exciting rate. 

Pierre Beauchamp is a mental performance consultant with Peak Sport Performance Mindroom, in Montreal, Quebec.

Marla K. Beauchamp, MScPT, PhD is a physiotherapist and PhD candidate in the Graduate Department of Rehabilitation Science at the University of Toronto, Ontario.

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Biofeedback & Neurofeedback Applications in

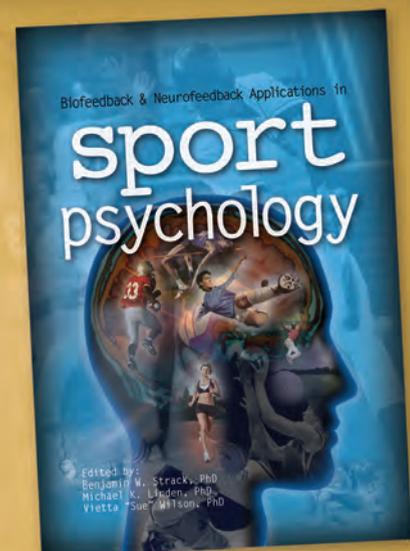
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TO Q OR NOT TO Q?

Cynthia Kerson, PhD, BCN, BCB

► THOMAS BEARDEN, PHD

I always QEEG before beginning training. It might not be absolutely necessary in all cases, but since I have the capability I do it routinely. I know it has prevented me from rewarding excessive beta on quite a few occasions and helped me tailor my training to be more efficient. I rarely do over 20 sessions. I also always do a pre-post training IVA. I find this enormously helpful for assessing training success. I would be interested in whether others routinely use pre-post CPTs.

► PAUL SWINGLE, PHD

Frankly, I'm surprised that this debate is still going on at the level of ISNR. Many of us have been trying to get the one-size-fits-all franchise-like operations out of business. They all argue that one need not understand what is going on neurologically in order to start fixing things. Rather like restricting all physical exercise to the stationary bicycle. The bored perfume salesmen who want to become neurotherapists buy franchises from people like Gerdes who has no training at all and set up shop claiming to be equivalent to professionally trained neurotherapists. The "Q or not to Q" debate at the level of ISNR can be interpreted as legitimizing their collective claim that the jury is out.

One example should suffice to correct this nonsensical notion that we should continue the business-as-usual top down method of treatment. Client's presenting complaint "depression." One-size-fits-all treatment has protocol for depression. However, depression can be associated with at least the following neurological conditions: Elevated 8-12Hz at F3 relative to F4 (the original Davidson finding); elevated 3-7Hz at F3 relative to F4; elevated 16-25Hz at F4 relative to F3; elevated 3-7Hz/16-25Hz at F3 relative to F4. For agitated forms lets add deficient 3-7Hz/16-25Hz at O1; elevated 16-25Hz at F3 relative to F4; elevated 28-40Hz/16-25Hz at Fz. Of course the previous examples do not include the hyper-vigilant forms associated with deficient 8-12Hz frontally or those with neurologically demonstrable emotional trauma markers found at Cz and O1.

There is some debate among professionally trained neurotherapists as to just

how extensive the initial evaluation must be. I prefer an abbreviated initial intake to provide instant feedback to the client followed if necessary by a full Q. Others feel starting with the full Q is preferable. Neither of these preferences supports the Q or no Q debate nor the profession-undermining activities of the one-size-fits-all franchises that require their trainees to have at least warm blood and a check book.

► STU DONALDSON, PHD

I require every person who is seen at this clinic to have a qEEG done. My reasons are as follows: a) how do you know what to treat, what frequency to decrease at what site, or visa versa, b) are there issues with phase and coherence which need to be treated and when using Z score training/coherence training what sites do you need to target, and c)

most importantly to protect my ass as I have an objective reason for doing what I am doing. What I am saying is every person seen at this clinic has an individualized treatment program. The qEEG directs this.

► DAN TUTTLE, LCSW

I strongly advocate doing a Q first. I have yet to have a case I haven't done at least a MiniQ II on. My reasoning is that while in general such a protocol "A" is indicated for condition "Y" there are exceptions to the rule. For example, there is a subset of ADHD kids I see that have excess beta through central and anterior portions of the head. If I were to apply traditional protocols like SMR, I'd be exacerbating their pathology. And that would be irresponsible of me and I would be doing harm . . . or as they taught me in school for my MSW, I would not be acting "in the best interests" of my client, which would violate my code of ethics. I know there are cases where things work out OK without a map, but to me that's a risk I'm not comfortable with taking.

► REX CANNON, PHD

In thinking about the empirical need for qEEG/LORETA-guided neurofeedback, one prevalent notion resounds; a starting point must be established to have an ending point. In simplest terms a pre-post outcome

must be established in order to measure treatment efficacy. In graduate school, we are taught to use the science-practitioner model to effectively evaluate and treat patients. Hence, every patient is a case study that ought to have sufficient data to support treatment and to report a successful outcome. This data should include within subject measurements (e.g. qEEG and psychometric testing) in addition to external ratings by parents, spouses or caregivers. As an example I will discuss a case of traumatic brain injury with the qEEG and LORETA maps over a course of NF therapy. Quantitative EEG (qEEG) is comprised of computerized imaging and statistical procedures to aid in the detection of abnormal patterns associated with specific pathological conditions. qEEG is a direct signature of neural activity and provides ideal temporal resolution in the millisecond time domain in addition to immediate recording of neural activity as opposed to delays in other methods (Coburn, et al., 2006; Hughes & John, 1999). Both qEEG and LORETA are important methodologies for demonstrating the direct associations between psychiatric conditions and symptoms with neurologic functions (Hughes & John, 1999) as well as monitoring pharmacological effects (Saletu, et al., 1997), and monitoring treatment outcomes (Czobor & Volavka, 1991; Prichep, et al., 2002). Additionally, qEEG affords the opportunity to examine the brain's electrical activity during longer periods of time in variable experimental conditions due to its noninvasive properties (Cannon, 2009; Cannon, Lubar, & Baldwin, 2008). qEEG combined with LORETA source localization will ultimately prove to be an invaluable tool in both research and clinical settings. This section briefly reviews research findings in normal populations and in psychiatric disorders. It is important to note that qEEG and LORETA can be a very powerful adjunct to other, more established techniques such as fMRI or PET. The benefit to using these types of measures in unison is increased discovery of frequency specific functions in the human brain.

The validity and reliability of qEEG has been an area of concentrated study (Corsi-Cabrera, Galindo-Vilchis, del-Rio-Portilla, Arce, & Ramos-Loyo, 2007; Corsi-

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TO Q OR NOT TO Q?

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Cabrera, Guevara, Arce, & Ramos, 1996; Corsi-Cabrera, Solis-Ortiz, & Guevara, 1997; Guevara, Lorenzo, Arce, Ramos, & Corsi-Cabrera, 1995). Therefore, many of the arguments against the computerized analyses of EEG may not carry much validity, especially concerning the fact that there are no existing studies published to date that show significant reliability for non-computerized analysis of the EEG signal (Hughes & John, 1999). The spectral analysis of the qEEG is useful for revealing additional signs of brain dysfunction in individual patients as well as a valuable research tool for revealing statistical differences between groups (Coutin-Churchman, et al., 2003). Hence it is very important to consider the clinical and research potential of qEEG computer assisted diagnostic procedures in performing accurately in some cases with the fundamental acknowledgement that only one source of information is not adequate criteria for diagnostic purposes (John, 1989; John, Pritchep, Fridman, & Easton, 1988).

The data shown is from a 37-year-old, Caucasian male presenting with traumatic brain injury (TBI) as a result of an automobile accident. Neurofeedback was implemented nearly 10 years post trauma, due to his desire to address memory problems, severe grand-mal seizures, prominent tremors and difficulty in communicating and self regulating. Figure 1 shows the pre-training eyes-closed baseline. Tremors were quite profound extending to upper extremities, thus his caregiver was asked to help stabilize body movements to obtain the recording. There are substantial amounts of high amplitude slow activity and increased fast activity in parieto-occipital regions. Figure 2 shows post training ECB (after 60 sessions of alpha training in left parietal-occipital region (between O1-and P3). This location was selected according to empirical data supporting the role of left parieto-occipital regions in language, memory and self-regulatory processes. Delta and low-theta (1 – 5 Hz) activity was inhibited at T3/F3. Figure 3 shows the differences between pre and post NF training. The most notable increase was at BA 31 posterior cingulate with additional significant increases in frontal and parietal regions. Figure 4 shows the pre training eyes-opened baseline. The tremors were more intense in the EOB and notable high amplitude slow activity is noted, as well as increased faster

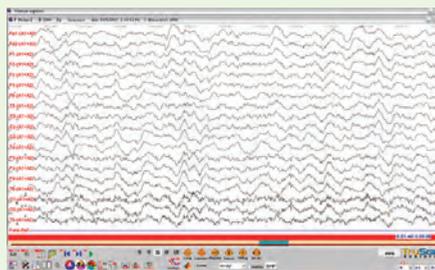


Figure 1 Pre NF training ECB. 6 seconds of EEG, note the high amplitude slow wave activity and faster frequencies in the occipital regions. Tremors were present and caregiver had to aid in helping the patient remain still for the recordings.

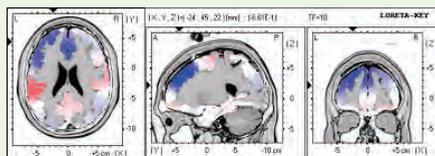


Figure 2 Pre ECB LORETA CSD. Severe deficits of alpha in frontal regions and contra-lateral parieto-occipital regions. He also showed extreme deficits in slower frequencies in frontal regions, with the exception of increased delta in left fronto-temporal regions.

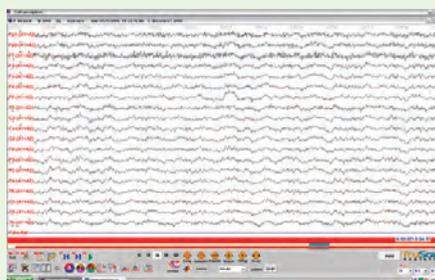


Figure 3: ECB recording at session 60. The high amplitude slow activity has subsided substantially. In combination with medications the seizures have reduced significantly, language has improved markedly and tremors have diminished; however, working memory has improved only moderately.

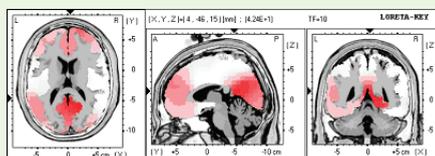


Figure 4: Difference between pre ECB and session 60 ECB. Significant increases were noted in frontal, parieto-occipital regions with the largest increase at BA 31 posterior cingulate. This is shown in the mid-alpha frequency (10 Hz).

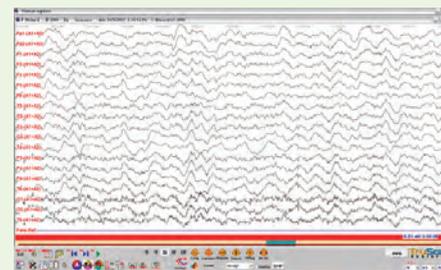


Figure 5: Pre EOB recording with high amplitude low frequencies and asynchronous patterns throughout the EEG. Excessive high frequency activity is shown in parieto-occipital regions.

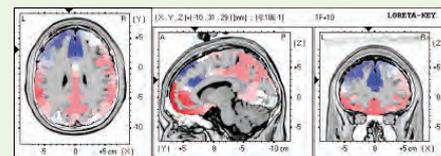


Figure 6: LORETA CSD for pre training EOB. Deficits in mid alpha are shown in left prefrontal and anterior cingulate gyri.

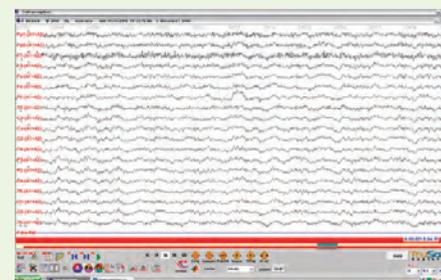


Figure 7: EOB baseline at session 60. The high amplitude activity has diminished significantly as did the higher frequencies in parieto-occipital leads.

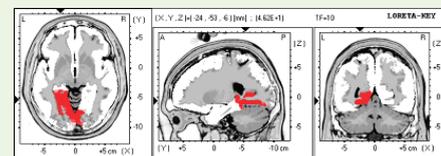


Figure 8: Significant differences between pre EOB and session 60 EOB recordings in the 10Hz alpha frequency range. The specific areas that show increase are BA 19/18, 30, and 32 – scaling does not show frontal increases as they were much lower than posterior regions.

frequencies in parieto-occipital regions. Importantly, there is agreement between ECB and EOB recordings in the regions of deficit or excess activity. This is but one example of how changes in the EEG can be monitored with qEEG and LORETA to demonstrate the efficacy of NF, as well as other treatment paradigms. Moreover, the presented data stress the importance of an interdisciplinary approach to specific syndromes. Certainly without medications and its effects on seizure activity specifically, the NF paradigm may not have been as effective. Notably, the memory issues are the focus of an updated paradigm to influence low-beta power at F3 and inhibit slower frequencies. This patient's results still show dramatic deficits compared to the database, while showing significant improvements compared to pre NF. His overall functioning has improved in motor control, tremor reduction, seizure reduction and speech production and communication. Thus the combinations of NF and medications have made a substantial impact on his global functioning.

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➤ JIM EVANS, PHD

For many years I have been doing qEEGs as part of neuropsychological examinations and generally find them to yield very useful diagnostic information. Over the same years I often have done them for neurotherapists, but am not as certain about their value for that purpose. In our group we always require a qEEG prior to starting NT, but I do many for another therapist (Othmer trained) who often requests one, but rarely uses results in actual training. She notes that she likes to have one, especially in cases of TBI, to check for EEG signs of anything warranting referral to a neurologist, and occasionally consults qEEG results when there is lack of progress in "stubborn" cases. My observation is that she gets excellent results even without a qEEG. But, our group also gets excellent results using qEEG-driven protocols, and very different training methods. I also am aware that there can be very different suggestions for specific training protocols given in regard to the same qEEG record by even the recognized experts in our field. Being a person who likes a systematic diagnostic procedure prior to beginning treatment (the X-ray before operation analogy), I plan to stay with the qEEG-first approach. But, I have to admit there is lots of evidence that it is not always necessary for success. I still have hopes that refinements in QEEG will enable great precision in treatment planning. And, even though X-rays don't always pinpoint the problem and even may sometimes suggest a problem that really doesn't exist, I still would want one done before any surgeon operates on me.

➤ GERALD GLUCK, PH.D., LMFT, BGN-SR. FELLOW

Neurofeedback has always been based on altering the EEG. When we went from

primarily analog to digital instruments we quantified the EEG. Quantification gave us more precision in matching human behavior to the EEG and helped to link the symptoms or target behaviors to brain function. We know now that NFB affects blood flow and changes in synapses and dendritic growth. Why should measuring the human EEG be subject to any different laws, or usefulness for that matter, than measuring personality traits (e.g. the MMPI) or liver or kidney function? The answer is that the human brain is a unique organ that creates meaning. We get seduced into measuring the changes in meaning and action and forget the underlying organ that generates the meaning and behavior. Few of us spend time discussing the philosophical impact of various liver enzyme levels. As a result of this special quality of the brain as both organ and meaning maker, we have bifurcated our thinking into only measuring one or the other. In fact we must measure both, the behavior of our patients, their actions, AND the behavior of the brain and link the two together. All that Quantitative EEG does is help us do this precisely, in a way that ties us to existing literature in related fields of neuropsychology and neurology and lays the foundation for our field as a science by making replication easier and reliability and validity more readily able to be established.

That being said misuse and abuse of the technique can lead to distortions and justifiable criticisms. We treat people not lab results, but we need information from BOTH in the most comprehensive and reliable way possible. QEEG is neither a substitute for clinical judgement nor experience but a marvelous lens to help us link what we see and hear in the patient to our training experience of a physical organ. It seems self evident that we should measure the very thing we are trying to change, just as psychotherapists have measured personality and behavior as they have endeavored to change those dimensions through psychotherapy.

➤ GARY AMES, PHD

I have a variety of equipment and use the procedures associated with them.

- **PIR HEG.** Needs no assessment. Just place on Fpz with everyone. Later I might search for a cold spot.
- **Zengar NCP.** C3-C4 for everyone. All get a relaxing experience.

Continued on page 36

TO Q OR NOT TO Q?

CONTINUED FROM PAGE 35

- **NeuroField.** Some standard protocols and the rest is symptom based.
- **Z-Score.** I start with a standard placement and then search for targets of interest. This is Q-like.
- **Alpha-Theta training.** Based on symptoms and trauma history.
- **LENS.** Mapping is part of the procedure, but this is single site at a time. Not really a Q, the relationship is controversial.
- **Roshi** merely requires a hardy brain. My thanks to Len Ochs for the concept of sensitivity.
- **HRV and biofeedback.** Symptom-oriented. Some do a psychophysiological assessment.

I am working on bringing Paul Swingle's QuickQ to the BrainMaster Atlantis and Discovery. This will be sufficient for 90+% of clients. Those with brain injury and seizures need a full Q.

Reasons not to Q.

- Messy, expensive, not reimbursed. Practitioners do not agree on how to follow the map. Too few post-training Qs.
- Qs generate a good feeling of substance in the practitioner and perhaps client. It may motivate clients to complete enough sessions.

Thus, do Qs if it fits your practice and personality. Do not be chauvinist about the ascending orthodoxy, which may now be waning.

➤ **LEA LEONARD, LCSW,
CTS, BCN**

I am humbly newly certified in neurofeedback. I have adopted the principal of using qEEG to guide each and every one of my neurofeedback protocols. It makes sense to me to get the whole picture before choosing a protocol; which also coincides with my philosophy of psychotherapy—I assess the whole person including their familial, social, emotional, occupational and cognitive environments before reaching into my toolbox for an intervention.

➤ **DEB STOKES, PHD**

I obtain minimaps on all my clients but full qEEGs only whenever there is a learning disability, head injury, neurological complaint or some unusual-appearing activity on the EEG or mini map. I work a lot with migraineurs and we do very well with these clients without having them go through the expense and time of obtaining a full qEEG. The minimap gives us an idea of how to proceed.

➤ **ROBERT E. LONGO, MRC,
LPC, NCC**

I think the responsible way to determine protocols is to do QEEG. If we ever hope to have formal recognition as a field, we can't have people treating with their best guess. Every medical procedure requires some level of assessment or testing, NFB should require a Q.

➤ **JOHN ANDERSON, MA**

I have 4 criteria that mandate a 19 ch EEG with database comparison:

1. Hx of traumatic head and/or brain in-



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jury (this can be a fuzzy issue – does the head injury qualify as serious enough, etc.? - it becomes a judgment call and I tend to favor doing the QEEG rather than not)

2. Hz of seizure disorder - either in client or family
3. Serious neurological and/or degenerative disorder such as Parkinson's, MS, etc.
4. Training has progressed for 5-10 sessions without discernible improvement and I need more information before continuing

I do of course do QEEG with clients who don't fit these criteria and I encourage all clients to agree to QEEG but I don't require it unless these criteria are present.

► **J. LUCAS KOBERDA, MD, PHD**

We use QEEG for guidance and use symptoms approach only for cases with normal brain mapping. In my neurology practice I perform QEEG and computerized comprehensive memory-cognitive testing and design neurofeedback protocols. I have 3 psychologists who work in their own practices and perform neurofeedback based on my recommendations. We have created neuro-science group where we discuss the cases and design protocols for our patients. Practice also has ancillary services including physical therapy and audiology and balance testing.

► **MERLYN HURD, PHD**

To Q or not to Q ... as probably others have stated, Hamlet has nothing on neuroscientists when they ponder that question. Of course, Hamlet was contemplating to live or to die which, when looked at from that standpoint, makes our question ring with a similar truth.

Dr. Daniel Amen's reflection in his book "Change Your Brain, Change Your Life" was the wake up call to me. He noted that in every rotation during medical training the organ being treated was intimately and thoroughly examined, except when he got to Psychiatry. The examination of the brain was not even considered. He thought that was incorrect and so do I.

As neurotherapists, we are working on the most complex, fragile, and most important system of the human being. When I began providing neurotherapy, I used Adam Crane's very simple 'measure the brain from

Cz' system. That seemed the way to go, and worked especially for addictions and anxiety. Then, as usual, there were clients for whom this did not work. More information was needed and Lexicor came up with imaging the whole brain from scalp readings. Wow! Now there was too much information so they had a normative database for us to define the connectivity and balance of the brain. More clients were being helped with even shorter sessions. Symptoms were always the bedrock of the selection of protocols, while the QEEG enabled defining the issues more accurately. Studying and being trained in using only symptom-based neurotherapy did not make sense, even though I tried to embrace the method. Certainly, if there was a way to treat the client without the expense and time consumption of a QEEG, it made sense. Since I had long before fallen in love with Luria's writings of the operation of the brain, the symptom-based approach and Luria's findings somehow did work together.

QEEG based neurotherapy has the advantage of being objective; either the connectivity, balance and distribution of energy is there or it isn't. Either there is change in the brain or there isn't. Our field needs to be based as solidly as possible on objective data. Self-reports are powerful and necessary and become even more powerful when corroborated with objective findings. Whether we can afford to be a field that sometimes uses objective data and sometimes not is an awesome question. We need to, at least, ensure that each practitioner has a deep, deep knowledge of the workings of the brain, the connectivity, balance and energy of the brain and the changes that can affect it. Without that we continue the medical model that Dr. Amen encountered, don't look at the actual organ, just figure out the medication (protocol) from the symptoms.

SIEGFRIED OTHMER, PHD

It is likely that neurofeedback informed by QEEG analysis has a glorious future ahead of it. I venture that forecast despite the fact that scientists have a poor track record for predicting the path of technology—particularly in their field of expertise. What is clear in retrospect is that QEEG-driven training has a less glorious past. So let us come to terms with the past briefly before we look forward.

In a discussion I had with Barry Sterman in the early nineties, he said that in his aspirational view of the neurofeedback

ideal he would wish that if a client showed no QEEG anomaly then the ethical neurofeedback practitioner would be compelled to say "I'm sorry, there is nothing I can do for you." This was the time when Barry was just getting acquainted with the Lexicor, so the facts to support this 'maximalist' position were clearly not in hand. Moreover, this was in complete contradiction to Sterman's own prior research history. His cats did not qualify for EEG training by any blotch on their QEEG.

The early position mandating QEEG analysis to guide neurofeedback was to a large extent faith-based. In the graphic terminology of the Downing Street memo, the 'intelligence was fixed around the policy.' As a result, the organic development of the field was profoundly distorted by the need to buttress what continued to be a poorly supported paradigm, albeit one that was still considered indispensable. Of course there were always sterling successes to be reported, but that held true for all the principal therapeutic approaches in the field.

The irony is that most of us continued to find success in the very approach that Sterman and Lubar firmly established in research originally, namely mechanisms-based training. Using targeting on the basis of functional neuroanatomy, we have been broadly addressing the self-regulatory properties of entire systems: arousal regulation; affect regulation; attentional and executive function; autonomic regulation and interoception; etc. Disregulations in these domains are not typically accompanied by localized cortical anomalies. Yet our experience is that these disregulations yield readily to standard training protocols, subject to some parameter optimization on the basis of client reporting and outcome measures.

An illustrative example is to be found in our experience with PTSD in veterans and active duty servicemen. Some 15% of such clients find their most critical symptoms largely resolved within less than ten training sessions. It would be difficult to make a case here for a major redirection of the approach. A further 60% of such clients respond with a typical learning curve pattern across essentially all symptoms associated with PTSD, and reach substantial resolution in twenty to forty sessions. One would love to do better, but already these outcomes exceed those of all prior therapies for PTSD. All this is accomplished with four basic training protocols, with two

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of these bearing the major burden. In fact, most of the early symptom abatement is accomplished with only a single placement.

Finally, there is a residual 25% or so that is either resistant to our ministrations or show very slow progress. Here we are clearly obligated to look for additional measures, and I believe this is where QEEG analysis will come into its own. Matters sort themselves out quite nicely when one simply tracks learning curves for all the particular client complaints. If all of the symptoms are on the same learning curve, or nearly so, then we are dealing with a system-scale dysregulation that will yield readily to simple protocols. On the other hand, if there is a large dispersion in learning curves then one may venture that more specific targeting on the outliers could confer an advantage.

We are on a continuum here from the more generalized dysfunctions to the more localized and specific. Undoubtedly somewhere on that continuum the advantage shifts from a protocol-based approach to a more focused targeting strategy that is informed by the QEEG. It remains true, however, that the vast majority of cases encountered in clinical practice yield substantially to a minimal set of standard approaches. The overwhelming bulk of the clinical evidence accumulated of the years documents the broad generality of neurofeedback training effects rather than the claimed narrow specificity. We are interacting with a highly integrated regulatory regime, after all, and our intrusion into the affairs of the brain effects a broad and diffuse renormalization. This has important bearing on the economics of a neurofeedback practice.

Quantitative assessments of the EEG will help us to push what we already do well to a yet higher level. Even though we do well with the ADHD spectrum generally, there remains the observation that this population is highly heterogeneous with respect to various aspects of executive function. This calls for much more functional testing, but in future that will be accompanied by simultaneous evaluation of the EEG. It is time for the neuropsychologists to finally show up.

In the main, neurofeedback to date has concerned itself with issues that surface in a mental health practice because they call attention to themselves. An even larger class of brain-based problems con-

sists of what may be called stealth conditions. These consist of the deficits that may have been accrued by virtue of minor brain insults in childhood, or at birth, plus specific learning disabilities and sensory processing disorders. Clients may be quite unaware of the limitations under which they are laboring because the deficits have never been tested for. This is the natural domain for basing remedies on QEEG-derived information.

The assessment challenge presents us with yet another irony, because the dysregulations of core functions tend to be associated with obvious EEG elevations at low EEG frequencies, whereas dysregulations in the cognitive domain tend to be associated with deficiencies at higher EEG frequencies which are not nearly so obvious. So the obvious features of a deviant EEG are not terribly helpful to us, whereas those that could be helpful are not terribly obvious. In consequence, much of the information of interest must be obtained under challenge conditions.

The two ends of the spectrum are well represented by two clinicians who both base their work on the QEEG. Tanju Surmeli, a psychiatrist in Istanbul, largely publishes on disorders involving severe core dysregulations, including mental retardation, Down Syndrome, etc. At the other end of the spectrum, Kirt Thornton largely addresses specific cognitive dysfunctions with highly targeted challenges. The methods used by Surmeli for the above conditions tend to be very straight-forward, mostly emphasizing training in the delta band. I take it as a given that the delta band is a fruitful target for training quite irrespective of any EEG anomalies observed there. The QEEG in these cases is not very instructive. In Thornton's approach, on the other hand, it is indispensable.

The development of our own work over the past five years makes the above distinction even more apparent. By working at infra-low frequencies, we appear to tie much more directly into cortical activation dynamics. It appears that the slow cortical potential yields a direct correlate of cortical excitability, which is just what we wish to train for the improved regulation of persistent states. And just to complete this picture in contrasts, we even have a set of 'stealth conditions' in this domain to which QEEG measures are largely blind. These include sociopathy and other personality disorders. They are in fact trainable with standard methods. Looked at from this vantage point, in which state

regulation training is mainly assigned to the infra-low and low-frequency EEG region with standard protocol-based targeting, then our perspective on frequency-based training should shift more to the issue of what it can offer specifically, and to that enterprise QEEG-derived information is indeed relevant.

There is a natural hierarchy in which one moves from general regulation issues to specific dysfunctions, and from general protocol-based training to specific targeting. In retrospect, then, QEEG-prescribed neurofeedback training has suffered from a case of what the FDA calls "mis-branding." In a well-meaning but misguided attempt to use the QEEG to confer upon our field the aura of quantitative rigor and of specificity, the QEEG was utilized principally in the task of general state regulation, to which it was not well suited. Once QEEG-based training is no longer yoked to this task---and not until then---it can assume its proper and exalted role within the field. We have been guilty of using a race horse to pull a plow.

The following hints at what the future may hold. Evoked potential studies are just now at the point of becoming useful for informing neurofeedback targeting strategies. This is because independent component analysis (ICA) allows the rather amorphous ERPs to be deconstructed into their specific functional constituents. With the aid of sLORETA for localization, these can then be specifically targeted with coherence-based training of critical linkages. With the additional insight provided by the determination of 'directed coherence,' neurofeedback strategies may be further refined.

Not all neurofeedback practitioners will take this road. In fact most will remain quite content to work with state regulation issues, just as most psychologists do not see it as a deficiency that they are not neuropsychologists. The diversity within neurofeedback will come to reflect the diversity that exists within the health professions, and it will come to match the diversity in the problems we routinely encounter. As one surveys this terrain, it is not unreasonable to forecast that the largest opportunity for neurofeedback will lie in boosting the functional competences of the non-clinical population, the key to which will be testing and characterization. Augmenting competences is the more natural frame in which neurofeedback should be discussed among ourselves and promoted to the outside world. 

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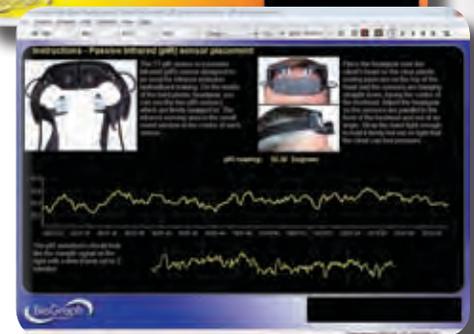


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