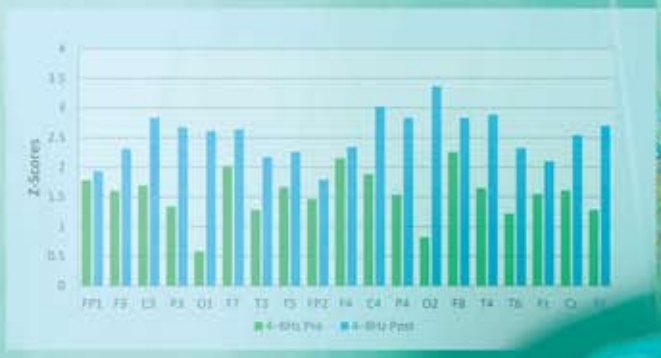
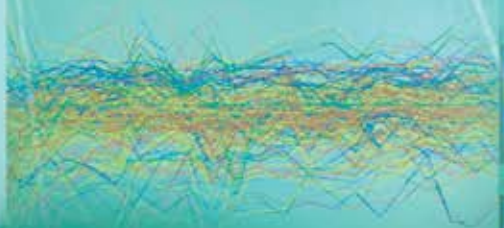
NEURO CONNECTIONS Newsletter

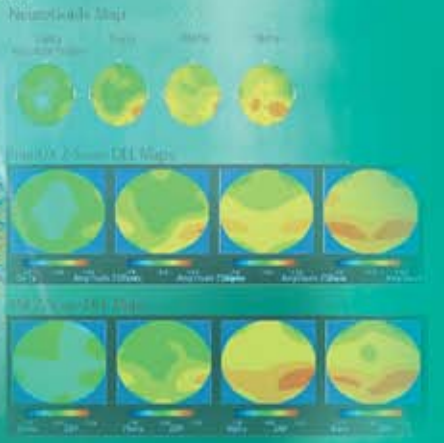
Spring 2014

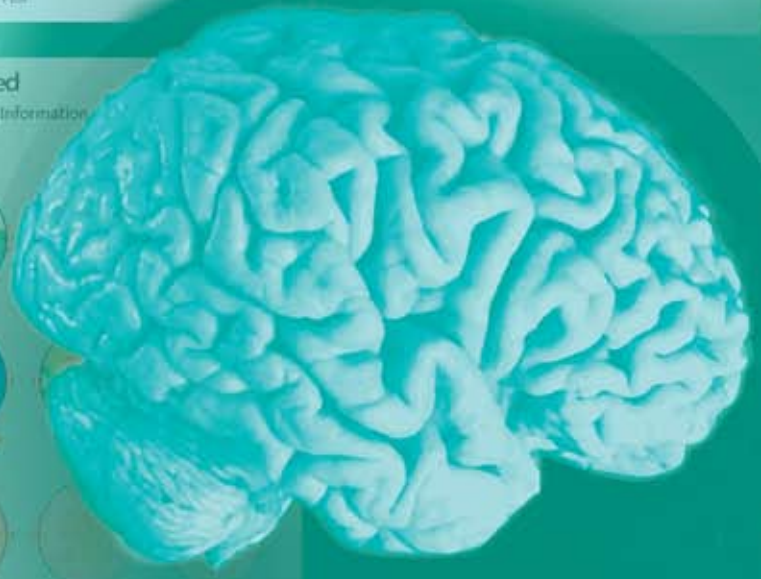
ISNR aapb

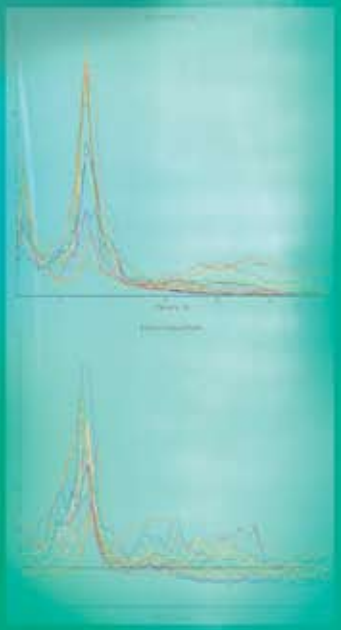
A joint newsletter from the *International Society for Neurofeedback & Research* and the *Association for Applied Psychophysiology & Biofeedback, Neurofeedback Section*

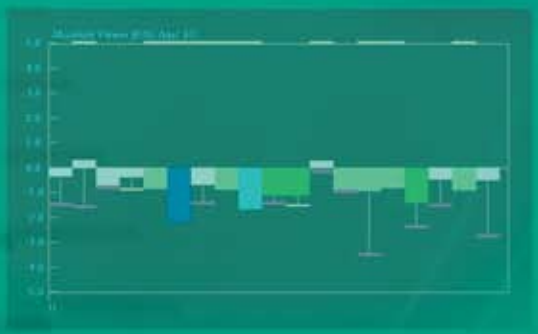












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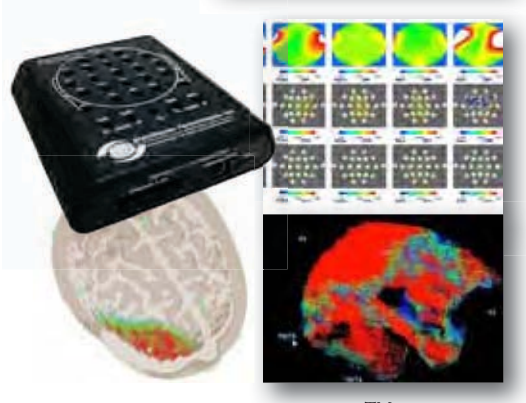

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by **Thomas Collura** PhD, QEEG-D, BCN, LPC

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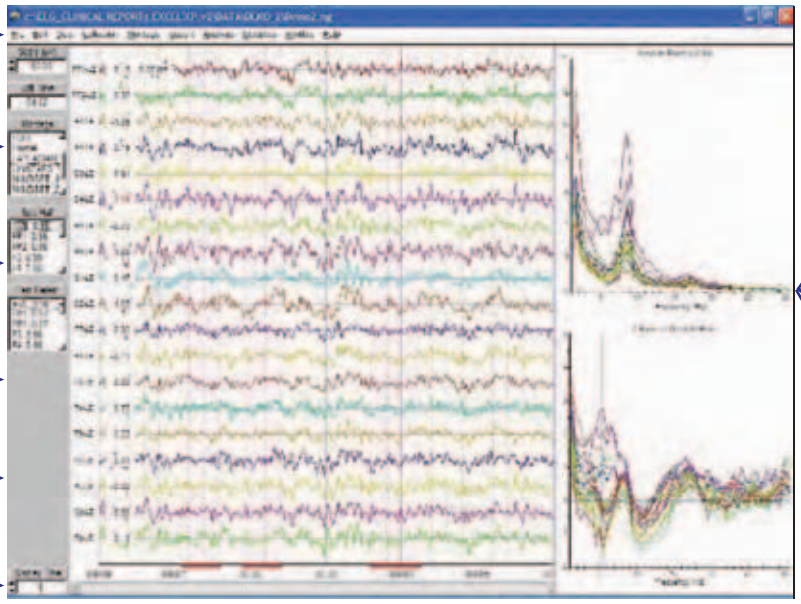

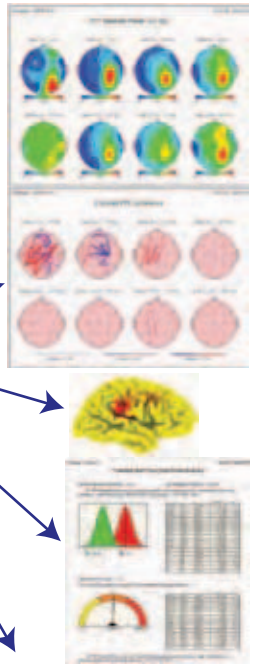

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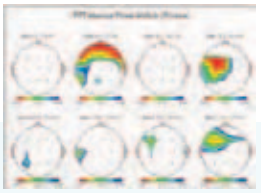
Pre vs. Post Treatment Statistics and Z Score EEG Biofeedback & LORETA Z Score Biofeedback



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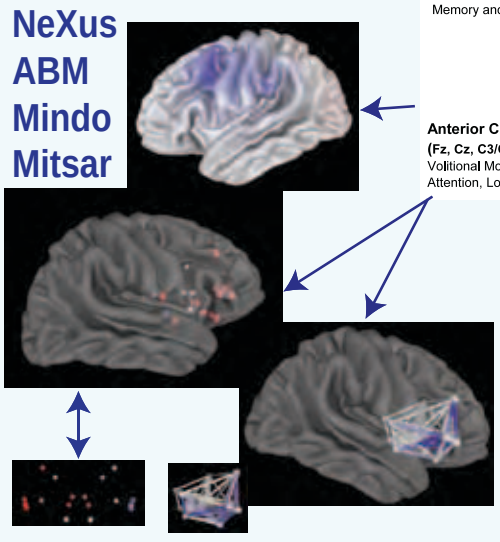

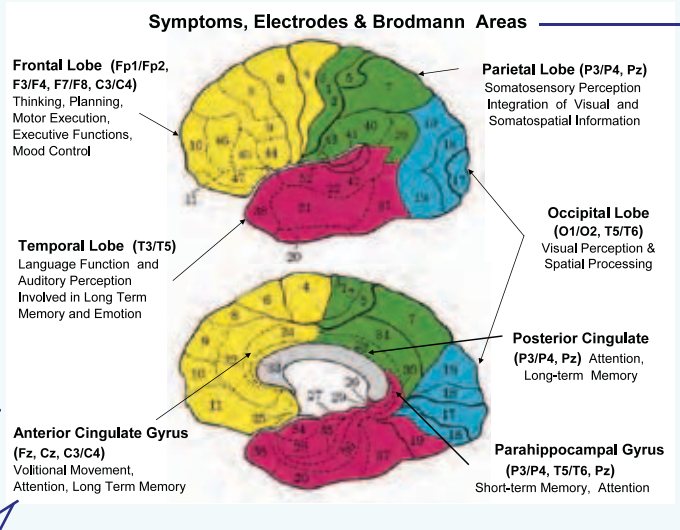

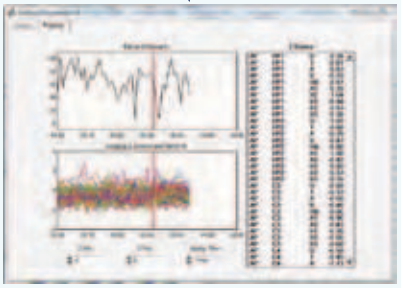

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NeuroConnections
ISNR Co-editor: Merlyn Hurd, PhD
merlynh@nyneurofeedback.com
AAPB Neurofeedback Section Co-editor: Roger H. Riss, PsyD
rriss@madonna.org
Managing Editor: Barbara Trumbo
ncmanagingeditor@gmail.com
Journalist for MindFull: David Kaiser, PhD
davidkaiser@yahoo.com
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Direct all correspondence and inquiries, including commercial advertising information and classified ads to:

NeuroConnections c/o
International Society for Neurofeedback and Research (ISNR)
1350 Beverly Road
Suite 115, PMB 114McLean, VA 22101-3633
Office: (703) 848-1994
Fax: (703) 738-7341
www.isnr.org
cyablonski@isnr.org

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NEURO CONNECTIONS

A joint newsletter from the ISNR & the aapb Neurofeedback Section

Contents

Letter from AAPB NFB Section Past President

Richard Soutar, PhD

The Two-Party System

Over the past year there was considerable interest among many members of the AAPB Neurofeedback Section and the ISNR to merge the two organizations together. Among many reasons cited were reduced costs for vendors, fewer organization fees for members and less yearly travel expense. A more general sentiment was that it would promote a greater sense of unity and enhance the focus of the two groups by combining their resources. In fact, a great number of major vendors signed a petition to integrate the meetings.

There was very active discussion by the leadership and heated debate on the list serves. We circulated a petition on all the major list serves to test the waters and see how strong the sentiment actually was in terms of numbers. Around 10% of the combined membership of both organizations rallied enough passion and enthusiasm to sign it. Perhaps we could have done a better job promoting the idea but we were really interested in seeing the actual level of commitment rather than marketing an exciting concept. Based on the response in the list serves, it appears that a great number of people considered the idea. I have no doubt that the idea will continue to generate excitement among sections of the membership in the future. This may just be an artifact of a two-party system. The actual task of combining two organizations such as AAPB and ISNR is daunting from both a personnel perspective and a legal perspective. There is more to it than I think many of the members realize, unless they have had the opportunity to actively serve on the boards.

There are, however, advantages to the two-party system as well. It provides more venues and if one organization's meeting is too far away to attend, the other might be closer. The two organizations have different atmospheres and a different emphasis on technology and theory. There are some very interesting workshops at AAPB that you would never find at ISNR and vice versa. This variety is important to the field's growth and development. The competition for the attention of the NFB audience helps to make both sides more responsive to membership interests. So, maybe we will all do fine with the two-party system we have generated. Variety, as they say, is often the spice of life.

Last year's AAPB meeting was one of the best I have attended to date. There was a lot of enthusiasm and an open mindedness that brought in a fascinating group of speakers and very innovative workshops. The meeting format was a refreshing change as well. It was a lot of fun to pay one fee and be able to freely move among workshops and choose as many as you like. Many of us felt like kids in a candy store. The NFB section meeting was quite well attended as well. This year the meeting was in my own back yard in Savanna, Georgia and it was an easy choice to attend, but I have always enjoyed the atmosphere of the AAPB meetings and the members who create it.

The actual task of combining two organizations such as AAPB and ISNR is daunting from both a personnel perspective and a legal perspective.

Richard Soutar, PhD, BCN

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 Section 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 section is organized for the purpose of carrying on educational and scientific objectives and is not to be operated for profit.

Letter from ISNR President

Rex Cannon, PhD

Greetings fellow ISNR members,

Your ISNR Board has envisioned a new direction for ISNR and many additions to our organization are in process and will continue over the next few years. I will briefly touch upon these items. President-Elect Robert Coben has spearheaded the ISNR education program (ISNR-U) which will begin providing educational programs for our members including webinars, workshops, instructional courses, and other educational resources for our members. This type of educational service falls under our mission statement, yet has not been fulfilled in adequate fashion until now. The education committee will be unveiling its program in the near future and at the 2014 conference. The new journal for ISNR, Neuroregulation, is accepting papers for its inaugural issue. These can include interesting clinical case studies, research, and review and perspective articles. Thanks to the Board and Past President Randall Lyle for the dedicated work on this project. The Board and conference committee have elected to revamp the conference for a more structured format. This year's conference will have a theme related to the brain and its functional connectivity, and most workshops, presentations and plenary sessions will utilize this particular theme or have data that relate to this theme. This will provide our members, as well as attending professional guests with a great impression of ISNR. We want all conference attend-

The education committee will be unveiling its program in the near future and at the 2014 conference.

Call for Papers & Posters

Consider sharing your knowledge and experience with your fellow Technologists at the **2014 ASET Annual Conference**. Put together an interesting case presentation, procedural update, interesting experience or even your research results. This is a great opportunity to expand your professional experience, develop presentation skills and network with other professionals in the field.

Benefits of Presenting:

- Complimentary meeting registration for the day of your presentation
- Astract will be published in *The Neurodiagnostic Journal*
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Two Ways to Submit:

1. Complete the form online at **www.aset.org** and attach your abstract.
2. Download the form at **www.aset.org** and fax or mail to Faye Mc Nall, ASET Director of Education. Then email your abstract to faye@aset.org.

Deadline: March 15, 2014

For specific questions and information contact:
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ees to have the best educational experience, emphasizing the brain, EEG, functional connectivity, empirical validity, rigorous data and hypotheses, and the most effective way to use neurofeedback and applied neuroscience in their practice.

Your Board would like to express much gratitude to the editorial staff of NeuroConnections. We are in the process of moving to an online only format. We do this for two important reasons. First, since we have created a growing social media presence, we can post links to NC and professionals as well as the public can gain access to research and clinical work related to neurofeedback and diagnostic techniques. As a society we must confirm our position as the premier organization for neurofeedback, neuroregulation, self-regulation, quantitative EEG (qEEG) and applied neurosciences. If you are interested in our social media presence, see our linked-in and twitter accounts (ISNRORG). We will be working on our Facebook presence next!

It is also with great delight that I offer compliments and gratitude to your current Board. There are often growing pains associated with change, and discomfort and resistance to novel ideas. This board has been courageous in forward thinking and your current executive director is a champion for ISNR. Cindy Yablonski is a great asset to our organization—thank her when you have a chance.

I am still excited about the future of ISNR and the exponential increase in interest for neurofeedback by the public and other professional organizations. We can meet all challenges and overcome all obstacles. Our members are bright lights in a dark cavern, offering hope where all else has failed. We are ISNR!

Rex Cannon, PhD

Letter from AAPB NFB Section President

Cynthia Kerson, PhD

Dear *NeuroConnections* reader,

I'm looking forward to the coming year and my term as president of the AAPB Neurofeedback Section. Let me introduce myself: in addition to being the section's president this year, I also hold a few other positions: I am the ED for the ISNR Research Foundation (ISNR RF), a member at large of the AAPB Board of directors, an adjunct professor at the Dept. of Psychology at Saybrook University and the Director of Education for BSI (Brain Science Internation-

al). I also have a small clinical practice in Northern California.

Were you at the meeting in Savannah? If so, I would like to explain in more detail a matter that should be of interest to all section members. What? You're not an AAPB Neurofeedback Section member? This is a great time to join and help us with the following issues.

During the AAPB meeting ISNR RF and FERB (Foundation for the Education and Research of Biofeedback and Related Sciences—the AAPB research foundation) awarded their very first jointly funded grant. The applications required the inclusion of both biofeedback and neurofeedback, and there were some very good ones; it was hard for the reviewers to decide. However, Matthew Goodman was awarded $3,000 ($1,500 from each foundation) for his project at Alliant University, which utilizes biofeedback and neurofeedback with autistic children. My congratulations go to him and his advisors, Drs. Dick Gevirtz and Jaime Pineda. The ISNR RF and FERB Board agreed to award a jointly funded grant annually. Richard Soutar of New Mind Center donated $4,000 ($2,000 to each foundation) to fund the 2015 grant. Thank you, Richard for your generous contribution.

It's an exciting time for research in our field. Both organizations intend to award over $15,000 during the coming year. Researchers in both disciplines (biofeedback and neurofeedback) are gaining the financial support they need to move forward. As NeuroConnections readers, I assume you have interest in neurofeedback research. So look for these announcements and let your colleagues know about them.

During the Neurofeedback Section meeting, I attempted to explain that FERB was holding approximately $8,100 that the NFB Section raised over 10 years ago. FERB is surviving on a shoestring budget, and asked that we release the funds to their general fund so that they may stay within their budget. (It really is a bookkeeping mechanism, rather than an actual handing over of funds.) Seems reasonable enough. However, I would have to get a majority vote from the members of the NFB Section to do so. Before that, though, I ask if you have another suggestion about how those funds should be managed. For example we could require that they be used for funding of some other sort. If you are a NFB Section member and have an idea, please let me know. Understand that the funds belong to FERB, so any decision would be how FERB may use them.

Richard Soutar, our past president, and I announced that the NFB Section has approximately $11,000 in our account from years of dues that we have not spent. This is money that belongs to the section, so it is not limited to research within FERB. Neurofeedback Section members decide what to do with these funds. I recently sent an email to them and have received the following suggestions:

- A named grant to FERB for funding research.
- A named grant to the ISNR Research Foundation for funding research.
- Invest it.
- Use it to fundraise further.
- Use it for student scholarships.
- Use it to sponsor NFB Section members.

If you are a member, please look for a survey in May. If you aren't, this is a good time to join and help us shape the future of the section and our field.

Regards,

Cynthia Kerson, PhD, QEEGD, BCN, BCB

Letter from AAPB Editor

Roger Riss, PsyD

Welcome to the Spring 2014 issue of *NeuroConnections*. With this issue we launch our new web-only distribution format, in support the Board's vision of *NeuroConnections'* potential, via social media and expanded web presence, to reach out to an audience well beyond ISNR and AAPB membership. A special shout out is due to managing editor Barbara Trumbo, without whose contributions this transition would not have been possible.

In coming months, you can expect to see our web presence, and the appearance of *NeuroConnections* continuing to evolve, as we take fuller advantage of web capabilities. We are very grateful for the support of the ISNR and AAPB neurofeedback section boards, our members, and our advertisers for their continued support during this transition, and welcome your feedback regarding how we can make the transition to web-based distribution even better. Your comments are welcome at NCManagingEditor@gmail.com

We hope that you enjoy the current issue as much as we have enjoyed working with the authors in its preparation. While repetitive transcranial magnetic stimulation (rTMS) has now received FDA approval for use in treatment-resistant depression, Hasan Asif, MD, offers case history data indicating that the efficiency of the rTMS intervention can be substantially enhanced via use of qEEG to guide treatment staging, paired with concurrent neurofeedback to address

patient-specific qEEG deviancies which are not addressed by standard rTMS treatment protocols. This combined intervention is a first in the literature and a compelling read.

Lucas Koberda, MD, adds to his recent series of papers and presentations re clinical efficacy of LORETA Neurofeedback, with case series data in treatment of depression and anxiety. Treatment of ADHD is hampered by reliance on behavioral diagnostic criteria, blurring the distinction between distinct phenotypes of the disorder, each requiring individualized medication and neuromodulation approaches. This is the topic of original research by Ron Swatzyna, PhD, in the current issue. Merlyn Hurd presents a related case history featuring prediction of treatment response via analysis of qEEG phenotypes. The Israeli team of Rivi Sela and Meirav Shaked-Toledano updates the extensive literature on neurofeedback in seizure control, with a very high-caliber case study demonstrating use of qEEG and LORETA-guided seizure control in children with treatment-resistant epilepsy. Cory Feinberg, MA, debuts in his first contribution to *NeuroConnections* with an extremely well written case history demonstrating the capacity of neurofeedback-based brain regulation to assist patients seemingly mired in therapeutic impasse and entrenched psychopathology. Lastly, Paul Swingle and Tom Collura illustrate use of qEEG-guided treatment planning from two very distinctive, yet powerful traditions. Despite their differences, their papers echo a common theme: that effective use of qEEG-guided training must go beyond a simple "training to average" approach to include a nuanced understanding of the individual patient, the clinical significance of qEEG neuro-markers, and technical factors informing interpretation of data base patterns.

Lastly, with this issue, we bid farewell and a well-deserved thank you to Richard Soutar, PhD, who is completing his very successful term as AAPB Neurofeedback Section Board President. Richard, it has been a pleasure to work with you. It is with great anticipation that we welcome incoming AAPB Neurofeedback Section Board President Cindy Kerson, PhD, who has played such an integral role in *NeuroConnections'* past successes.

Roger H. Riss, PsyD

The I in BCIA Really Does Mean International

Judy Crawford

BCIA changed its name in 2009, and since then, the international response has been very encouraging. The growth has come from a global request to offer different and better non-drug options in health care, an increasing body of research and science, and a need to quantify the knowledge base that supports the field.

Technology has been on our side. With the advances in secure online testing, easy translation of web pages, and distance education and clinical training resources, certification has seen a marked upswing in the number of professionals who are able to complete the certification requirements without geography as a barrier.

Certification by the Numbers

This year shows exciting promise for international training and certification. There are currently 28 countries with BCIA-certified providers. Live, face-to-face, BCIA-accredited, didactic training workshops are offered in ten countries. There are plans to extend training to more countries. BCIA exams will be held and proctored at four international meeting sites in the Netherlands, Italy, Australia, and South Africa, with more sites added as the need arises and as the arrangements can be made within the boundaries of scientific meetings.

Why Licensed/Credentialed Only?

BCIA does not have the ability to accurately assess international education, training, and health care standards. BCIA also lacks the ability to pursue an ethical complaint.

Licensure/credentialing/registration of health care professionals serves

Continued on page 47

Spring 2014 ISNR Research Foundation Update

The Foundation Thanks Our 2014 Grant Sponsors

First Funding Cycle | Grantees Announced at AAPB | Savannah, GA | Mar 19-22

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$2,000
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FERB and ISNR RF
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Renaissance Austin Hotel

Austin, Texas

MARCH 11-14, 2015

(during SXSW 2015!)

Austin is proudly weird, proudly Texan (it is the capitol after all). Austin is home to the University of Texas, the state capitol, famed music clubs on Sixth Street, great restaurants and world-class spa retreats, AND the SXSW- South by Southwest Interactive Film & Music Festival which happens to coincide with the AAPB 2015 Meeting. With entertainment and culture, inspiring cuisine and stunning outdoor settings, Austin lets you create a soundtrack all your own. Austin boasts more than 250 music venues and a vibrant arts scene. Make plans to join us for the AAPB 2015 Annual Scientific Meeting: The Many Faces of Biofeedback and put Live Music Capital of the World® on your playlist.

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Optimizing Treatment Efficacy and Success Rate for RTMS with qEEG Analysis and Neurofeedback

Hasan Asif, MD, Zoe Simmons, BS

Abstract

In our clinical applications of repetitive Trans-Cranial Magnetic Stimulation (rTMS) we have increased treatment efficacy via the added use of qEEG and neurofeedback. Pre-session qEEG analysis provides a baseline measurement, which is vital in informing our decisions for treatment protocols. Post-session qEEG recordings unveil protocol efficacy. Furthermore, the acquisition of pre- and post-treatment qEEGs has allowed us to quantitatively interpret the positive anecdotal evidence we have acquired in reference to combination rTMS and neurofeedback therapy.

The following report includes a series of case studies in which we provide support for: *(1)* the vital use of qEEG in rTMS therapy, and *(2)* the subjective and quantified benefit of combining rTMS with neurofeedback therapy. In our first study, daily qEEG analysis provided support for our decision to use rTMS to deactivate the right DLPFC during the first 6 days of treatment and then to activate the left DLPFC during the last 4 days of treatment. In combination with rTMS, we administered neurofeedback sessions and were successful in providing complete remission in only 10 days. Our second case study provides subjective and quantitative support for combining rTMS with neurofeedback rather than the singular use of rTMS. In combining rTMS and neurofeedback therapy, we show greater quantifiable and subjective improvements within a five-session combined treatment than in a 25-session rTMS treatment.

Case Study 1

Introduction

The commonly administered rTMS procedure for depressed patients involves activation of the left DLPFC for at least 20 consecutive sessions. Recent literature, however, has urged the use and the quantitative understanding of alternative rTMS coil placements (Downar & Daskalakis 2013).

In our own clinical investigations, frequent monitoring of pre- and post-rTMS sessions via qEEG has provided us with quantitative evidence that in some cases, the source of a regressed affective state is not the under-activation of left prefrontal cortical activity. Alternatively, activation of the right prefrontal cortical regions may result in anxiety and rumination especially around the issues of attachment loss. Normal regulation of negative emotions evoked by negative visual stimuli has been shown to be associated with an inhibitory effect on the amygdala provided by the prefrontal cortical area. More specifically, activation of the left DLPFC may be a direct cause of the ability of non-depression-prone individuals to inhibit fear and anxiety when exposed to negative visual stimuli. Unlike non-depression-prone individuals, depression-prone individuals show right DLPFC activation when asked to dampen their negative emotional response to the same negative visual stimuli. The conclusion can be made that right DLPFC activation in depression-prone individuals is directly linked to the inability to willingly inhibit rumination and anxiety when exposed to negative stimuli (Johnstone et al., 2007). Therefore, rTMS protocols should certainly not be limited to conventional left DLPFC activation. Furthermore, patient-specific prefrontal brain wave activity should be determined before commencing with an rTMS protocol. In this case study we expose an rTMS treatment in which the use of qEEG was critical.

The acquisition of pre- and post-treatment qEEGs has allowed us to quantitatively interpret the positive anecdotal evidence we have acquired in reference to combination rTMS and neurofeedback therapy.

The patient, a married white female, 60 years old, sought psychiatric attention after being hospitalized for attempted suicide. Prior to the attempt, the patient was able to maintain remission of her depression while on four medications: Effexor 300mg/day, Wellbutrin XL 300mg/day, Buspar 45mg/day and Lithium 300mg/day. However, upon the approach of the anniversary of her friend's death, the patient began to constantly ruminate about her loss. Rumination evoked a suppressed feeling of hopelessness, which progressively worsened. The patient became irritable at work and started experienc-

ing difficulties in getting along with her employers.

The patient's initial qEEG showed an imbalance of frontal lobe HiBeta which exceeded 3 standard deviations in the right DLPFC; therefore, to regulate DLPFC activation, we began the patient's treatment by targeting a 1Hz rTMS pulse between F4 and F8. Eventually, the patient's subjective and quantitative presentation prompted us to stop right DLPFC deactivation and begin left DLPFC activation. We also chose to add daily neurofeedback sessions, (post-rTMS), during the second half of her therapy. In just 10 total sessions of therapy, the patient reported complete remission, which is also evidenced in her PHQ-9 self-rating scale.

Methods

Over the course of 10 consecutive weekdays, the combination of rTMS and neurofeedback treatment was administered. Daily qEEG measurements allowed us to monitor our patient's most current brain activity in order to adjust the rTMS and neurofeedback protocols accordingly. Our neurofeedback protocols provided z-score training inclusive of absolute power, relative power, coherence, phase, and asymmetry of all bands, but specific to several electrode channels. Additionally, we created separate events with the intention of reducing right DLPFC HiBeta activity. The patient provided frequent subjective assessments in the form of anecdote and the Patient Health Questionnaire 9-question version (PHQ-9) self-rating scale.

Procedure

The quantitative objective of therapy aimed to decrease the high amplitude deviation of HiBeta in the right DLPFC while maintaining normal activity in the left DLPFC. Therefore, activity read specifically by the F4 and F3 electrode was monitored closely. Daily qEEG readings correlated with the patient's daily subjective assessments prompted us to alter protocol accordingly.

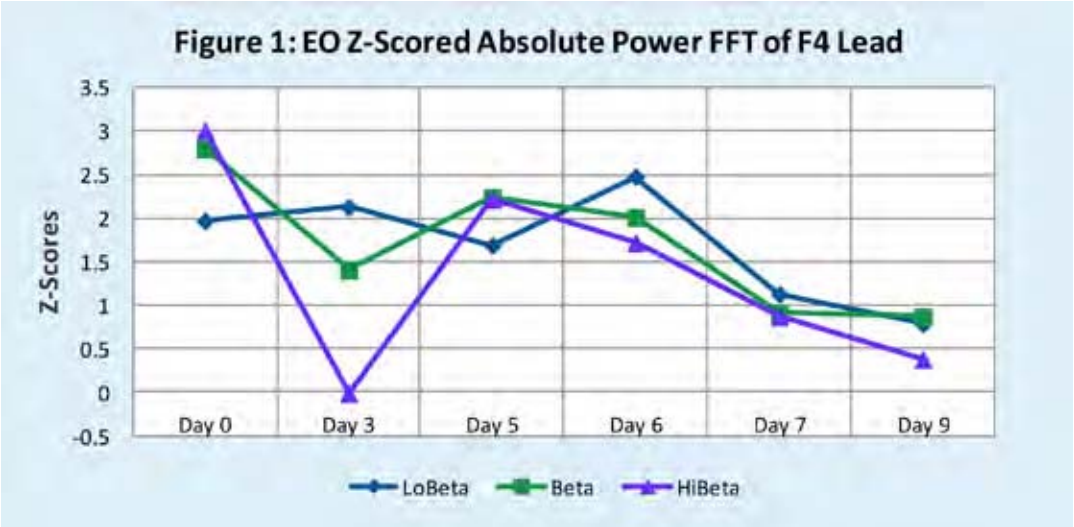
Results

Over the course of treatment, the patient's LoBeta (12–16Hz), Beta (16–20Hz), and HiBeta (20–28Hz) Eyes Open Z-Scored Fast Fourier Transformed (FFT) absolute power deviations show an overall decline despite a spike in Beta and HiBeta activity on day 5 and a spike in LoBeta activity on day 6 (see figure 1). Figure 2 shows a spike in deviation of the Theta and Delta ranges on day 5 of treatment and a subsequent decline in deviation through the rest of treatment. The Beta, HiBeta, Delta, and Theta spike on day 5 as well as the LoBeta spike on day 6 were a vital finding, which altered the course of our treatment and is further elaborated upon in the discussion. The patient's subjective state measurements assed by the PHQ-9 self-rating scale show steady decline (figure 3).

Discussion

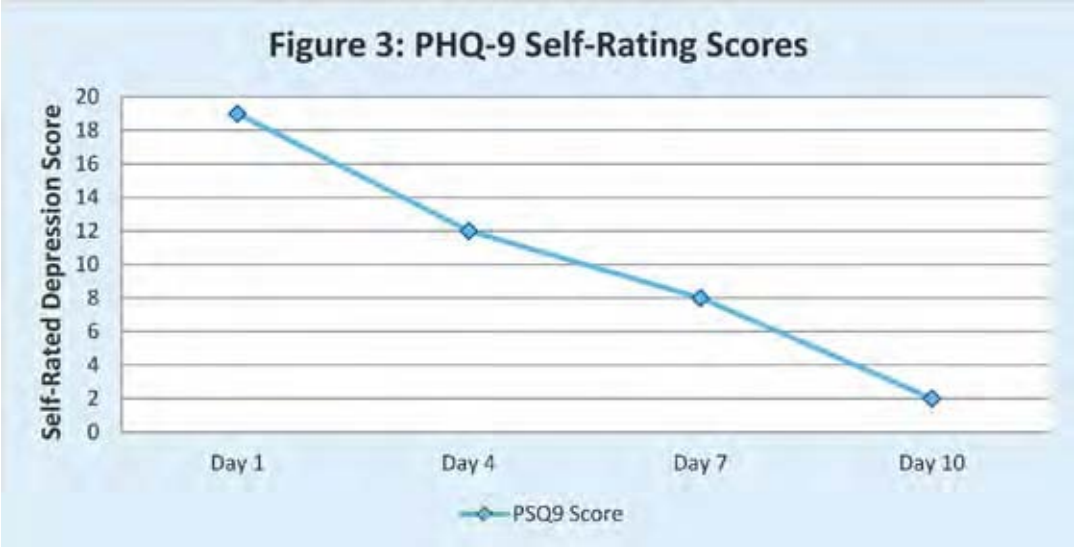
The above-mentioned case study is an example of a potential method of increasing efficacy of rTMS with fewer

Day 1	rTMS was administered at the RPFC for 1pps in 1-second intervals, (2200 pulses total).	
Day 2	rTMS was administered at the RPFC for 1pps in 1-second intervals, (2200 pulses total).	
Day 3	rTMS was administered at the RPFC for 1pps in 1-second intervals, (2200 pulses total).	
Day 4	rTMS was administered at the RPFC for 1pps in 1-second intervals, (2200 pulses total).	
Day 5	Z-Scored neurofeedback was administered via F3, F4, C3, C4, T3, T4, and Cz.	
Weekend Break		
Day 6	rTMS was administered at the RPFC for 1pps in 1-second intervals, (2200 pulses total).	Z-Scored neurofeedback was administered via F3, F4, C3, C4, T3, T4, and Cz.
Day 7	rTMS was administered at the LPFC for 10 pps in 4-second intervals, (3000 pulses total).	Z-Scored neurofeedback was administered via F3, F4, C3, C4, T3, T4, and Cz.
Day 8	rTMS was administered at the LPFC for 10 pps in 4-second intervals, (3000 pulses total).	Z-Scored neurofeedback was administered via F3, F4, C3, C4, and Cz.
Day 9	rTMS was administered at the LPFC for 10 pps in 4-second intervals, (3000 pulses total).	Z-Scored neurofeedback was administered via F3, F4, C3, C4, and Cz.
Day 10	rTMS was administered at the LPFC for 10 pps in 4-second intervals, (3000 pulses total).	Z-Scored neurofeedback was administered via F3, F4, C3, C4, and Cz.



Eyes Open LoBeta (12–16Hz), Beta (16–20Hz), and HiBeta (20–28Hz) z-scores recorded by the F4 lead throughout treatment.

Eyes Open Delta (1–4Hz) and Theta (4–7Hz) z-scores recorded by the F3 lead throughout treatment.



Trend of PHQ-9 Depression Self-rating Scale throughout treatment.

TMS treatment sessions, with the help of quantitative EEG analysis guiding us in coil placement. Furthermore, use of Z-score neurofeedback along with region of interest (ROI) training right after the rTMS session was noted to be a potential adjunct tool in rTMS treatment. Starting from day 1 of treatment, the decision regarding coil placement was helped by qEEG analysis in which Beta2 activity was found to be over 3 standard deviations in lead F4, corresponding to an overactive right prefrontal cortical area. Z-score neurofeedback sessions were further supplemented with protocol to lower HiBeta activity in the F4 lead right after rTMS sessions. The patient's subjective state and improvement of affect directly corresponded with decline in HiBeta activity in F4 lead. This correspondence was further confirmed with a slight rebound of Beta activity the day after patient missed the session (see graph above). The patient felt back to her initial improvement level right after the session of day 6. Change of coil placement to the left side was implemented after noticing an increase in her left frontal delta and theta activity. In three remaining sessions rTMS was provided on the left side with activating 10Hz pulses along with neurofeedback training to activate left frontal area with resultant decrease in remaining subjective distress and decline in depression rating scale.

In the patient's case, daily transparency in both quantitative and subjective, neurophysiological and psychological states were crucial to her rapid and effective treatment. Had we been without qEEG assessments, we may not have changed the patient's right DLPFC activation protocol on day 7 of treatment, potentially allowing the left DLPFC Delta and Theta activity to increase. Upon continued increase of Delta and Theta activity in the left DLPFC, the patient may have been sent into a worse state of depression.

Case Study 2

Introduction

Repeated research has identified alpha wave desynchronization as a robust phenomenon among healthy individuals. Desynchronization occurs when, upon opening of the eyes, visually evoked alpha generation superimposes upon spontaneous alpha generation. The event of light adaptation decreases the amplitude of visually evoked alpha generation resulting in no alpha peak frequency recorded by the EEG (Kirschfeld 2005). Furthermore, increasing cortical activity when transitioning from eyes closed to eyes open has been shown to be associated with alpha wave desynchronization (Barry et al. 2007). The measured amplitude difference between alpha oscillations in the eyes closed and eyes open state indicates an individual's ability to desynchronize alpha waves (Bazanova & Vernon 2013).

Recent literature suggests that an individual's ability to desynchronize alpha can be improved with neurofeedback training. Moreover, Alpha desynchronization has the effect of increasing cortical excitability (Ros & Gruzelier, 2011). A topographically specific decrease in alpha oscillations (in the eyes open state) can also increase cortical excitability, which allows neurons to more easily elicit a response to an rTMS pulse (Sauseng et al., 2009). Therefore, we speculated that using neurofeedback to increase cortical activity in the eyes open state in addition to using rTMS would improve overall cortical excitability and overall treatment efficacy.

The subjective outcomes of past clinical attempts in which we administered neurofeedback therapy in combination with rTMS were substantially positive. Our acquired anecdotal evidence suggested that the combination treatment was not only more effective in potency but could be completed in fewer sessions than are commonly required for rTMS therapy alone. This case study unveils quantitative data that can be correlated with subjective anecdote regarding a five-session combined neurofeedback and rTMS therapy. The quantified and subjective differences made during the combined treatment therapy are also compared to the quantified and subjective differences made during a 25-session rTMS therapy preformed on the same patient, (6 months prior to the combined treatment).

The intent for the patient's initial rTMS treatment was decided on the bases that for the last 10 years, the patient, (an elderly, white, separated, male), had been treated for bipolar disorder in both inpatient and outpatient settings. His temperament had been predominantly depressed with one episode of mania in 2010, which later subsided after an addition of a mood stabilizer. In the winter of 2013 the patient presented with general anhedonia. In the last 2 years, our patient's psychomotor activity had been progressively declining such that he was almost confined to his bed. Prolonged physical confinement caused him to be socially isolated and with markedly exacerbated anhedonia. At 74 years old, the patient had significant muscle atrophy and, despite recommendation for physical therapy, refused to participate in his physical rehabilitation. The patient had been administered Vyvance 50mg/day, Pristiq 100mg/day, and Wellbutrin XL 300mg/day. His depression medications had been gradually increased and a stimulant was added, but despite the increase of medication to the maximum prescribed dosage, our patient continued to show signs of affective and physical deterioration. Therefore, in the winter of 2013, the patient and his family were

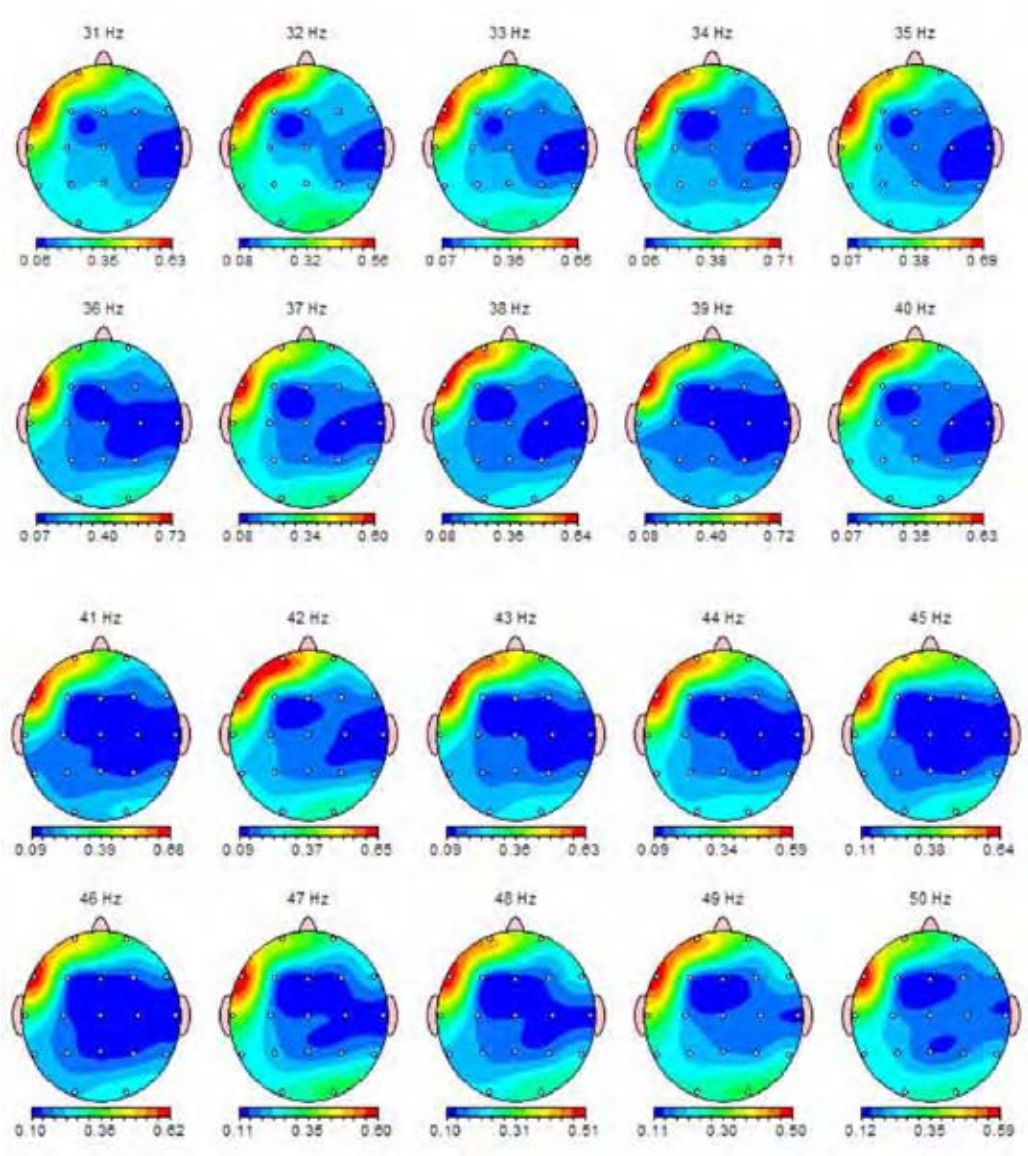
advised to consider a trial of rTMS for at least 25 sessions. At the start of his rTMS treatment, he was affected by extreme psychomotor retardation, bradyphrenia, abulia, and dulling of affect. The patient appeared notably depressed and anxious and ruminated about his family and their financial future. The efficacy of the patient's 25-session rTMS treatment had been subjectively described as only a 30% overall increase in mood affect.

Six months later, the patient and his daughters were back to seek aid in his emotional support as he recovered from hip surgery. The patient's qEEG assessment during this time did not differ much from his winter evaluation; therefore, we decided that in our patient's second round of therapy we would combine rTMS with neurofeedback. After only five sessions of the combined treatment, the patient claimed a 60% increase in mood affect. The quantitative results and interpretations of their associated neuropsychological effects between the 25-session rTMS treatment and the five-session combined treatment are included in this report.

Methods

QEEG was used to measure pre- and post-treatment brain wave activity for the 25-session rTMS treatment, which

Figure 4A: *Post-25-Session Absolute Power of Gamma Activity Measured in uV².*



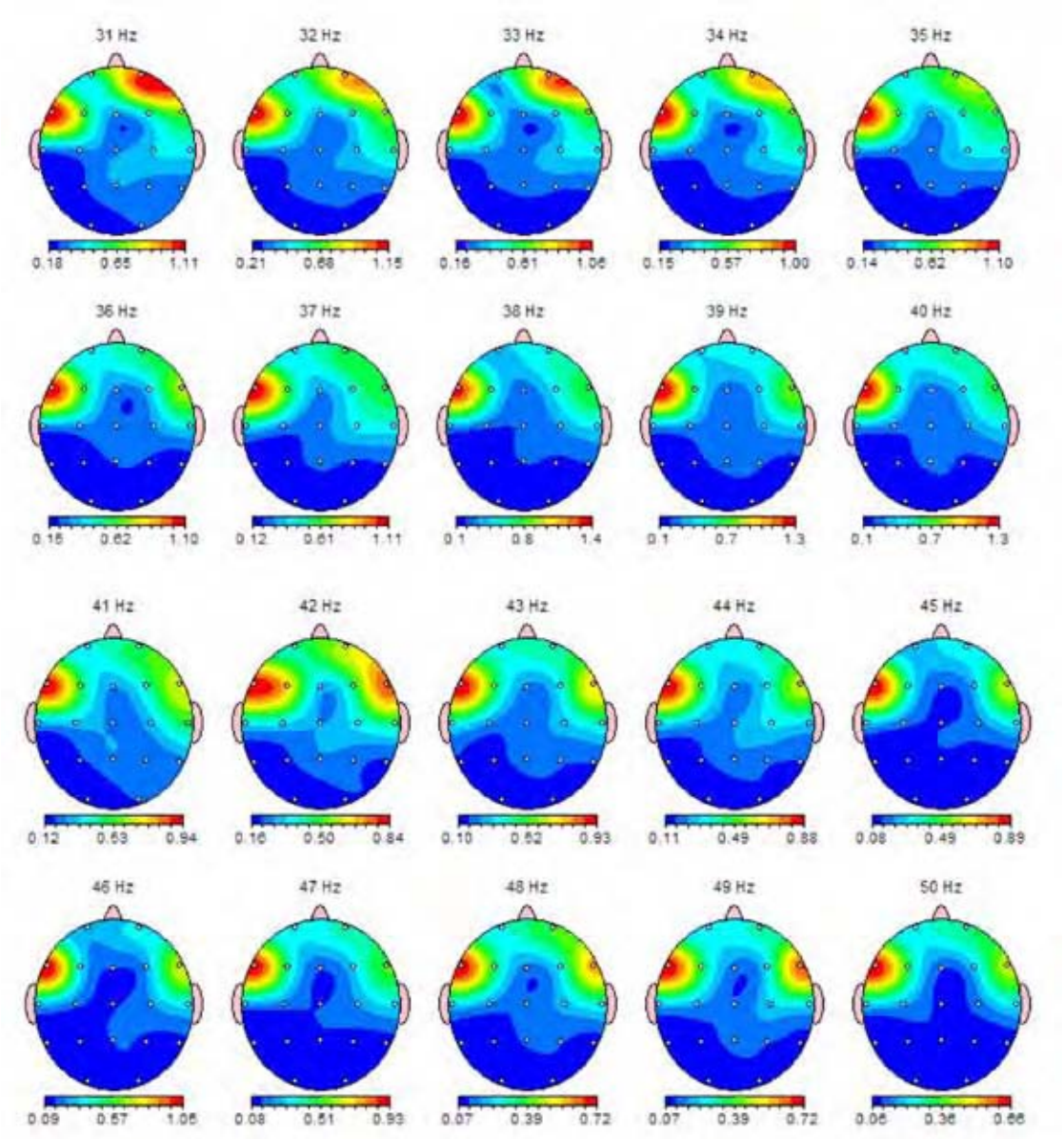
Post 25 sessions of rTMS therapy, the patient's Gamma activity has increased dominantly in the left DLPFC to a maximal value of 0.72uV² seen at 39Hz.

occurred between January and February of 2013. During the five-session combined neurofeedback and rTMS treatment in August of 2013, qEEG was used to assess each pre-rTMS, post-rTMS, and post-neurofeedback session. The administered neurofeedback protocols provided Z-score training inclusive of absolute power, relative power, coherence, phase, and asymmetry of all bands, all 19 channels. Additionally, we created separate events, which aimed to increase frontal Gamma, sensory motor Beta, and insular Alpha. The patient and his family provided their own subjective rating of efficacy.

Procedure

A 25-session rTMS treatment consisted of 10Hz activation, 10 pps in four-second intervals, (3000 pulses total), over

Figure 4B: *Post-five-session Absolute Power of Gamma Activity Measured in uV².*



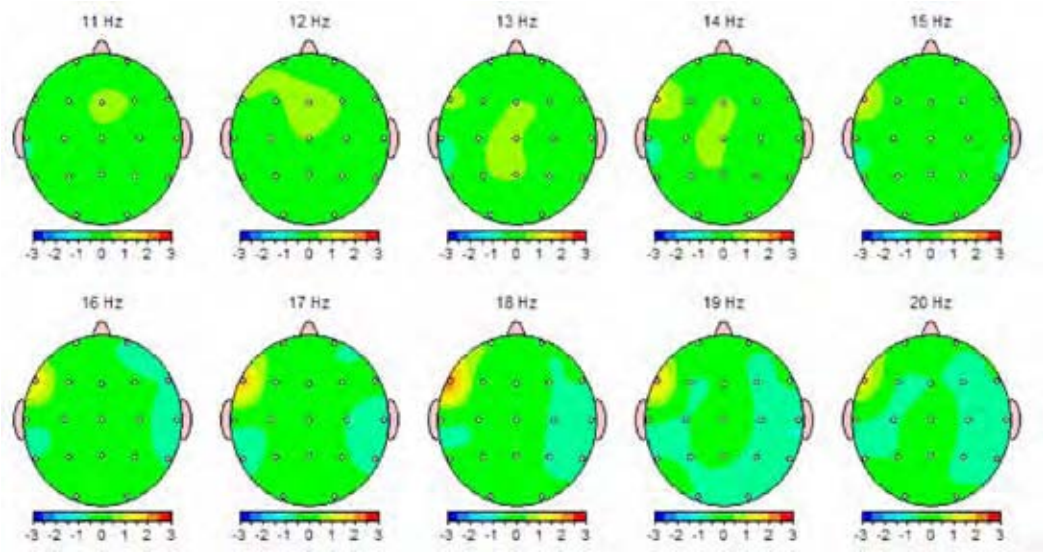
Post five sessions of combined neurofeedback and rTMS therapy, the patient's Gamma activity has increased in all areas of the pre-frontal cortex to a maximal value of 1.3uV² seen at 39–40Hz.

the left DLPFC. Six months post-25-session rTMS treatment a five-session combined neurofeedback and rTMS therapy was administered for five consecutive weekdays. rTMS was administered at the left DLPFC for 10 pps in four-second intervals, (3000 pulses total). Z-Scored neurofeedback was administered for four consecutive weekdays, succeeding each rTMS session via all 19 channels.

Results

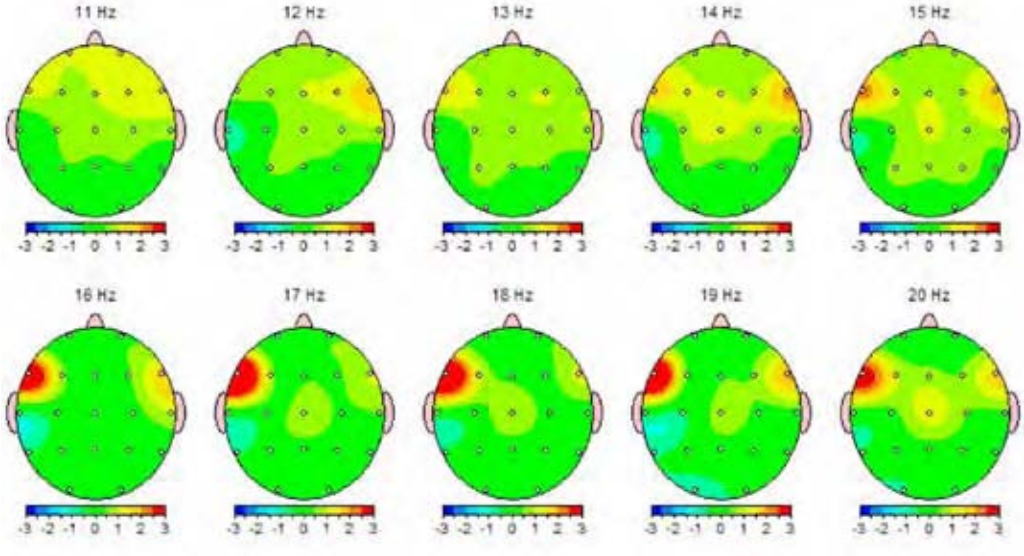
In examining the efficacy differences between the five-session combined treatment and the 25-session rTMS treatment, we will address the changes particular to

Figure 5A: *Post-25-Session Absolute Power of SMR and Beta Activity Measured in Z-Scored FFT Absolute Power (uV^2).*



Post 25-sessions of rTMS therapy, the patient's SMR and Beta activity appears to be +2 standard deviations in the left DLPFC. Beta activity appears to be -2 standard deviations over the sensory

Figure 5B: *Post five-session Absolute Power of SMR and Beta Activity Measured in Z-Scored FFT Absolute Power (uV^2).*



Post five-sessions of combined neurofeedback and rTMS therapy, the patient's SMR and Beta activity appears to be above +2 standard deviations and spread throughout the cortex. Beta activity appears to be at least +3 standard deviations in the left DLPFC

the frequencies we trained with neurofeedback: Gamma, Beta, and Alpha.

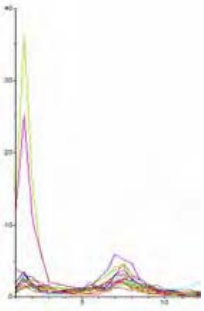
Figures 4A and 4B indicate the squared micro-voltage of the patient's Gamma activity post 25-session rTMS treatment and post-five-session combined treatment respectively.

Figures 5A and 5B indicate the z-scored absolute power of SMR and Beta frequencies post- 25-session rTMS treatment and post-five-session combined treatment respectively.

Assessing the difference in the patient's Alpha frequency generation, we were able to signify the changes in terms of: *(1)* The patient's measured individual alpha peak frequency (iAPF) measured pre- and post-25-session rTMS therapy and five-session combined treatment, see figures 6A, 6B, 6C, and 6D. *(2)* The difference in 4–12 Hz z-scored FFT absolute power (uV^2) measured pre- and post-25-session rTMS therapy and five-session combined treatment, see figures and 7A,

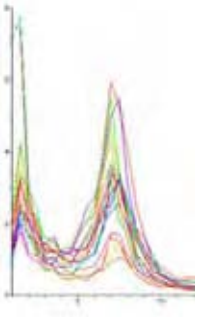
Figures 6 A-D: *iAPF Pre- and post-25-session rTMS therapy and five-session combined*

6A. EO Alpha Peak Frequency
Pre-25-Session TMS



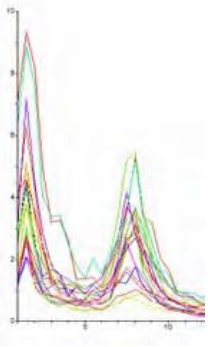
"Low alpha" peak frequency lies within 7–8Hz range.

6B. EO Alpha Peak Frequency
Post-25-Session TMS



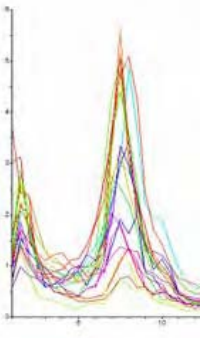
"Low alpha" peak frequency lies within 7–8Hz range.

6C. EO Alpha Peak Frequency
Pre-5-Session TMS



"Low alpha" peak frequency lies within 7–8Hz range.

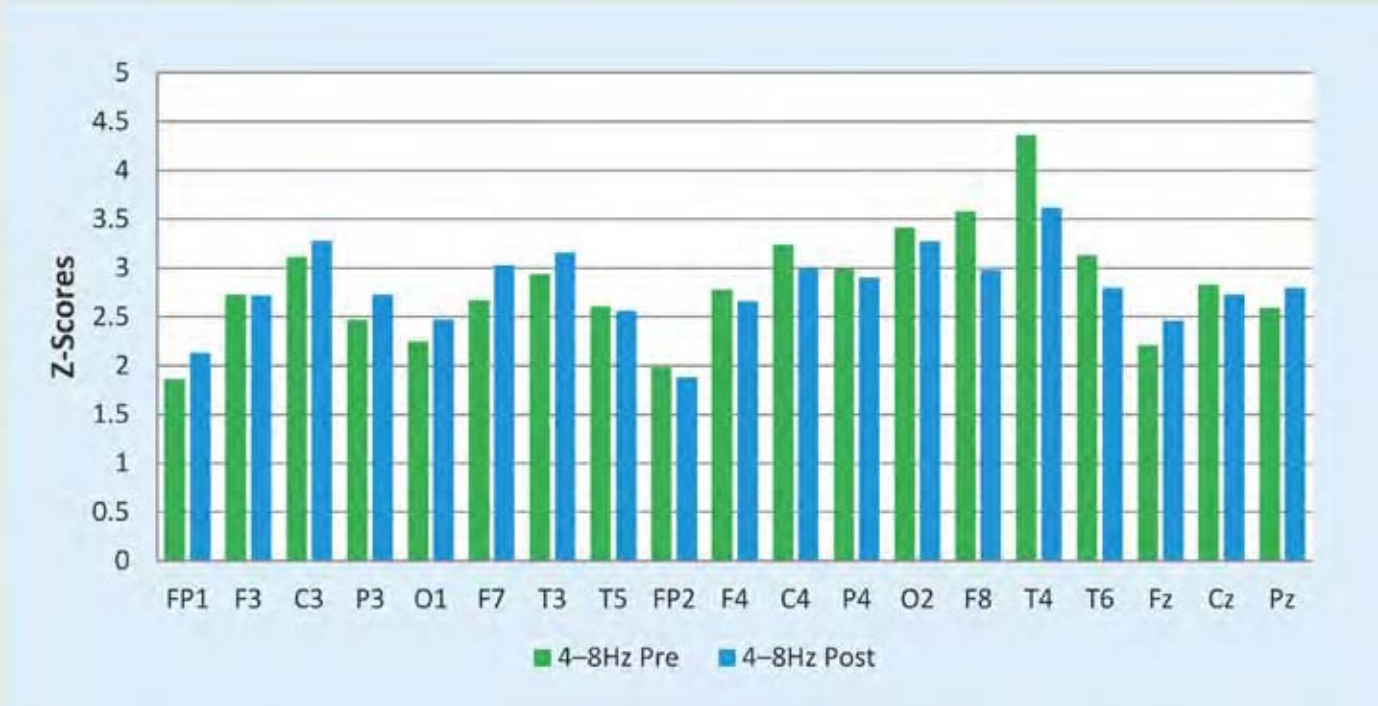
6D. EO Alpha Peak Frequency
Post-5-Session TMS



"Low alpha" peak frequency lies within 7–8Hz range and 10Hz peak is forming.

The patient shows a persistently low (7–8Hz) iAPF until the end of his five-session combined treatment. The measurement of the patient's post-five-session combined treatment iAPF indicates the appearance of a 9–10Hz peak.

Figure 7A: *EO Z-Scored FFT Absolute Power for 4–8Hz Pre- and Post-25-Session Treatment*

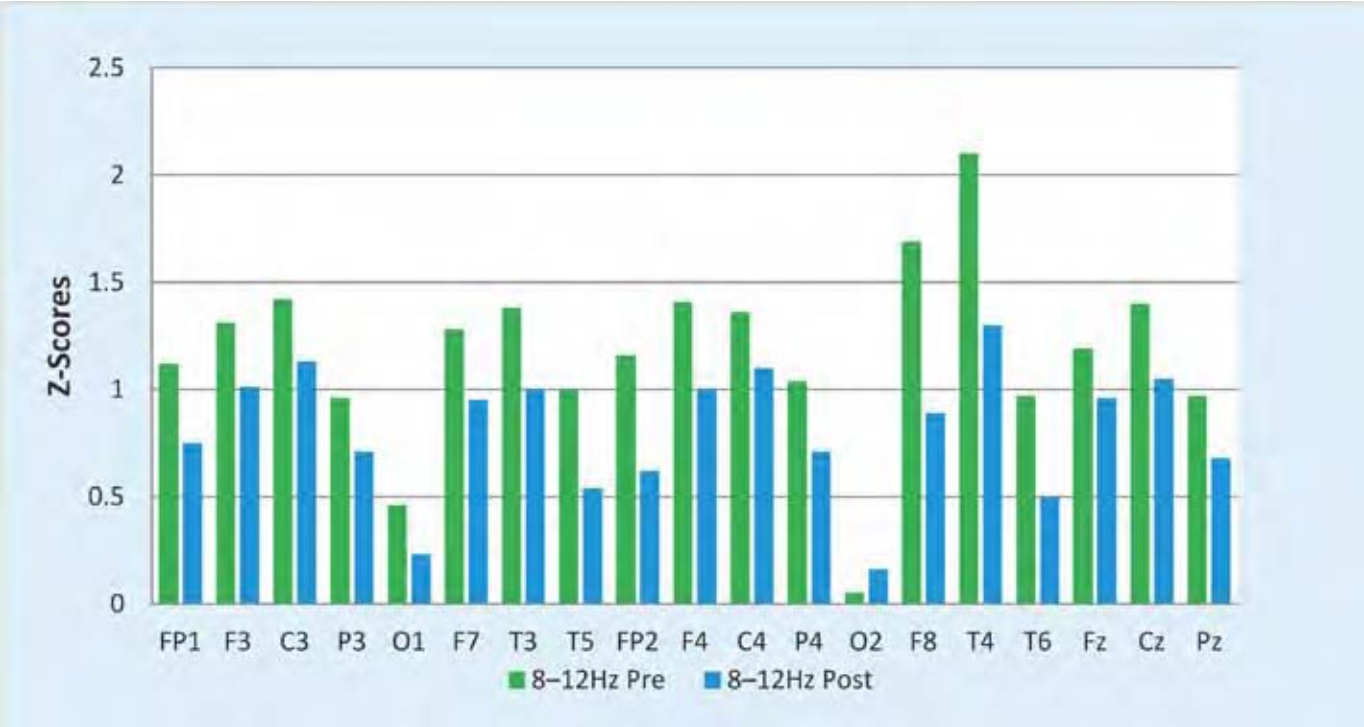


The patient's EO 4–8Hz Z-scores pre- and post-25-session rTMS therapy show no trend pre-or post-treatment in the negative or positive direction.

Figure 7B: *EO Z-Scored FFT Absolute Power for 4–8Hz Pre- and Post-5-Session Treatment*

The patient's EO 4–8Hz activity Z-scores pre-and post-five-session combined treatment show a general trend in the positive direction post treatment.

Figure 8A: *EO Z-Scored FFT Absolute Power for 8–12H Pre- and Post-25-Session Treatment*

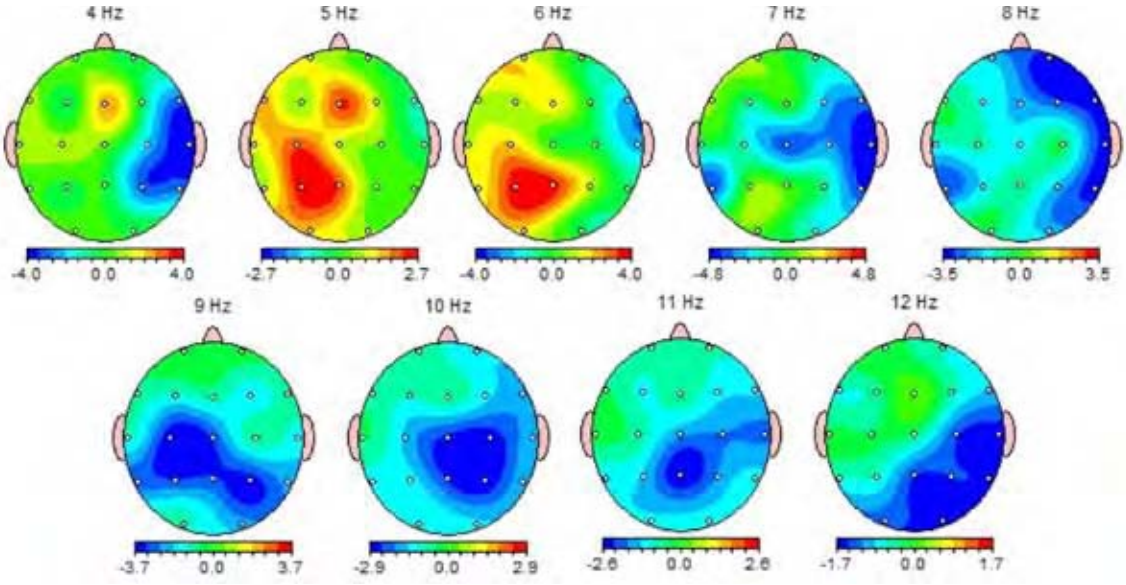


The patient's EO 8–12Hz activity Z-scores pre- and post-25-session rTMS therapy shows a trend post treatment in the negative direction.

Figure 8B: *EO Z-Scored FFT Absolute Power for 8–12Hz Pre- and Post 5-Session-Treatment*

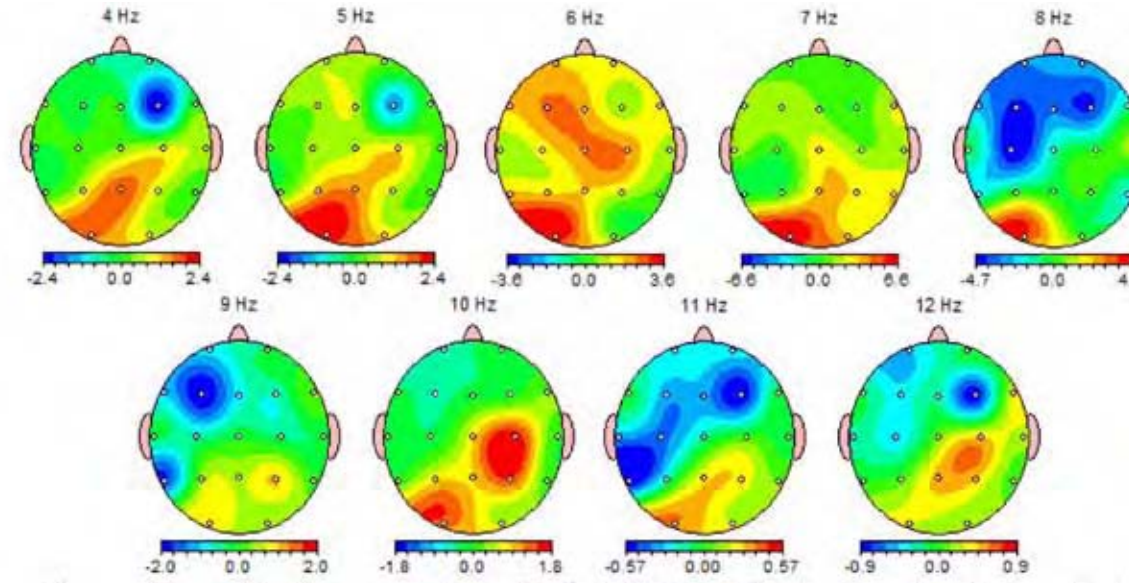
The patient's EO 8–12Hz activity Z-scores pre-and post-five-session combined treatment therapy shows a trend post treatment in the positive direction

Figure 9A: *EO post-25-session treatment/pre-25-session treatment difference measured in uV2 of 4–12Hz.*



Post-25-session rTMS, the patient's 4–6Hz has increased in the regions of P3 and Fz while his 7–12H activity has generally decreased.

Figure 9B: *EO Post-five-session treatment/pre-five-session treatment difference measured in uV2 for 4–12 Hz.*

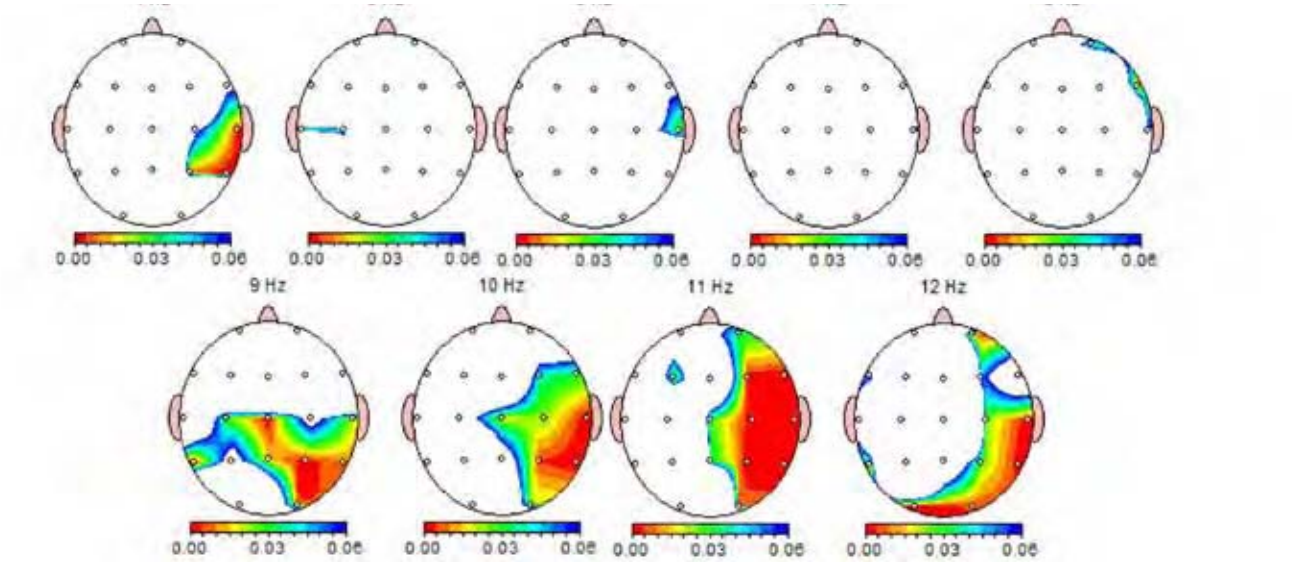


Post-five-session combined treatment, the patient's 4–12Hz activity shows a robust increase in the regions of O2 and C4.

7B, 8A, and 8B. *(3)* The FFT absolute power squared micro-voltage difference made pre- and post-25-session rTMS therapy and five-session combined treatment, see figures 9A, and 9B. *(4)* The statistical significance of the FFT absolute power squared micro-voltage difference made pre- and post-25-session rTMS therapy and five-session combined treatment, see figures 10A, and 10B.

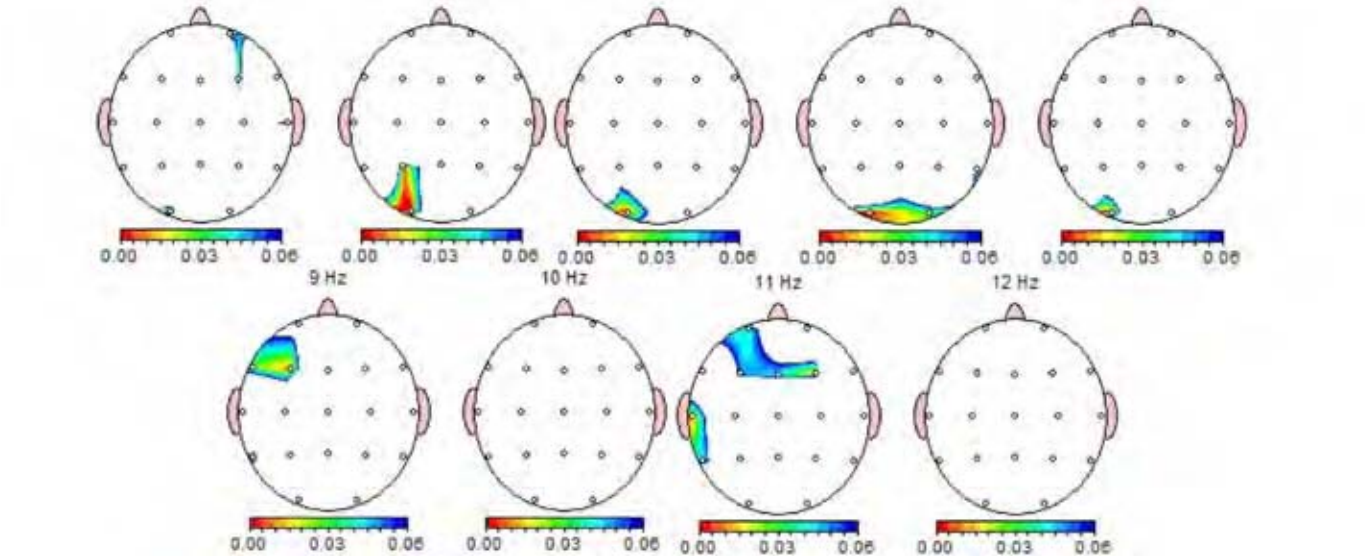
After the five-session treatment was 80%. Frank and his family related increased mood, increased conversational ability, increased gait, increased strength, and decreased overall stress.

Figure 10A: *Significance of change in EO FFT absolute power (uV2) post-25-session treatment/pre-25-session treatment*



The patient's 4–6Hz FFT absolute power increase in the regions of P3 and Fz shown in figure 9A has not been deemed significant. The 7-12Hz FFT absolute power decrease shows a P-value of 0.00 dominantly in the right hemisphere.

Figure 10B: *Significance of change in EO FFT absolute power (uV2) post-five-session treatment/pre-five-session treatment*



The patient's 5–8Hz FFt absolute power increase shows a p-value of 0.00 dominantly in the regions of O1. 9 and 11Hz have also shown a significant increase in the temporal and frontal areas.

Frank's subjective rating of efficacy post treatment was 20–30%. His family noted minor improvements in his gait, physical strength, and overall outlook. Frank claimed to ruminate less about his older age and the implications his passing may have on his family.

Discussion

Positive changes in the patient's overall affect became almost immediately apparent at the start of his combined treatment. With each day, his psychomotor activity became more fluid and his gait widened. His mood resonated a brighter affect such that by the end of the week his enthusiasm was contagious. The patient's expression of abulia had clearly diminished, and his rate of speech had caught up to wit. The patient's overall improved mood created an impressive energy, which was further supported

by the claims of his family. Quantitative results of the patient's combined treatment show changes to his Gamma, Beta, and Alpha frequencies.[1] In this discussion we will provide interpretations of our quantitative findings in relation to the patient's neuropsychology.

Increased Gamma activity was witnessed post five sessions of combined therapy, see figure 4B, and according to figure 4A, the result of the 25-session rTMS therapy did not have a similarly potent effect. Frontally induced Gamma wave activity has been understood to correlate with increased mood affect, heightened consciousness, attention, and improved sensory percept formation (Lutz et al. 2004, Jensen et al. 2007, and Castelhano et al. 2013). Therefore, we speculate that the patient's increased absolute power of frontal Gamma activity may be most directly associated to the patient's decreased expression of abulia, and his generally brightened affect. The minimal improvements made to affect induced by the singular rTMS treatment are no contestant to the rapid onset of the patient's overall improvement provided by the combined therapy. We postulate that increased treatment success rate as well as potency may be attributed to an increase in cortical excitability due to neurofeedback-induced Gamma activity.

Literature has shown the effects of increased SMR and Beta frequencies to correlate with increased preparation for motor execution (Baker, 2007, Wyrwicka & Sterman, 1968). Notable changes to the patient's improved psychomotor activity and gait may be attributed to the increased absolute power of SMR and Beta activity over the sensorimotor cortex, (figure 5B), an event not seen post 25 sessions. The inclusion of twice-weekly physical therapy sessions should also be recognized as an additional mode of the patient's overall surgical recovery plan. The patient's rapid improvement in physical strength may have resulted from a synchronistic effect of physical therapy and increased sensorimotor SMR and Beta frequencies induced by neurofeedback. During the 25 sessions of rTMS therapy, the patient always required assistance in order to position himself in the rTMS chair. However, during the last couple days of his combined treatment, the patient astonished us with his newly found energy and balance; he required no assistance.

At the conclusion of the patient's combined treatment, thorough data analysis of both treatments lead us to discover that what had initially appeared to us as large deviations in Theta activity was misinterpreted low iAPF. A known marker for low rTMS efficacy, low iAPF has been shown to inflict the elderly causing slowed processing speed, under-arousal, and memory deficits as well as general underperformance in storage, transfer, and retrieval of sensory information (Spronk et al., 2011, Grandy et al., 2013, and Anokhin & Vogel, 1996). The difference made to the patient's iAPF as a consequence of his combined treatment was a significant emergence of an increased iAPF; a difference not shown as a consequence of his 25-session rTMS treatment (figure 3). We deduce that the inclusion of higher iAPF frequencies is the cause for the shown increase in the patient's 4–12Hz z-score deviance despite no significant increase in the micro-voltage of his iAPF.

In the months succeeding the patient's completed therapy, we have found that the patient's improvements have been sustained. Although we propose that the combined method of therapy had improved the effect of consolidation and allowed for such a short duration of therapy, the possibility for relapse still remains unknown. However, the subjective improvements we have seen anecdotally and in qEEG from combining neurofeedback with rTMS have appeared to be much more substantial than the singular rTMS therapy we have administered. Therefore, we urge study of the neurological mechanisms that may be enhanced from the combination of treatments.

About the Author

Hasan Asif, MD, founder and Medical Director of the Brain Wellness Center, is a board-certified psychiatrist who has been in private practice for over 20 years.

A 1990 graduate of A.I.M.C in Pakistan, Dr. Asif finished postgraduate training in Psychiatry at New York Medical College in Valhalla, New York in 1995. He joined the University of West Virginia as an assistant professor of Psychiatry and later on worked as a Medical Director of adult partial Hospital in Four Winds Hospital in Katonah, NY. Currently, he is a consulting psychiatrist at Lawrence Hospital Center in Bronxville, New York. His private practice at Brainwellness Center provides TMS, S-Loreta neurofeedback, neurostimulation and med management based on Quantitative EEG analysis.

Zoe Simmons, BS, is a graduate of the State University of New York at Stony Brook, where she earned a Bachelor of Science in Biology with a concentration in Neuroscience. As a PhD applicant, she as-

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1. Due to muscle inflicted EEG acquisition during the patient's first evaluation we are limited in our ability to compare and discuss changes made above 12Hz as a result of the 25-session rTMS therapy. The patient's initial EEG recording had been acquired with a tin-electrode cap, which proved to be difficult in acquiring clean cortical readings. However, the post 25-session EEG recording was captured with the same tin-electrode cap and this time a clean reading was acquired. For EEG acquisition of the pre-and post 5-session combined treatment, a newly acquired German electrode cap was used and provided us with exceptional readings.

Specifying and Developing References for Live Z-Score Neurofeedback

Thomas F. Collura, PhD, QEEG-D, BCN, LPC

Introduction

Live Z-Score neurofeedback training (LZT) has been in practice for close to 10 years, and has evolved considerably in that time (Collura et al, 2007; Collura, 2013). There is now a proliferation of methods that incorporate live Z-Scores for neurofeedback as well as for other purposes. One of the cornerstones of LZT is that there must be some reference as part of the system, which provides the basis for computing the live z-scores that are incorporated into the feedback process. As the field evolves, it is appropriate to ask what constitutes a useful reference for Live Z-Scores, and how a reference may be chosen or developed, with various priorities and concerns in mind. It will be shown here that there is a wide range of possible choices for LZT references, and that the field has only begun to explore how to develop and use references.

This article will discuss the choice of references for LZT training, as well as considerations with regard to the development of such references. For the purposes of qEEG assessment, it is generally accepted that references should be based upon a representative population of individuals, so that results put the client in the context of a particular group. While a normative sample is clearly important for qEEG assessment, when a reference database is to be used for LZT training, it is not clear that one can assume that a population of "normal" individuals constitutes an ideal reference. When working with individuals, it is more likely that the reference needs to reflect the individual profile of the client, as well as the particular goals of the intervention.

All LZT training takes advantage of the same fundamental equation:

$$z = \frac{x - \mu}{\sigma}$$

Or in more recognizable terms,

$$zscore = \frac{measurement - mean}{stdev}$$

Where z is the resulting z-score, x or "measurement" is the current sample value, "mu" or "mean" is the reference mean value ("target") and "sigma" or "stdev" is the standard deviation value in the reference table. So the z-score is no more complex than a number that tells you how far a measurement is from some target, in terms of the normal distribution. Although it is often assumed that the mean and standard deviation should represent a "normal" or "typical" population, this is not necessary in the definition of a z-score. These reference values might represent some "normal," "typical," or "desirable" values, but they might just as well represent some ideal, an individual, or someone in any particular state of self-regulation or dysregulation. The key point is that a reference for LZT neurofeedback consists ultimately of a set of means and standard deviations, and there is more than one way to arrive at values that will be valid, useful, and effective.

LZT training is accomplished in real time by computing instantaneous metrics, and then comparing them with some reference values and standard deviations. The simple choice of these two numbers completely determines the resulting z-scores. Generally, it is assumed that the reference mean and standard deviation are derived from some appropriate statistically representative sample. This is true, and is a requisite condition for the computation to have validity with respect to the intended sample. However, what constitutes a representative sample is open to interpretation. A statistical average and standard deviation from n individuals from a well-controlled sample is one way to derive a reference, and has until recently been the primary reference used not only for LZT but also for qEEG in general. However, as we shall see, a set of values from a chosen sample, or even from a single individual is also a valid source of reference data, and other methods, such as synthesizing values, or constructing references for specific purposes, is also possible.

There is more than one way to arrive at values that will be valid, useful, and effective.

Incorporating LZT References in biofeedback

The following block diagram shows the software design of a general-purpose Live Z-Score training system (figure 1). It provides several options for the selection (or development) of LZT references, as well as flexible feedback capability providing visual, auditory, vibrotactile, or electromagnetic feedback. Input data can include, in addition to z-scored values, convention qEEG metrics, Infra-Slow Fluctuations (ISF), and peripheral biofeedback modalities. One emphasis is to provide various options that can be combined or customized, rather than dictating a single, monolithic approach to LZT neurofeedback.

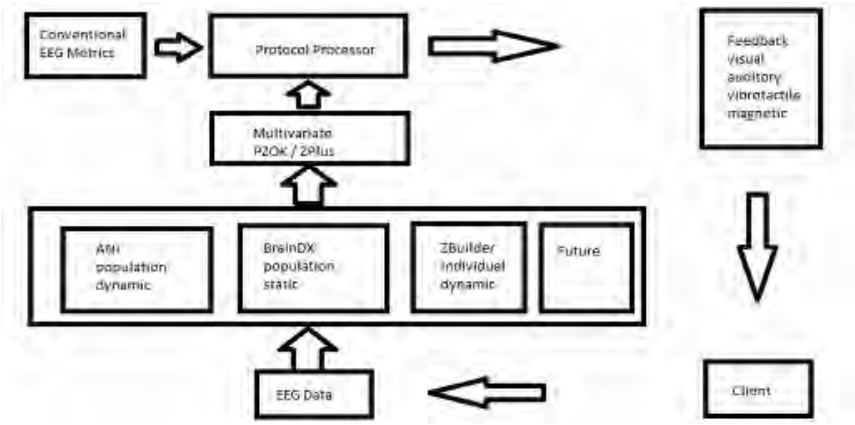


Figure 1: *Software design of a general-purpose Live Z-Score training system.*

When the LZT method emerged, the reference chosen was the Lifespan database, which is comprised of 725 individuals meeting specific acceptance and rejection criteria, and derived according to well documented principles (Thatcher). It includes eyes-open (EO) and eyes-closed (EC) conditions, which must be selected by the user, along with specifying age, when initializing the software. Therefore, each client receives feedback that indicates how well that client's EEG matches, in certain ways, the EEG of a population of selected individuals who were sitting still, being monitored in accordance with the Lifespan protocol. Subjects were not performing any particular task, and they were not selected with any goal in mind other than representing a population of symptom-free individuals who were not diagnosed with any mental or emotional disorders.

More recently, the BrainDX database reference has been added (John et al.) as an option for mini-assessments, and for neurofeedback training. This latter reference consists of static data acquired, also at rest, and reflecting the population data when averaged over a minimum 2-minute epoch. As will be described below, the target values (means) for both references are theoretically, and in practice, the same, and the only difference in principle is in the variation of the data. This difference, which has long been recognized as a scale factor of approximately 2X, is easily compensated for in practical training and assessment, by accounting for the simple fact that the observed z-scores will be larger when using the static database reference.

One of the early concerns that was raised with the emergence of LZT was the appropriateness of the reference. It was suggested that by using a population average of typical individuals, particularly individuals who were not under any task, was not an appropriate reference. It was argued that the reference EEG might not well represent the EEG that would be desirable for any particular client at any particular time. An additional concern was that individuals may or may not have EEG characteristics that were not typical, but that were appropriate for them. The idea of individualized references, as well as "optimal functioning" or "peak performance" references was elevated early on, and remained in the background as a concern as LZT continued to develop and proliferate.

What is "normal?"

Because the typical "normal" reference is based upon a population statistic, certain observations may be made at the outset. The first is that this reference EEG does not in fact represent any particular functioning brain. In fact, there may be no brain that meets these conditions at all. As an example, if we were to compute the typical "normal" man in terms of height, weight, body proportions and muscle and fat proportions, hair color, blood chemistry, and so on, we would have a portrait of an average man.

However, not only does no such individual necessarily (or likely) exist, but also there is no a priori reason to expect that someone would benefit by becoming more "typical." Surely, normalizing critical levels such as excess blood sugar, hypertension, or obesity, would be expected to be of general benefit. However, if an individual has a personal profile that puts their optimal functioning at some other level, then the normative comparison cannot take this into account. To put this in perspective, one might ask, how often a practitioner tells a client "your problem is that you are not more average."

In relation to EEG z-scores, or for

any biologically-related metric, we can ask the question, do we really expect everyone's brain to be the same, such as these dancers all lined up in a row?

Or is this not a more realistic scenario, with individuals expressing their own individual characteristics, strengths, and weaknesses?

It is clear, intuitively and practically, and has been borne out by research and clinical experience, that not everyone who is asymptomatic and with an unremarkable medical history ("normal") has the same EEG pattern (Johnstone et al). Even normal individuals, including those in the database, reveal particular patterns that reflect personal style, strengths, and weaknesses, but do not necessarily imply pathology. That is the reason that the z-scores have their particular standard deviations. The values vary within the sample population, and a certain percentage are, by necessity, not near the center of the distribution. It is a necessary fact that, for example, 40% of the population will be outside the plus or minus one standard deviation limits, for any arbitrary metric. That is how the metrics are constructed. It also means that 1 in 20 readings will, statistically, be expected to be at or beyond two standard deviations. Given that an individual may be characterized by thousands of different z-scores, we must necessarily expect deviant z-scores, even among normal individuals. Furthermore, there is no particular reason that anyone is any better off if they are near the centers of the distribution. In particular, it is not necessarily true that anyone with a z-score of two on any metric will necessarily be any better off if that parameter moved toward the center of the population mean.

As a theoretical ideal, the best and only "pure" reference for a given individual during a neurofeedback task would be the EEG of that individual, in a more desirable state. Whether that corresponds to characteristics of a population average is not a presumptive fact. The concept of normative qEEG was introduced independent of the idea of live training to z-score norms. It is not at all clear that the normative average sample is the only, or even an optimal, target for operant learning. However, this view may be taken a priori based upon a mechanistic, interventional model that subscribes to the idea that all brains should be the same.

As a more specific example of a limitation of normative database reference is that a certain percentage of individuals will, by definition, be on a deviant part of a distribution. However, since all entrants into the database are purportedly "normal," a given percentage of the population will necessarily be significantly deviant. For example, the following figures show the EEG of an asymptomatic, high-performing individual who happens to have a fast posterior dominant rhythm (PDR). Based on a visual inspection, it is clear that this individual simply has an alpha peak frequency at or near 12.0 Hz, which is at the high end of the "normal" distribution. By definition, some percentage of the normal population will present with this finding.

Two findings are evident from this analysis; one is that because the EEG alpha frequency is significantly fast, yet normal for this individual, the z-score computations produce misleading results. It appears that this individual has elevated levels of beta activity, as well as high beta. The excess beta is due

to the fact that some of the EEG alpha actually exists in the beta band, as defined. A second fact, which is a limitation of any Fourier-based method, is that there appears to be a second harmonic to the fundamental, evident as a broad spectrum of energy, centered at exactly twice the dominant alpha. This harmonic is not due to any aspect of the equipment, aside from the fact that Fourier analysis uses simple sinewaves as the basis function, and any deviation from a simple sinewave appearance will produce higher harmonics. The alpha is visibly nonsinusoidal in this case, consisting of a sharper top wave and a flatter bottom wave. This does not constitute any "real" beta activity, but simply shows that the wave is not a simple sinewave. There is no a priori reason that EEG waves should be sinusoidal, and in many cases they are not, such as the boxlike shape of theta, or the wicket shape of mu waves.

We therefore see several limitations of a sinewave-based metric that assumes the presence of exact frequency bands and pure sinewaves. The following example (figures 2 and 3) shows a perfectly functional, asymptomatic individual, who happens to have a peak alpha frequency at the high end of the population distribution. Moreover, the alpha waves are not purely sinusoidal, and have a different shape at the top of the wave, compared to the bottom of the wave. These two characteristics combine to produce a qEEG result that appears to show excess energy in the beta range. Of particular concern is the fact that the nonsinusoidal wave morphology introduces a first harmonic at twice the fundamental, so that the FFT analysis shows abnormal energy in the vicinity of 24 Hz, in a broad band. This phenomenon will occur with any Fourier-based method, including JTFA analysis. Therefore, whether one uses an FFT or JTFA-based method, the presence of rhythms in the boundaries of the component bands, or with a nonsinusoidal waveform, will produce these types of anomalous readings.

map is interpreted on its own, one might consider this individual significantly abnormal, and having excess beta and high beta. However, this is not at all the case. This example shows that a normative sample may fall short of providing a useful assessment basis for those at the extreme of the population. It also suggests that LZT training that depends strongly on normalizing these aspects is likely to emphasize factors that are either irrelevant, or even counterproductive, to appropriate clinical progress. For example, not only is it not clear that reducing the amplitude of these signals, or the frequency of alpha would be beneficial, but anecdotal experience has shown that a client may or may not find that a training bias toward "normalization" will produce positive results.

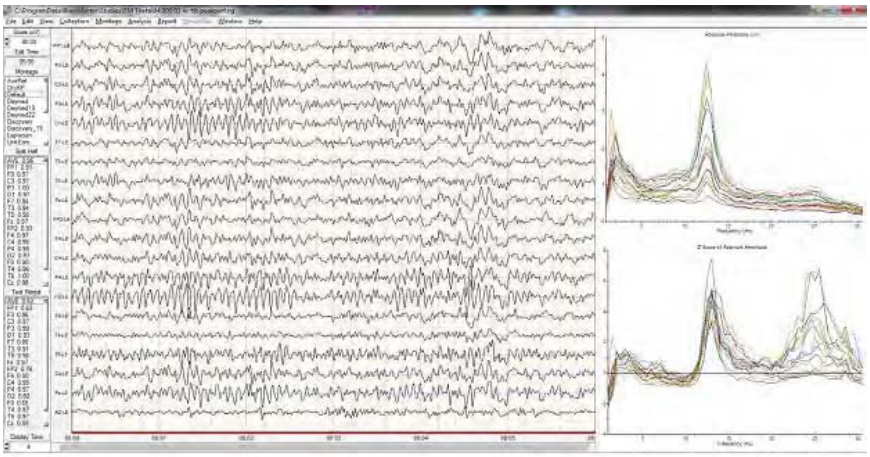


Figure 2: *Spectral distribution. Non-sinusoidal peak alpha at the high end of the population distribution, producing g artifactual beta.*

The resulting maps show these excesses. They are not related to any aspect of the equipment, but reflect rather the vagarities of using an FFT to analyze a peculiar, yet normal, waveform. If the

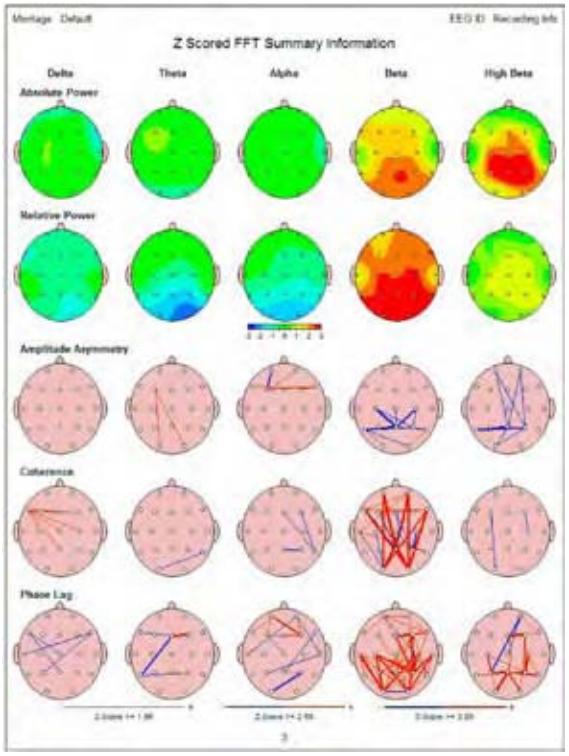


Figure 3: *Topographical z-score map. Non sinusoidal peak alpha at the high end of the population distribution, producing g artifactual beta. Using FFT or JTFA-based method, the presence of rhythms in the boundaries of the component bands, or with a nonsinusoidal waveform, will produce anomalous readings.*

Figure 4 (courtesy of D. Kaiser) shows the distribution of alpha peak frequency in a normal population. It is evident from this graph that a significant segment of the normal population will have a peak alpha that is either at or below 9 Hz, or at or above 11 Hz. Because these individuals lie at the edges of a typical qEEG alpha band, their EEGs will tend to produce "abnormal" results when subjected to a statistical comparison such as a z-score.

A further complicating factor occurs with respect to aging. A database can attempt to compensate for age-related changes by either using a "bin" method, or by regressing values against age. This will effectively ensure that the database has age-appropriate norms for the chosen bands. It does not, however, ensure that the bands chosen are appropriate for any age.

Figure 5, (from http://www.iomonitoring.pro/eeg.htm) shows the typical values of posterior dominant rhythm (PDR) as a function of age. It shows that the PDR changes quickly from ages one through five, in particular. One result of this fact is that, as the PDR moves from one band (theta) into the other (alpha) in the analysis, abrupt changes in scores may be observed (Mulder, 2013). For example, a child of age 4 will have a PDR that lies at the cusp of the two bands, and will not be adequately represented. This suggests that, particularly with respect to age, fixed bands may be a limitation. Furthermore, customized bands may be more desirable, both with regard to age, and with regard to individual differences.

Effects of eye and task conditions

A further consideration relates to the conditions of the reference acquisition. Most existing reference databases include an eyes-closed (EC) and an eyes-open (EO) condition. Some also include one or more task-related conditions. Any of these references might be used either for assessment, or for LZT training. Therefore, it is important to understand how the brain responds to these conditions, with respect to particular EEG frequency bands and amplitudes.

The following graphs (developed in collaboration with D. Kaiser) show the typical effect of closing the eyes in a normal adult population (figure 6), as well as task-related changes (figures 7 and 8). This confirms the well-known observation that alpha increases by a factor of up 2.2, maximally in the occipital leads. Because this set of curves is based upon a population statistic, it accurately represents the differences that will exist in a z-score reference of eyes-closed EEG, when compared to the corresponding eyes-open EEG. It may be noted that the particular sets of leads can be separated by their response to the eyes-closed condition, and break naturally into bands such as 8-12, 4-7, and 12-15, based upon the observed separation of curves. This provides an interesting validation of the choice of the standard bands, showing that they do reflect something about how the brain is wired, and how it responds to changes, in this case, the closing or opening of the eyes.

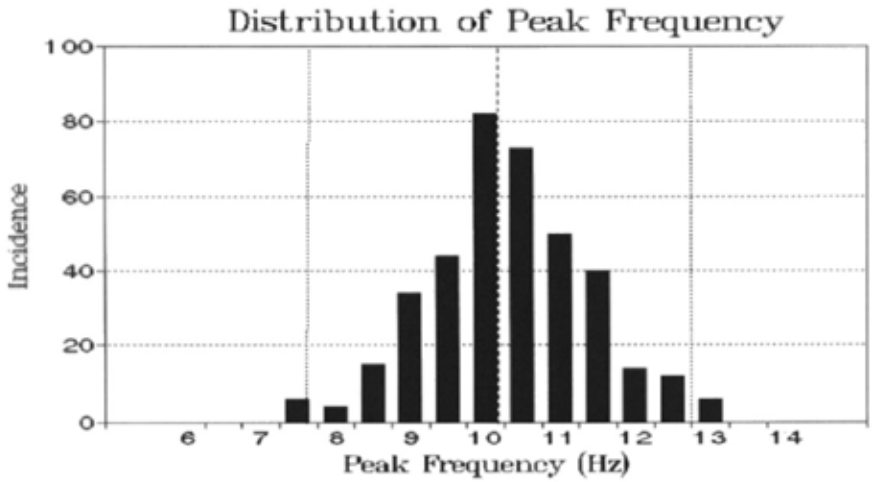


Figure 4: *Distribution of alpha peak frequency in a normal population (courtesy of D. Kaiser).*

The following

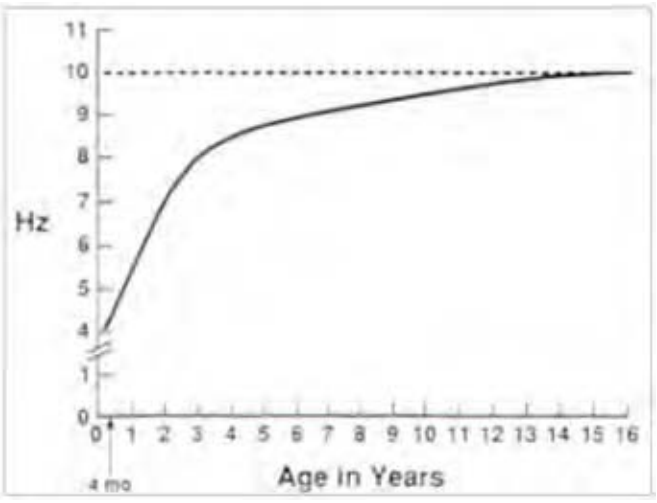


Figure 5: *Posterior dominant rhythm as a function of age.*

Typical values of PDR as a function of age

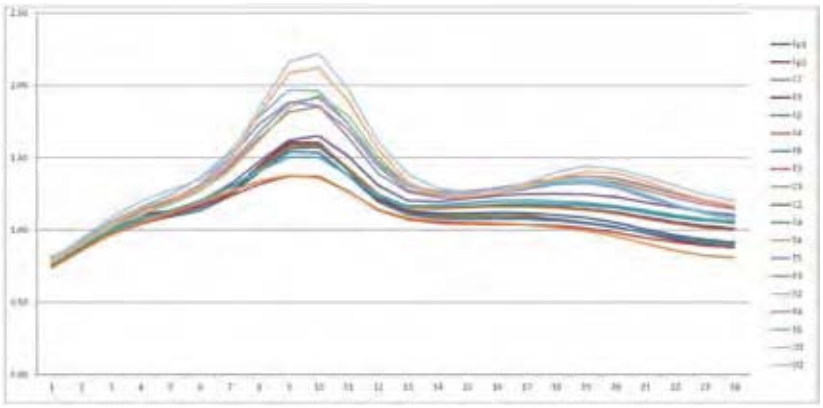


Figure 6: *Effect of eyes closing on EEG amplitude.*

The following graph shows the EO EEG compared to a task (age-appropriate reading). In this case, we observe that the population shows increases of up to 100% in the low delta range, increases up to 1.5 in the alpha range, and less change in the theta and beta ranges.

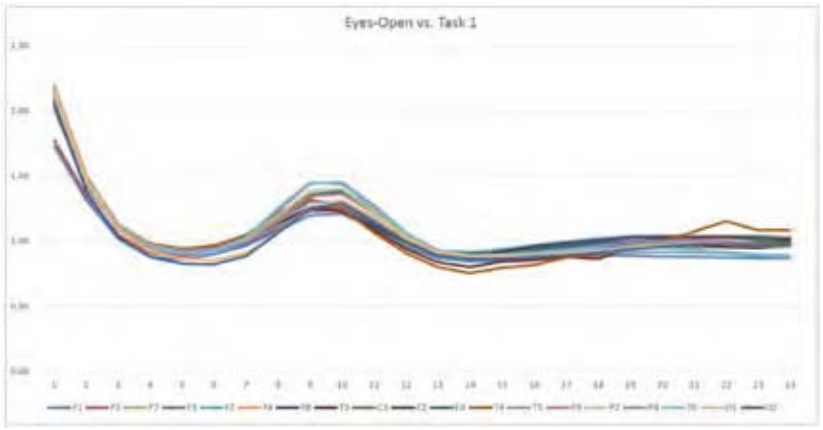


Figure 7: *EEG amplitude. Eyes open vs. Task 1.*

The following graph shows the EO EEG compared to a different task (serial 7's). In this case, the changes are even more pronounced. In this case, the difference in alpha increases to a factor of up to three, and a further dependence on beta occurs, in both directions, in the range of 15 to 24 Hz.

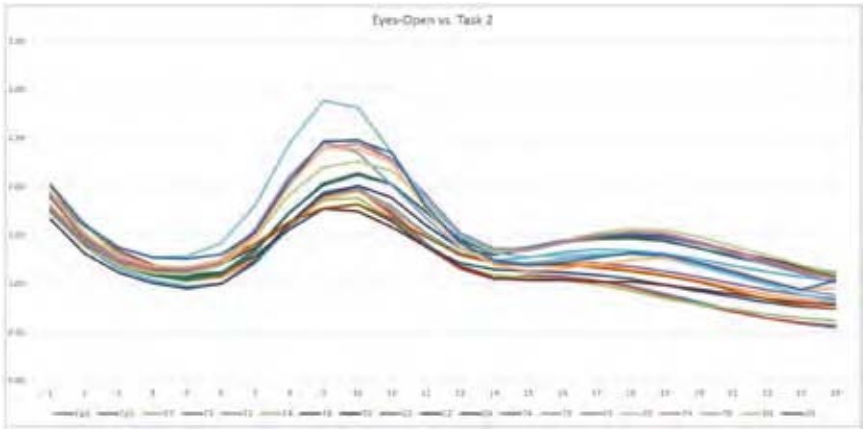


Figure 8: *EEG amplitude. Eyes open vs. Task 2.*

These two comparisons between eyes-open resting condition and task conditions provide several important observations. One is simply that a brain under a task can have an EEG amplitude pattern that differs significantly from that at rest. Another observation is that the differences are frequency dependent, and are most significant in the alpha range.

When put into practice, the intent of the LZT reference may be open for interpretation, and there is room for creativity in this aspect. For example, a reference may be designed to place a demand on the client, other than to simply "be more normal." There is an analogy to other forms of therapy, such as paradoxical intention in psychotherapy, which facilitates change by moving the client into an extreme position, and then allowing for learning to occur. There is no authoritative reason why neurofeedback must be done using a reference that purports to be some "ideal" or "most efficient" pattern.

Neurofeedback, flexibility, and variability

The primary issue with neurofeedback can be considered to be one of flexibility, not necessarily adherence to a particular norm. For example, figure 9 shows mean z-scores (colored bars) as well as z-score variation ("error" lines) for a 1-minute sample of EEG. It is evident that the z-scores that are closest to normal also exhibit the greatest variation. The few z-scores that are the most deviant also show the least amount of variability. It is almost a rule that any variable that is more deviant will have a tendency to be less variable, in a system in which variability is one of the key elements of self-regulation.

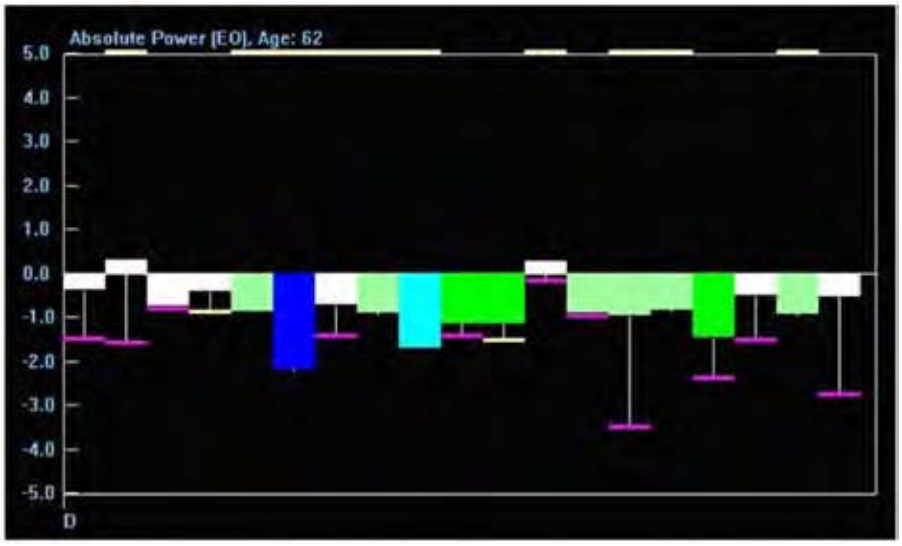


Figure 9: Mean z-scores (colored bars) as well as z-score variation ("error" lines) for a 1-minute sample of EEG.

certain percentage (usually 10% to 40%) to lie outside the target range, while the client still gets positive rewards. This allows the client's brain to adjust in an individual manner, and to allow some values to remain "deviant." Without this provision, the necessity would arise to pay more attention to the specific choices of z-scores, and to avoid z-scores that do not specifically relate to the complaint or disorder under care. This also provides a robust approach to "optimal functioning" and "peak performance" z-score training, because an individual's unique characteristic(s) would naturally tend to occupy the population of outliers that is ignored, hence neither reinforced nor inhibited, by the training protocol.

The concept of paradoxical training has existed in other areas of psychotherapy. By challenging an individual in a particular way, it becomes possible to enable a system to explore different boundaries and modes of behavior. As one example, a golfer might temporarily place a weight on a club, in order to exaggerate the motor activities associated with a swing. When the weight is removed, the swing is improved in the unweighted case, as well.

Choice of population (or individual) references

It is important that a reference can be associated with a normally distributed population of values. However, this does not require a population of individuals. A series of samples from any individual, taken over time, is in itself a statistical sample. Once the relevant values are reduced to simple tables of means and standard deviations, all that matters is that the reference values are correct, and that there is some normal distribution that underlies them. As an example of a normal population of values derived from a single individual, figure10 shows the distribution of instantaneous values over a 1-minute epoch. The gaussianity of this distribution is visually evident.

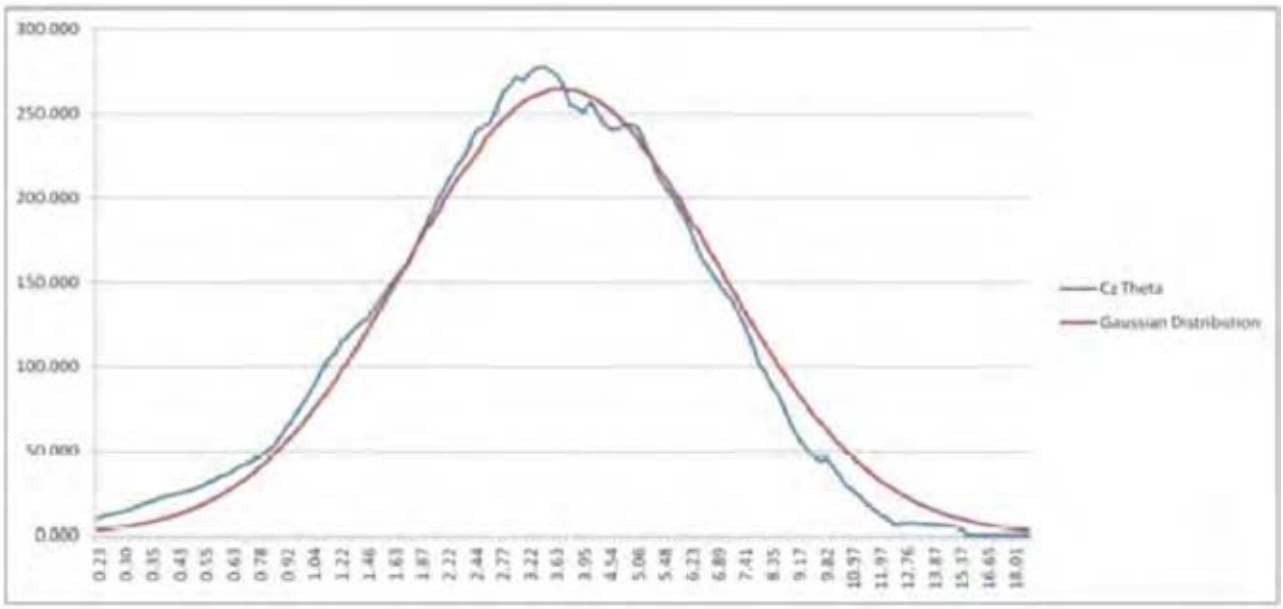


Figure 10: *Distribution of instantaneous values from a single individual over a 1-minute epoch. The gaussianity of this distribution is visually evident.*

och. The gaussianity of this distribution is visually evident.

The validity of an individual reference for LZT use can be further validated statistically. The following table summarizes an example of goodness-of-fit values for all 19 10-20 sites, for 10 frequency bands, for an example 1-minute sample. Figure 11 shows these values in graphical form. The bars represent the goodness of fit for every site, and for every component band. It is evident that a high-quality fit is achieved for every value and every site, from this sample of EEG.

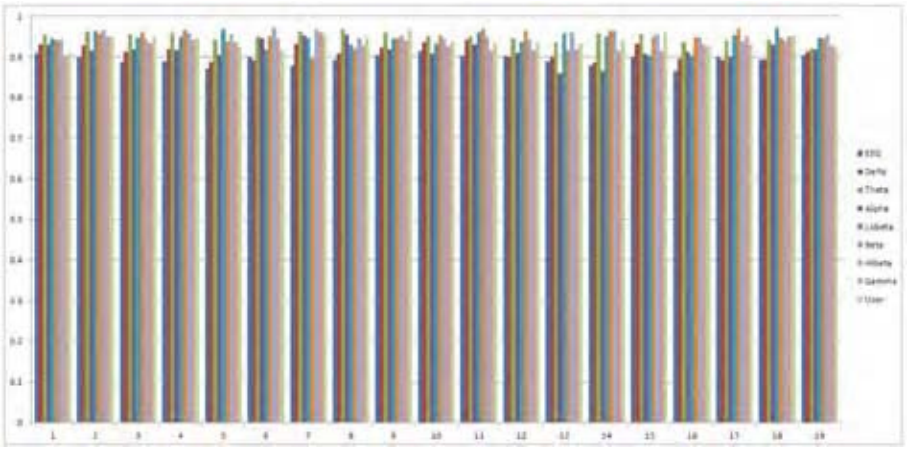


Figure 11: Goodness-of-fit values for all 19 10-20 sites, for 10 frequency bands, for a 1-minute sample.

qEEG Z-Score cryogenics

The ability to construct an LZT target from an arbitrary sample of EEG opens the door to many possibilities. One is to capture EEG signatures from individuals as a precaution for future events or conditions. For example, if EEGs are taken from all participants in athletic competition or other potentially dangerous activities, these can be used as references to assess the effects of injury or other adverse events or conditions. Decisions regarding whether an athlete has been significantly impaired, and should or should not return to play, can be well addressed by comparing the EEG with a known healthy condition. In the practice of optimal aging, it is also possible to capture EEG from individuals in healthy states, before age-related decline sets in. By training to one's own EEG during healthy phase, one can avoid the possibility of less than optimal results if one's neurofeedback is directing the client away from their own healthy operating parameters.

In many practices, the qEEG reference database is used for both assessment, and for LZT neurofeedback. In the original embodiment, one set of computed references was used for the assessment phase, and a different set was used for LZT neurofeedback. This was done so that the instantaneous values used for training would correspond to the instantaneous variation observed in the reference sample. For the instantaneous references, both the between-subject variation and the within-subject variation were included in the data. As a result, since the reference standard deviations are larger, the resulting computed live z-scores are smaller, typically by 1 to 1.5 standard deviations. When this was first observed, there was some confusion and concern, and it was necessary to explain the statistics before users became comfortable with this difference. The question then arose, why cannot the instantaneous values correspond to the assessment values, so that an EEG that produced a 3.0 SD excess of beta on a report, would also produce a 3.0 SD excess in the live display. The fact is that this is possible, and

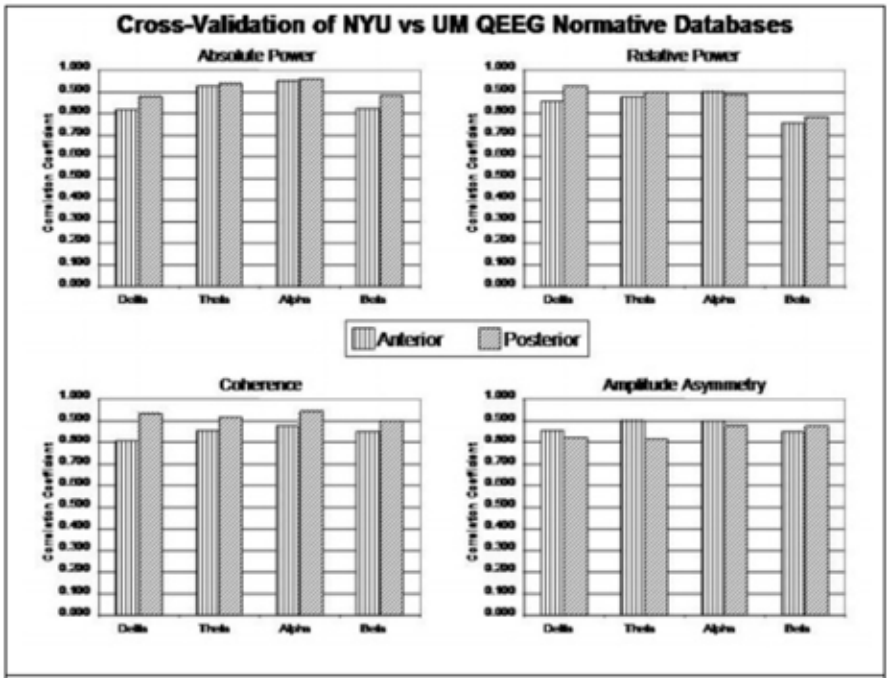


Figure 12

by using a static reference for live training, the expected correspondence can be observed.

Static versus Dynamic Z-Score References

When normative databases are constructed using similar principles, it is an expected, and observed, result that they will produce similar references. Figure 12 (from Thatcher & Lubar) shows the match, for example, between the Lifespan and the BrainDX databases. This match therefore demonstrates that different databases can be used as references, and will produce equivalent results.

Figure 13 shows the relationship between the dynamic EEG data and the population values, as well as how the static distribution compares with a dynamic distribution. The static norms reflect the average values for multiple individuals, but do not reflect individual variation. The dynamic population norms, by incorporating all of the variation, both between and within individuals, produces a very large distribution. However, this distribution does not represent the range of any particular individual's optimal functioning. The figure also shows three individual distributions, representing individuals on the high, middle, and low parts of a population.

Consider Mr. "Red" for example. His normal range of function is represented by the red parameter values, and the red bell curve describing his distribution. If Mr. Red is somehow dysregulated or meets with some adverse conditions, his EEG may deviate from that normal set of values, either by becoming hyperactive (excess) or hypoactive (deficit) in that particular value. If a standard normative reference is used to train Mr. Red to recover, then the system will, by definition, tend to reward Mr. Red when his values move more toward a "normal" level, which may not be optimal for him. The assumption that a population statistic is optimal for all individuals is tantamount to assuming that everyone is a "Mr. Green" or would be better off by being more like Mr. Green. However, this contradicts the foundational assumption of the database, which is that everyone in the population is healthy, even if they occupy outer regions of the normal distributions.

There is a need to demonstrate the equivalence between obtaining live training data from more than one possible real-time implementation. In particular, although static references are generally computed using FFT's, the live data is more often obtained using real-time method such as using digital filters or a related complex demodulation technique (Collura, 1990)

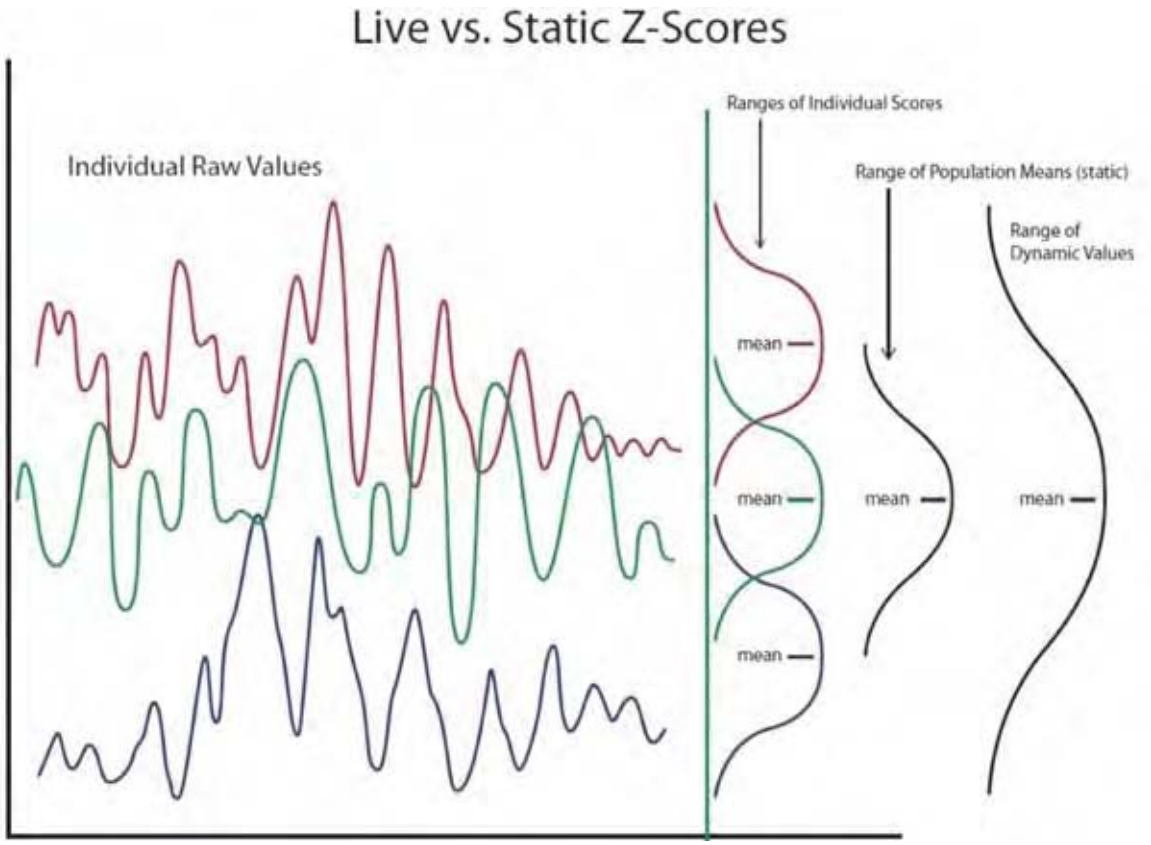


Figure 13: *Relationship between the dynamic EEG data and the population values.*

When comparing static and dynamic results, it is important to consider relevant similarities, and differences, in the methods used to compute parameters.

If one method is used to compute the reference values, and a different method is used to compute the real-time values, then it will be of concern whether there is concordance, or consistency, between the methods. If one chooses a particular method, e.g. FFTs of 2-minute samples, for the static values, and uses a different method, e.g. some other transformation, for the dynamic values, there may not be sufficient consistency to consider them to provide similar information. For example, if I use height as a measure of growth each month and weight as a measure every year, and attempt to correlate them, there will be a poor match, because these two parameters measure essentially different characteristics. With regard to waveforms, there are parameterizations that relate to, for example, height (e.g. peak-to-peak amplitude), while others relate to weight (e.g. total power). The assumed or actual match between two different metric approaches cannot be assumed; it should be demonstrated.

There are implementations in which the static data are derived from FFT analysis of long samples, while the dynamic data are derived from a different type of transform, such as a Hilbert or Gabor transform. When one examines these transforms, they are all essentially Fourier-like methods, but with variations in the kernel of the integral. When these methods are compared, differences of up to 18% between different types of transforms can arise, due primarily to differences in the windowing or kernel function.

The value of JTFA transforms is that they produce information in time as well as in frequency. However, all such transforms are technically defined over an infinite time interval, and for them to produce output related to the signal of interest, the signal must be in the center of the window. Signals near the epoch edges are effectively reduced or even removed by the windowing function.

For this reason, a transform might be selected for the real-time computation, as well as for the reference database. However, transform methods are not well suited to real-time implementation, because of the inherent delay associated with the epoch and windowing operation. With a 1-second analysis epoch, any transform-based method will experience a response delay on the order of ½ of the epoch length, or 500 milliseconds. This is rather long, when compared with the response times of methods based on digital filters.

Most neurofeedback software employs digital filters, or a related method, to compute real-time data for biofeedback purposes. This is because a digital filter provides a faster response time than any transform-based method. Digital filters provide a continuous processing of the data, and proceed one data point at a time, without having to use any particular epoch size or windowing technique. Digital filters emphasize the most recent data, and gradually deemphasize earlier data, with a continuous function that is defined by the filter type. Digital filters are designed using different methods, such as Butterworth, Chebychev, Elliptical, or other methods. All digital filters proceed by adding a single data point to the computation, and combining it using weighting coefficients, with the previous data and results. This is in contrast with transforms, which always look at a finite extent of past data, usually on the order of 1 second, and analyze it in isolation, so as to estimate the immediate value of a relevant parameter. It is this windowing and epoch selection that causes all transforms to suffer from a built-in delay that is independent of how fast the computer is. Even if a computer is infinitely fast, a transform will always introduce a response delay because of the way that it looks at the data.

The ability to match static references and dynamic calculations cannot be taken for a given, unless either the same method is used for both, or if the correlation can be justified and demonstrated. As an example, if one takes weight on a clinical medical scale, and also at home on a cheap scale, the match may be as poor as 5 pounds, maybe more. But if both scales are at least calibrated to the same reference, a match within 1 pound or so can be expected. Similarly, if one takes care of the relationship between a static and a dynamic computation, and can demonstrate the appropriate relationship, then dynamic measures can be referenced to static data, even if the methodology of the computations is not identical.

In the results shown here, care was taken to implement a digital filter using the method of complex demodulation (Childers), which ensures that the instantaneous values converge to match values that would be obtained from an FFT of the same time frame. Rather than being another epoch-based transform, the filter used here is continuous, and provides output that instantaneously reflects the most recent data, without any delay due to fixed epoch size or windowing. In the steady state, the values would match identically. Due to the effects of the time-variation in the signal, small differences will occur, because the digital filter is actually doing a better job than the FFT of tracking changes in the EEG. However, the ultimate degree of matching can be shown to be within a few percent, even in the face of a dynamic EEG. This matching would not be

possible if the dynamic method used a transform such as Hilbert of Gabor. It is made possible by the fact that the digital filters are designed with an eye to producing results that are comparable to FFT results, even when dynamic and static data are compared.

In summary, rather than there being a hard distinction between dynamic and static data, there is a continuous relationship. As dynamic data are considered over longer time periods, they converge to match the static data, if the computations are done correctly. A long damping factor, or time-constant, when applied to dynamic statistics, produces a result that necessarily matches that of a long-term analysis. If the basic scaling factor between a windowed method (e.g. FFT) and a digital filter is taken into account, the agreement is essentially perfect. In the results shown here, the responses of each filter band were carefully matched between the static norms (BrainDx/NxLink) and the dynamic data (BrainMaster digital filters) so that the agreement is obtained. When a played-back EEG is viewed, revealing short-term changes in z-scores, maps, and sLORETA images, and the damping factor is increased to slow down the responses, the resulting data are essentially identical to that which would be obtained if a longer segment were selected and processed as a unit, providing averaged results. This provides the bridge between dynamic and static data, bringing the worlds of traditional qEEG assessment and live neurofeedback training together into one connected whole.

There are two key advantages to this approach, when contrasted to one that uses one method for static data and another method for dynamic data. One is that the maps and z-scores are entirely consistent. Live maps "look like" static maps, and reveal similar z-score deviations. This eliminates the previous confusion that has resulted when live z-scores did not match static z-scores, but required a compensation of 1 to 1.5 standard deviations to convert from one to the other. A second, more important advantage is that one database can be used for both live neurofeedback and for summary statistics. Rather than having to have one set of norms for assessment and a different set of norms for training, a single set of targets can be used. The difference between short-term and long-term variations can be accounted for by adjusting the size of z-score targets. In the method that uses a single set of references, it is found that z-score targets are more often in the range of 1.0 to 2.0 standard deviations, which is what is intuitively expected, rather than the 0.5 to 1.0 standard deviations that is used when a separate database constructed using a different dynamic computation method is employed.

Figure 14 shows, in real-time, a comparison of results obtained from an FFT (top) with those obtained from a quadrature digital filter that implements complex demodulation (bottom). It is clear that both signals have the same behavior in time, and one appears to be essentially a replica of the other.

The similarity in the time-progress

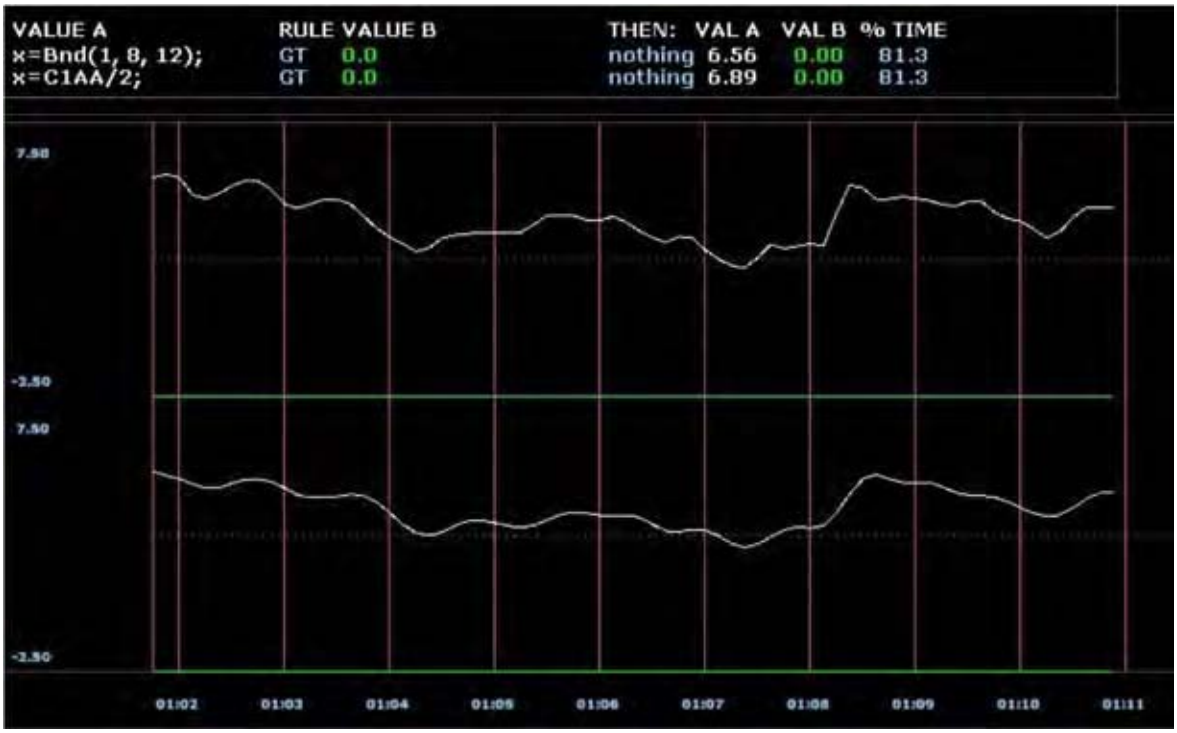


Figure 14: *Real-time comparison of results obtained from an FFT (top) with those obtained from a quadrature digital filter that implements complex demodulation (bottom). Each signal has the same behavior in time, and one appears to be essentially a replica of the other.*

of the two signals is visually apparent, and can be further confirmed by plotting the values against each other. Figure 15 shows a comparison of live values obtained from FFT, and from complex demodulation, plotted against each other. A scatter plot of this type is used to confirm a match between two variables, in a linear fit. In this case, a goodness of fit of 97.23 percent is observed. There is also a constant ratio, or scale factor, of 1.0629, which amounts to a consistent six percent ratio. This is explained by the difference in that an FFT uses a tapering "window," while the JTFA does not. When this window is accounted for by this constant scale factor, the resulting accuracy is therefore roughly 2.8 percent, or plus or minus 1.4 percent. This difference is insignificant for z-scores, which, particularly if they are taking into account population and/or individual variation, must vary much more than a few percent, to produce a change of even a tenth of a standard deviation.

The following comparison shows that this match is valid in practice, as it shows comparison maps taken from 10 seconds of EEG, and plotted using three methods. The top set is generated within NeuroGuide using the ANI database, the second set is generated within the BrainAvatar software using the BrainDX references, and the bottom set is generated within the BrainAvatar software, using the ANI references. The maps are essentially identical in all bands, with the proviso that the BrainAvatar ANI maps, being derived from a dynamic reference, show slightly smaller z-scores. There is a further slight difference in the precise definitions of the frequency bands, which would account for some of the minor differences observed.

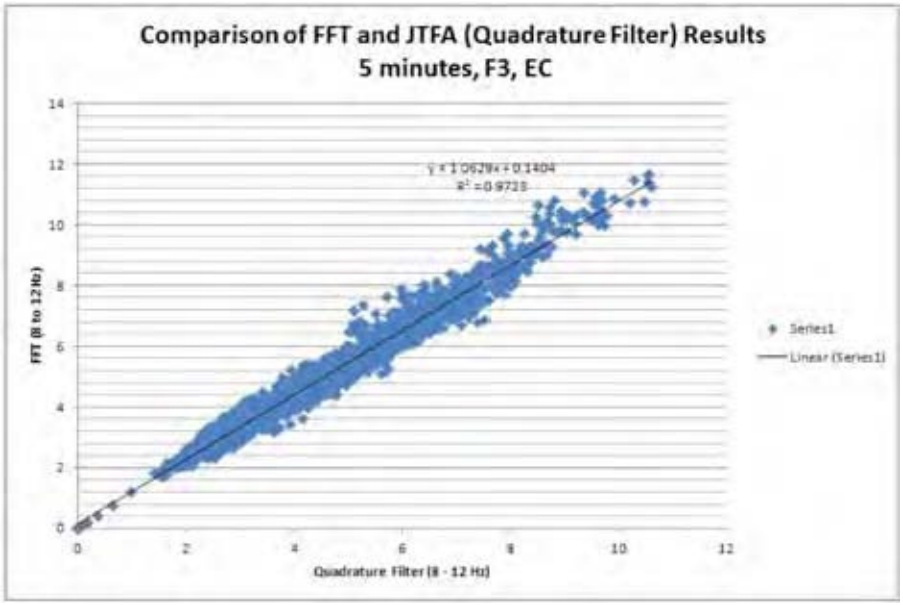


Figure 15: *Scatter plot comparison of live values obtained from FFT, and from complex demodulation.*

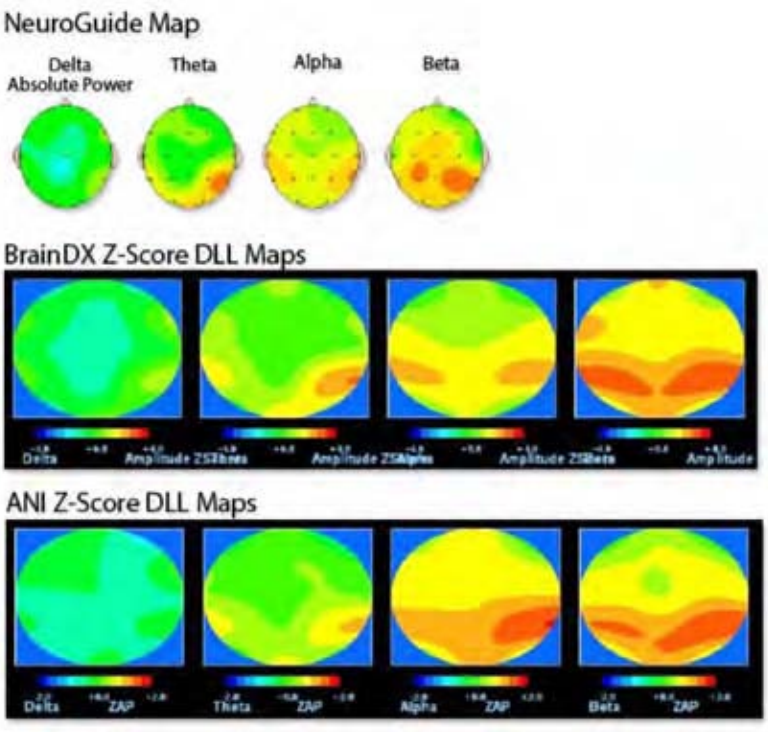


Figure 16: *Comparison maps taken from 10 seconds of EEG, and plotted using three methods. Top row NeuroGuide using the ANI database. Middle row BrainAvatar software using the BrainDX references. Bottom row BrainAvatar software, using the ANI references.*

As a further example of the ability to match dynamic with static statistics, Figure 17 from Collura shows the agreement between a coherence measurement derived from a digital filter implementation (BrainMaster) with those obtained using an FFT (NeuroGuide). It is clear from this example that both methods produce comparable results, across the entire range of coherence values from very low (<10%) to very high (80%).

As a verification that the static reference can be used for LZT neurofeedback, Figure 18 shows the progress of an LZT session with a client using the BrainDX Live Z-Scores and Percent Z-OK training:

The observed behavior in this session is typical, and is essentially the same as has been observed when using the original ANI LZT implementation. Z-Scores typically require a few minutes to begin to adapt, and significant training effect is generally seen between 5 and 15 minutes into the session. It is also typical that sometime after the 10-minute mark, the client may begin to tire, and z-scores will begin to diverge again. At this point, the session should be ended. This session summary example confirms that even when using static targets, it is possible to perform effective LZT training, providing simply that the target ranges are chosen at an appropriate level.

Figure 19 shows a summary of the relevant raw values during this session, demonstrating that key EEG parameters shifted during the session as a result of the z-score feedback. This includes decreases in slow-wave activity (delta, theta, and alpha), as well as increases in beta activity.

Online references and further detailed examples of static and dynamic z-scores and maps can be accessed from: http://www.brainm.com/kb/entry/540/

Conclusions

Ultimately, neurofeedback therapy is as much an art as a science. While technical principles underlie its effectiveness, what occurs in the end is that the brain is informed, challenged, and lured into various conditions of awareness and responsiveness. How the brain responds is very much a function of each individual's unique characteristics, the approach of the clinician, and finally, the specifics of the equipment. There is often more than one way to achieve results, and the process is not a linear one, but a complex nonlinear interaction. For example, LZT training can be and is combined with other modalities such as conventional directed EEG training, HEG, or audiovisual or electromagnetic stimulation. Also, protocols can be designed to

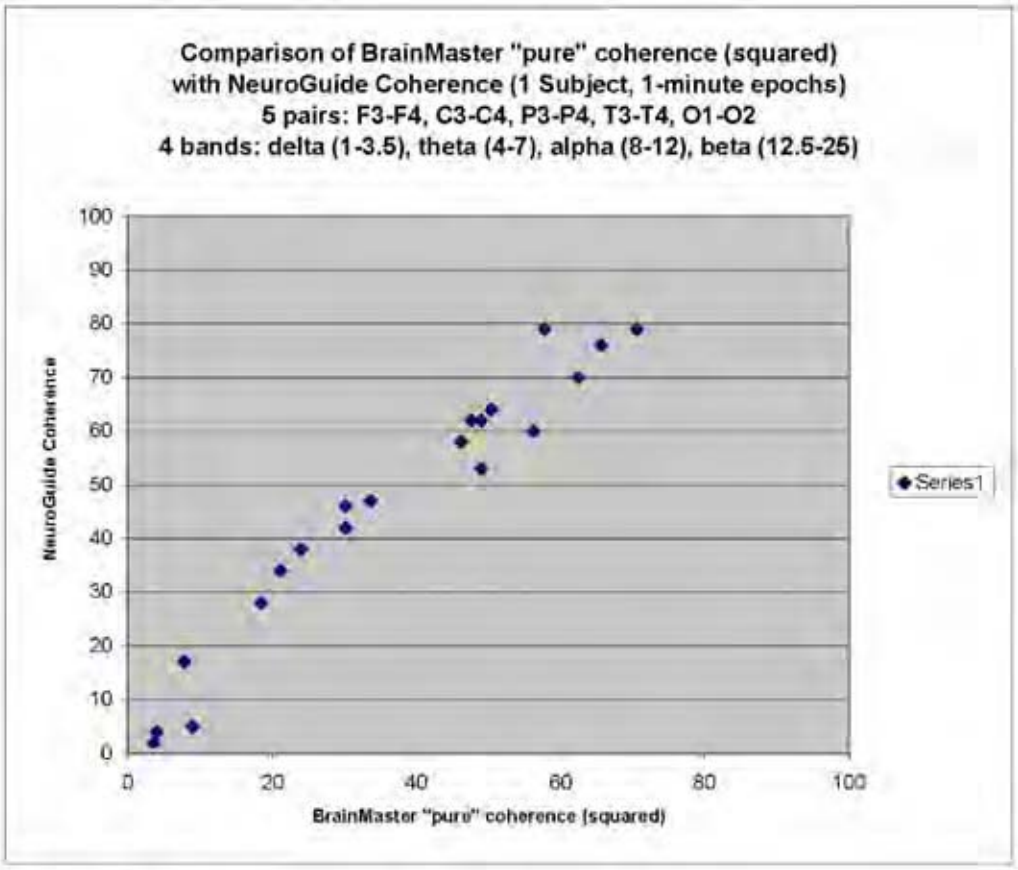


Figure 17: *Agreement between a coherence measurement derived from a digital filter implementation (BrainMaster) with those obtained using an FFT (NeuroGuide).*

ing/compensating, or individual qualities related to personal optimization or preference of brain state and function.

The brain's goals are effectively supplemented with additional goals related to its internal state and qualities of self-regulation (or not). By using various references and different approaches, it becomes possible to work with regard to the client's progress as a process that may include principles of direct challenge, alternating challenge and rest, paradoxical, and other types of information. The position taken here is that there is a wide range of possible approaches to creating z-score template references, including individualized, specialized populations, and task-related methods, which have yet to be fully explored.

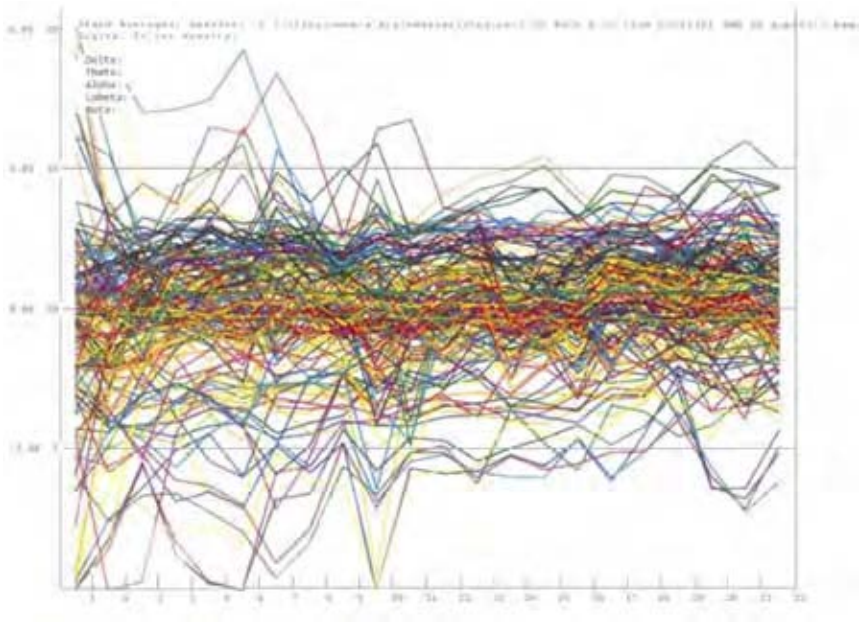


Figure 18: *Progress of an LZT session with a client using the BrainDX Live Z-Scores and Percent Z-OK training*

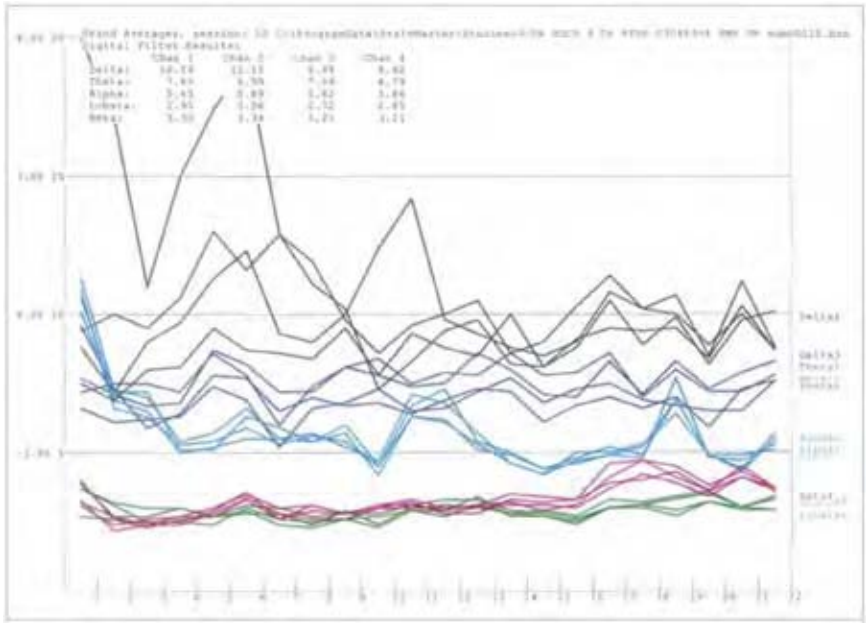


Figure 19: *EEG parameters shifts during the session as a result of the z-score feedback included decreases in slow-wave activity (delta, theta, and alpha), as well as increases in beta activity.*

References

Collura, T.F. (2013) Technical Foundations of Neurofeedback. New York: Routledge: Taylor and Francis.

Collura, T.F. (2008) Towards a coherent view of brain connectivity. Journal of Neurotherapy. 12, 2–3, 99–110.

Collura, T.F. (2008) Whole-head Normalization using Live Z-scores for Connectivity Training (part 1), NeuroConnections April, 12–18.

Collura, T.F. (2008) Whole-head Normalization using Live Z-scores for Connectivity Training (part 2), NeuroConnections July, 9–12.

Collura, T.F. (2009) Neuronal Dynamics in Relation to Normative Electroencephalography Assessment and Training, Biofeedback Volume 36, Issue 4, pp. 134–139.

Collura, T.F., Thatcher, R.W., Smith, M.L., Lambos, W.A., and C.R. Stark (2009) EEG Biofeedback training using Z-scores and a normative database, in: (Budzynski, T., Budzynski, H., Evans, J., and A. Arbanal, (eds) Introduction to qEEG and Neurofeedback : Advanced Theory and Applications, Second Edition. New York: Elsevier.

John ER, Karmel BZ, Corning WC, Easton P, Brown D, Ahn H, John M, Harmony T, Prichep LS, Toro A, Gerson I, Thatcher RW, Valdes P, Kaye H, Valdes P, Schwartz, E., Neurometrics: Numerical taxonomy identifies different profiles of brain functions within groups of behaviorally similar people. Science 1977; 196: 1383–410.

John ER, Ahn H, Prichep LS, Trepetin M, Brown D, Kaye H. Developmental equations for the electroencephalogram. Science 1980; 210: 1255–8.

Cantor, D. et al. see: http://www.braindx.net/

Thatcher, R. & Lubar History of the Scientific Standards of qEEG normative Databases. In: Budzynski, T., Budzynski, H., Evans, J., and A. Arbanal, (eds) Introduction to qEEG and Neurofeedback: Advanced Theory and Applications, Second Edition. New York: Elsevier. Note: Certain of the methods described herein are protected by US, Canadian, and foreign patents and patents pending.

A Case of History

Corey Feinberg, MA, Elsa Baehr, PhD

Many cases have passed through the doors of our small clinic on the northern outskirts of Chicago. Amongst the hundreds of these cases, there was one in particular that can be distinguished, for it was as interesting and powerful as it was efficacious. It was a case filled with many intricate layers and subtle nuances that to this day remains unparalleled, deeming it well worthy of recounting in this forum.

The subject of this narrative is a woman in her mid-fifties who, in the interest of confidentiality, we will refer to as "Mary." From an outsider's perspective, Mary may appear to lead a typical, if not privileged, life. She is an attractive woman, well groomed, articulate, and a successful artist by trade. Mary lives with her husband in an affluent suburb raising three beautiful children. However, as most of us in the psychological community know all too well, it is not what is on the outside that matters, and for Mary, what lies below the surface tells an entirely different story.

Mary came to our clinic in March of 2011 as a last resort. She had been diagnosed with Posttraumatic Stress Disorder (PTSD) years earlier by a marriage counselor who then referred her to a therapist that specialized in PTSD and other trauma-related disorders. Mary continued treatment with that therapist for several years and is still in treatment with her to date. Together they explored many different types of treatment approaches including techniques such as EMDR, and although significant gains were made over the years, Mary was still plagued with symptoms of dissociation, anxiety, and deep depression on a regular basis.

I had the opportunity to interview her therapist for the purpose of case presentation. Our conversation was centered on her observations regarding Mary's condition both prior to and after she underwent neurofeedback treatment at our clinic. I asked her opinion about the severity of Mary's diagnosis at the onset of therapy and her reply was that she regarded her situation as severe due to the degree of impact her symptoms had on most every relationship in her life and the level to which it interfered with her daily functioning. She went on to say that Mary was very shut down by feelings of abandonment, intense anger and shame, and that she had virtually no coping strategies to deal with those feelings. She described a cyclical process where Mary would have psychological "triggers" or reactions to various behaviors, situations, and even certain sounds that would cause her to dissociate or lose touch with her own feelings. This period of complete emotional numbness would often last for days at a time, according to her therapist. Mary would re-emerge in a state of deep depression when her feelings finally returned. "It was very debilitating" she remarked. Just prior to coming to us for treatment, she told Mary that she thought she was "stuck" and that she should strongly consider a pharmacological approach (to which Mary was vehemently opposed).

In response to her roadblocks in psychotherapy, coupled with mounting pressure from her therapist to "get medicated," Mary sought out neurofeedback treatment at our clinic, which had previously treated one of her children with considerable success. At her initial intake interview, she described her symptoms, much in the same manner that her therapist had described them to me. She pointed out that, on average, she was having nearly twenty triggers a day and explained her dissociative episodes as the sudden sensation of "feelings flowing out of my feet." Sleeping was no time for rest, according to Mary. Incessant teeth grinding and "Every night at the movies!" was the phrase that she sarcastically uttered to depict the relentless onslaught of nightmares she experienced for years on end.

As Mary went on to describe the nature of her dysfunction, it was as if she were painting a picture on the canvas of our minds, with every detail bringing the image into higher resolution. In the end, she had created a portrait of a woman paralyzed with indecision, imprisoned by anger, and demoralized by dissociation. At that point I could

Amongst the hundreds of these cases, there was one in particular that can be distinguished, for it was as interesting and powerful as it was efficacious.

only wonder what set of circumstances could have occurred to lead to such degradation of Mary's psyche.

In order to fully understand how Mary's state of being had come about, it is appropriate to first understand a little bit about her family history. Mary's maternal grandparents resided in a small German town of 2000 people, where they were one of only two Jewish families. They lived in the house that had been passed down in their family since they helped settle the town in 1721.

Mary's grandfather was a decorated veteran of WWI who, sensing growing anti-Semitic sentiments in Germany at the time, decided it would be best to try to send Mary's mother away from the region to safety from Nazi persecution. He soon realized that the doors of opportunity for Jewish refugees to escape were closing rapidly and very few countries were even willing to offer them asylum. Fortunately, the family had acquired considerable wealth, and Mary's grandfather began to bribe anyone and everyone he could. He learned of a small American rescue operation organized by Lutherans, Quakers and Jewish organizations that brought children, ten at a time, from Europe to America on cruise ships. This organization was able to bring over about 100 children a year, saving about 1000 children in all between the years of 1932 and 1945 (Frankel, 2013). Finally, in 1938, at only twelve years of age and entirely by herself, Mary's mother was sent out on a ship bound for America and became one of what are now known as the "One Thousand Children." Upon her arrival, Mary's mother was placed with a blood relative in the Chicago area who had come to the U.S. in the 1920s. She slept on a couch in the dining room of a small two-bedroom apartment and was treated more as a servant than a family member. It was there that she later learned the horrifying news that both of her parents had been killed in concentration camps.

At first glance it may seem as though her narrow escape from the Nazi agenda and the fate of her parents was a story of triumph, but in reality, there were no winners that came out of the Holocaust, just those who survived and those who did not. And neither she, nor her daughter Mary for that matter, could find refuge from the devastating psychological impact that it would have on their family for years to come.

Needless to say, Mary's mother had suffered significant loss. Not only did she lose her parents at the hands of genocide, but along with that her homeland, her language, and her identity were also lost. Mary described her mother as being "arrested psychologically at twelve years old." Her mother's traumas became the cause of extreme anxiety which manifested as mental illness. Mary recalled her being forcibly removed from their home on multiple occasions for lengthy hospitalizations. Her mother's instability and erratic behavior severely affected the manner in which she reared her children. Mary reported her first memory as crying in her crib and nobody coming to sooth her. From childhood through adulthood, Mary identified their interactions as an inversion of the mother/daughter relationship. She always felt the burden of taking care of her mother and to this day she admits that she has a difficult time shaking the feeling that her needs are not being attended to.

Mary used the term "suffocatingly neglectful" to best express her mother's style of parenting towards her. She shared a story from her childhood to illustrate what she meant by that remark. Once, when she was five years old, someone had come to the door and rang the doorbell. In a fit of paranoid delusion, Mary's mother was convinced that the visitor was Adolf Hitler coming to take her children away. In a panicked attempt to save her, she pulled Mary into the bedroom and grabbed her around the neck so tightly that she began to asphyxiate her. Mary remembered going in and out of consciousness and truly fearing she would be choked to death, which might have happened had her father not intervened and pried her away from her mother's grip.

Unfortunately, Mary's relationship with her father offered very little consolation for her mother's unpredictable behavior and emotional neglect. He was a prominent, successful surgeon and researcher who had made significant contributions to the advancement of medical science. Fully immersed in his work, he spent most of his time away from the family. According to Mary, when her father was at home, he was mostly tired and irritable and he ruled the household in a dictatorial fashion, armed with an explosive temper. She described him as "powerful, absent, and frightening."

It wasn't until Mary was in her early twenties that she really became aware that she might be suffering from some kind of psychological condition. Up to that point, she had always regarded her experience of life as somewhat normal. It was all that she knew and she had little basis for comparison. Mary recounted an incident from her college years where she came to the shocking realization that something might be wrong with her.

Prior to her deciding to pursue a career in the arts, she had attended the University of Wisconsin-Madison in their undergraduate psychology program. She was working with a professor who was conducting behavioral studies using chickens. Coincidentally, they were performing their research at the same time and in the vicinity of

Harry Harlow's work at the university. At that time, he was near the end of his well known experiments with rhesus monkeys on maternal separation, dependency needs, and social isolation (Coe, 2013). One day, Mary's professor brought her into Harlow's primate lab so that she could witness his experiments first-hand. It was there that she had her epiphany. It was as if the very sight of Harlow's monkeys rocking and clinging to their wire and cloth-covered surrogate mothers in an obvious state of duress stirred up something deep inside of Mary's core. "That was the moment that I knew there was something different about me that I didn't fully understand," she reflected. Mary was experiencing a disturbing sense of empathy for what those primates were feeling. The irony of Harlow's experimental findings regarding the importance of maternal contact on proper development came crashing down on her like a tsunami (Coe, 2013). From that day forward, her view on life would never be the same.

As Mary evolved into young adulthood, so did her neurosis. Her dysfunctional relationships with her parents continued to plague her as she grew. "Every step towards independence was seen as a betrayal," she explained. The persistent guilt and shame that she carried with her spilled over into every decision and relationship in her life. Eventually she met and married her first husband who, according to Mary, was as "narcissistic and controlling" as her father. She bore three children from that marriage before it crumbled into bitter divorce years later. With no healthy example of motherhood to emulate, Mary encountered many difficulties in raising her own children. "It was strange. Somehow I was able to give love to my children, but I had no idea how to receive it," she elaborated. It was as if she knew what she was supposed to *do* as a mother, but was unsure about how she should *feel* about it.

Life went on and Mary did her best to manage the challenges brought about by her rocky marriage, raising her children, and developing her career. Her symptoms of depression, anxiety, and disassociation seemed to be increasing in intensity as if they were the prelude to some inevitable crescendo in the overture of her existence. In particular, she felt more powerless than ever over her feelings of rage towards her mother. Mary's dark past followed her wherever she went, like it had been tethered to her back by a short cord. She commented that she could always sense it, looming over her shoulder, sneaking up on her when she least expected it. It wasn't until the turn of the millennium, with the passing of her father that she first

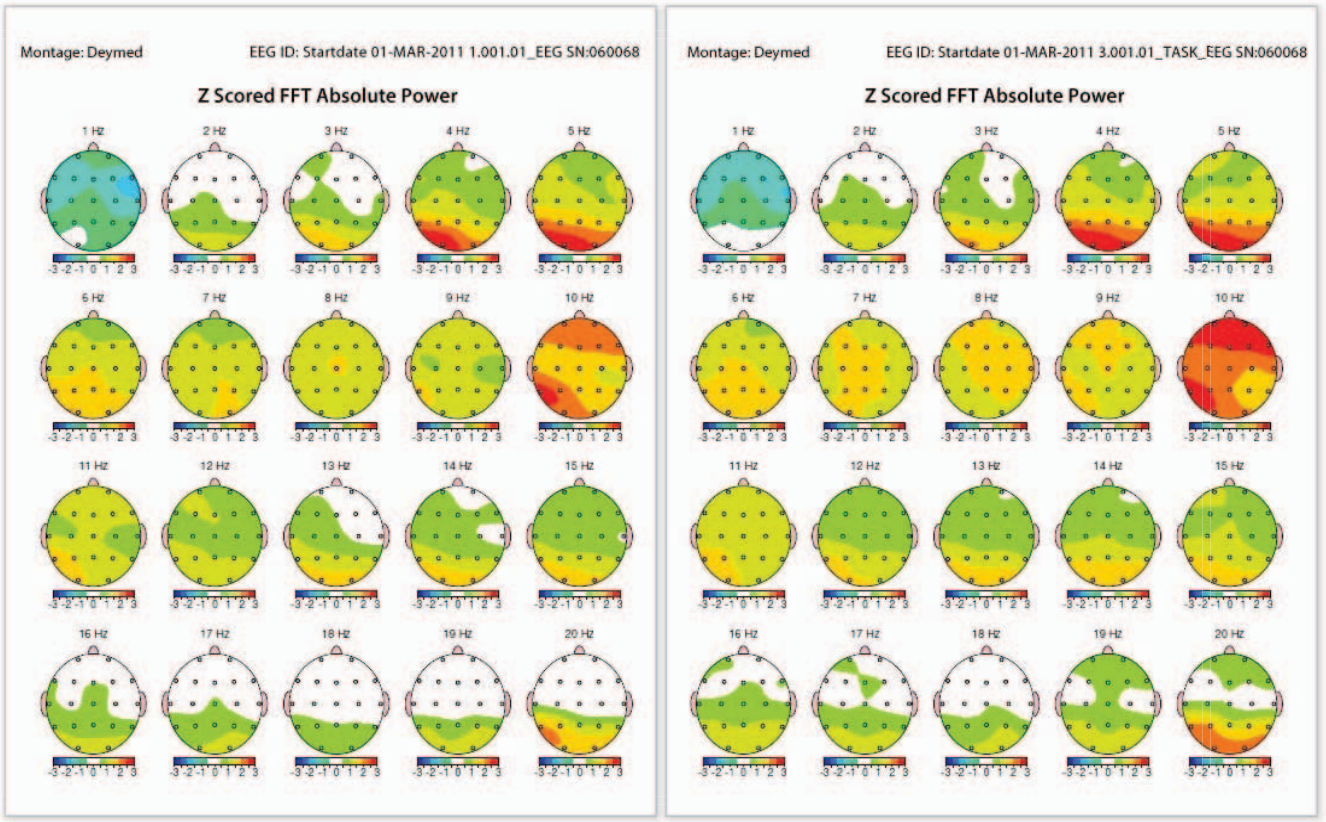


Figure 1: *A comparison of Mary's pre-treatment qEEGs for Absolute Power. The left page was recorded in a conventional eyes-closed relaxed state. The page on the right was recorded minutes later in a "symptomatic" experimental condition.*

decided to seek psychological treatment for her ongoing issues.

With her torrid history fully exposed and all of her cards laid out on the table like an open-handed game of poker, the onus was now on us to try to help Mary find some reprieve from her conundrum. Naturally, the first step involved assessment and evaluation. We administered Beck short form inventories for depression and anxiety. Her self-report baseline measures revealed only a mild level of depression, barely exceeding the reference range. However, her anxiety inventory indicated a more moderate level of symptoms.

In order to avoid any assumptions regarding the accuracy of her previously given diagnosis of PTSD, we decided to perform a quantitative EEG (qEEG) to examine Mary's unique individual neurology before determining which neurofeedback treatment protocol would be most appropriate for her situation. Initially, we administered a standard 19 channel recording in a resting state for both the eyes closed and eyes open conditions.

We then took it a step further by implementing an experimental condition. For the third recording, Mary was to remain eyes closed, only this time she was instructed to conjure up the kinds of anxiety-like symptoms that she would typically feel when she was triggered. "That shouldn't be hard to do," she aptly replied to our request.

A double analysis was performed utilizing a reference database comparison for surface and LORETA regions of interest using a linked ears montage for all three conditions. The results were fairly concise. The surface maps for the eyes open condition showed very little significant deviation from the database as compared to the eyes closed recording, which did reveal elevated Z-scores in absolute power primarily in theta and alpha frequencies posterior at O1 and O2 as well as increases frontally, specifically at 10 Hz.

Interestingly, the experimental or "symptomatic" condition showed a virtually identical analysis to its non-symptomatic counterpart recorded minutes earlier, but with one key difference. Even though the same deviations occurred in the exact same locations in the same frequencies, the Z-scored values for the deviations in the experimental condition had all increased in their level of statistical significance (see figure 1). In other words, they had gotten worse. We concluded that the differential in severity between the two conditions was most likely indicative that those deviations were specifically related to her symptoms by virtue of the fact that the numerical increases in Z-scores mirrored the increases in her subjective experience of anxiety. Additionally, the LORETA analysis suggested that the source of the pattern of

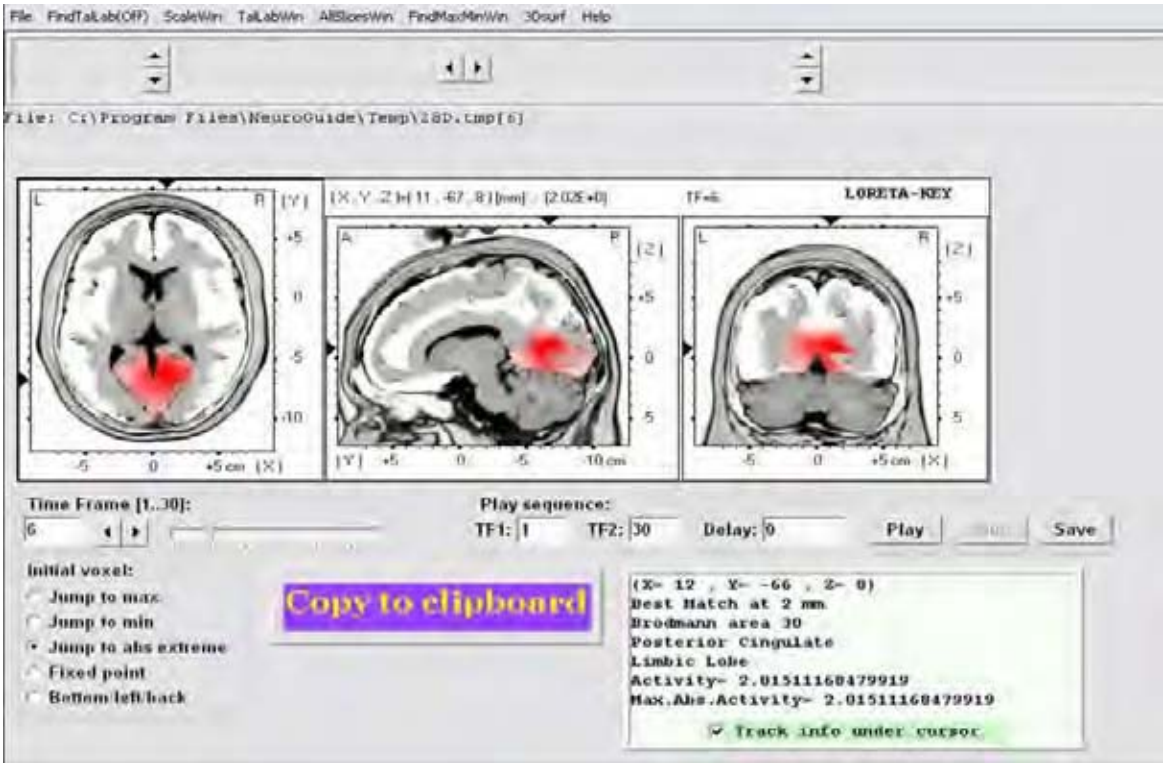


Figure 2: *Pre-treatment LORETA analysis indicating significant increased Theta activity of the Posterior Cingulate in the Limbic Lobe.*

neural dysregulation as depicted by the surface deviations was located in the limbic lobe, specifically in the posterior cingulate (see figure 2)

With our baseline measures behind us and her qEEG as our compass, we were ready to take aim and begin Mary's neurofeedback training process. All sessions began with five minutes of heart rate variability (HRV) training accompanied by a paced breathing audio track. The first neurofeedback protocol that we implemented was pretty simple. We used a standard amplitude training at occipital sites O1 and O2 using a bipolar montage to inhibit theta and alpha. Due to the fact that Mary's qEEG specified greater statistical significance during the eyes closed condition, we chose to administer all of her neurofeedback training with her eyes closed. Thus whenever Mary's alpha and theta amplitudes fell below the thresholds that we predetermined, music would begin to play. Likewise, if the amplitudes exceeded the thresholds, the music would cease to play. In all, forty sessions were administered. We tracked post-session amplitude averages for both alpha and theta frequency bands. If the training was successful, we expected to see a drop in the amplitude averages over time (see figure 3).

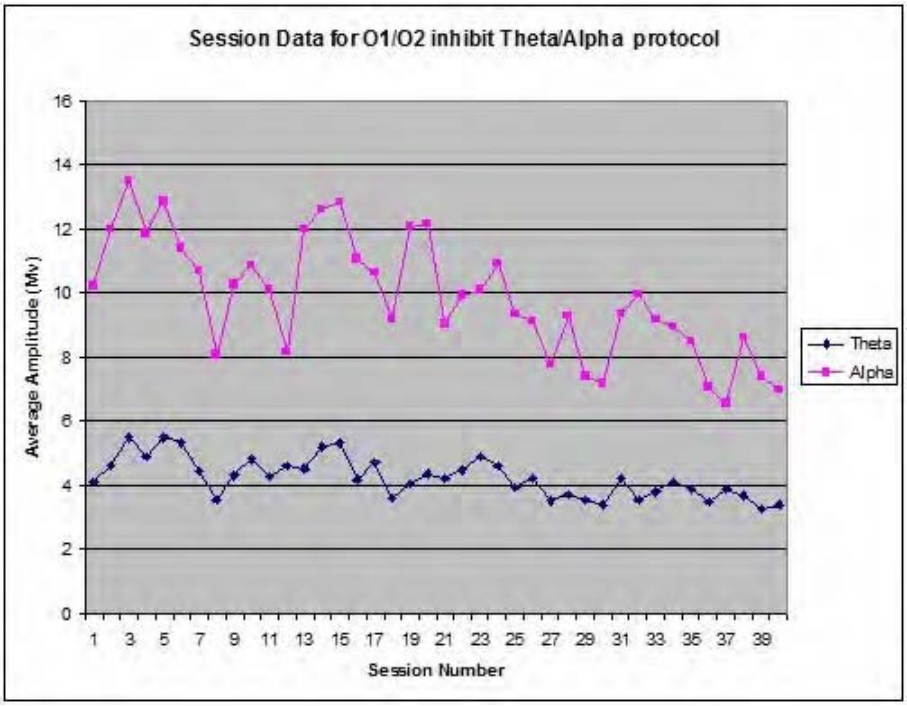


Figure 3: *Graph of post-session amplitude averages in Mv for Theta and Alpha across 40 sessions of neurofeedback training at sites O1 and O2.*

In addition to the standard amplitude training, we also utilized live Z-score training (LZT) software to regulate real-time surface Z-scores in various

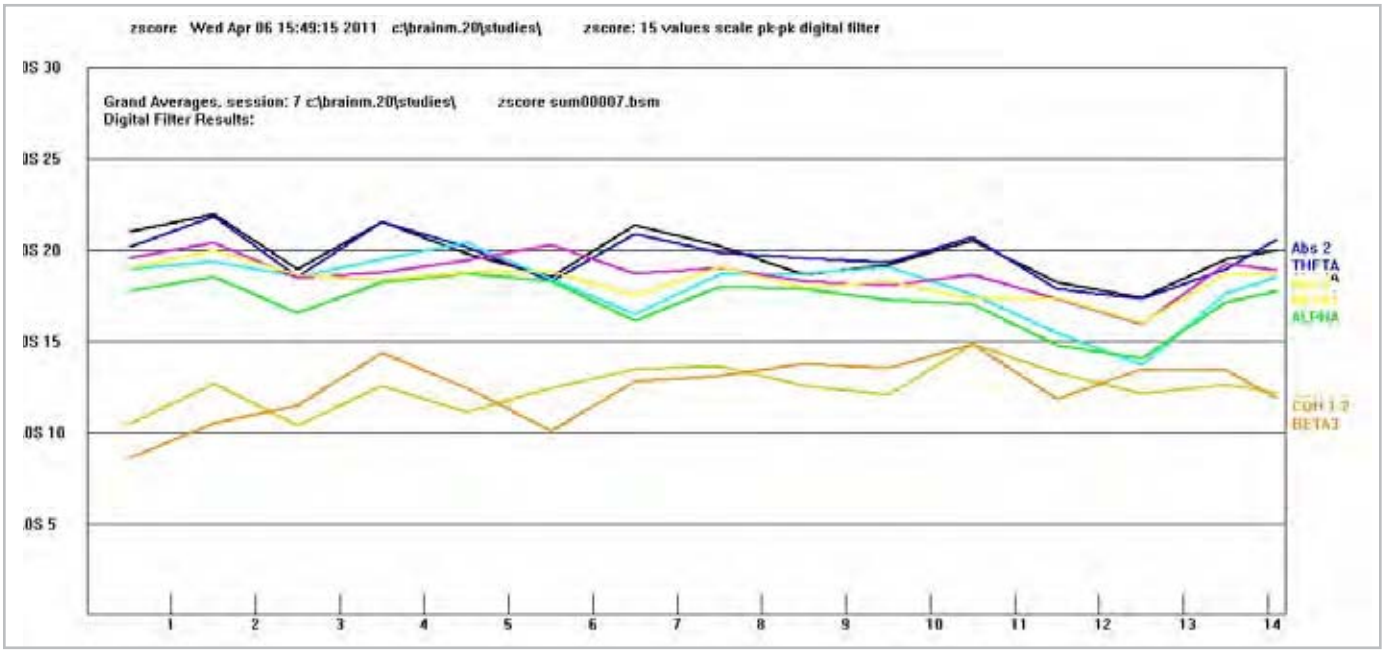


Figure 4: *An example of a performance graph of a Live Z-score Training session. Note as the amount of time of training increases, the Z-scores move closer to a value of 0.0SD.*

locations using 4-channel combinations consistent with Mary's qEEG data. We employed a typical PZOK protocol and trained values for absolute power, relative power, power ratios, asymmetry, coherence, and phase lag. Again, all training was done with the eyes closed using music as the feedback module. Performance was assessed at the end of each protocol run by reviewing session graphs to evaluate progress both within and between trainings. Productive training was determined by the average movement of specific Z-scores toward a standard deviation of zero (see figure 4).

From the beginning, Mary proved to be an exemplary trainee. She had a tenacious work ethic and caught on very quickly. An interesting phenomenon occurred in the early stages of her treatment that I still have no plausible explanation for. Somewhere around her sixth or seventh session, Mary informed me that she had been noticing something peculiar during her training sessions.

While training with her eyes closed, Mary observed that every time the music would play loud and clear, she would see a distinct hue of the color purple. "You probably think I'm crazy, but I had to say something," she exclaimed. "Well, there's an easy way to find out," I replied. I instructed Mary to close her eyes while I ran the protocol, but this time, I turned off the sound so that she would not receive any feedback. I then asked her to verbalize to me every time that she noticed the presence of the color purple while I was monitoring her EEG to determine if her experience of the visual sensation actually coincided with a decrease in her alpha and theta amplitudes. Within minutes, Mary began notifying me every time she saw purple. "Okay, it's there now…and now it's gone…it's back again."

As the scenario unfolded, I felt a strange sense of déjà vu come over me. I began to become aware of the parallel between what we were doing and the very first neurofeedback experiment back in 1958 at the University of Chicago in the laboratory of the legendary Dr. Joe Kamiya. And, as was the case with Kamiya's grad student, Mary was able to identify the presence or absence of alpha (and in this case theta too) with astounding accuracy (Kamiya, 1968). Her

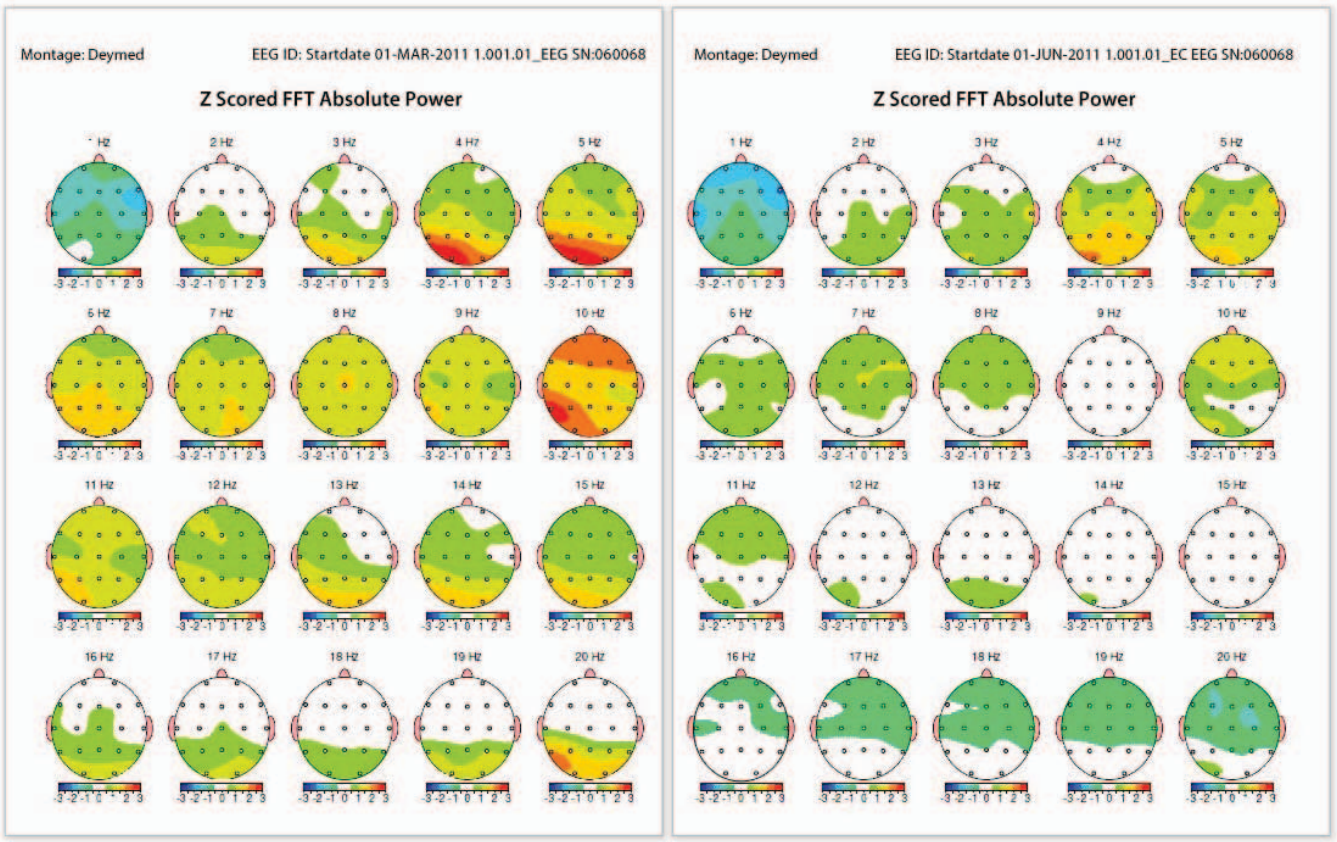


Figure 5: *A comparison of Mary's qEEG after 3 months of treatment (right side) to her pre-treatment baseline (left side). Significant decreases in Z-scores for Absolute Power can be observed.*

predictions were spot on. Every time she indicated seeing purple, the amplitudes diminished. Likewise, when the visual sensation disappeared, the amplitudes became excessive again. Aside from the validation that Mary was not, in fact, crazy, this discovery empowered her to accelerate her results by essentially being able to emulate her training sessions at home using her unique internal cue as a kind of personalized neurofeedback system.

Mary's treatment regimen started at a more rigorous pace of three to four sessions a week and then tapered off as time went on. By the end of her treatment, she was coming in once every five to six weeks just for maintenance or "refresher" sessions. The positive effects of the training were rapid and abundant. Mary commented that the most significant changes in her symptoms occurred within the first four weeks. By the time she had reached three months of treatment, she was able to go for long stretches of days, and even weeks, without any triggers or dissociation. Her nightmares had ceased completely and, for the first time in her life, she was finally able to let go of the intense anger that she harbored towards her mother. "I feel as though my past is behind me now," she said with great pride and emotion.

Mary's psychotherapist corroborated the effectiveness of her neurofeedback treatment as well. She commented that they were finally able to progress in their therapeutic process and that she was more resilient overall. She also noted that Mary no longer questioned her decisions to the point of obsession and that she was better able to handle whatever challenges life presented her.

By all accounts it appeared that her treatment was a success, but it was the hard data that revealed the true nature of her progress.

By all accounts it appeared that her treatment was a success, but it was the hard data that revealed the true nature

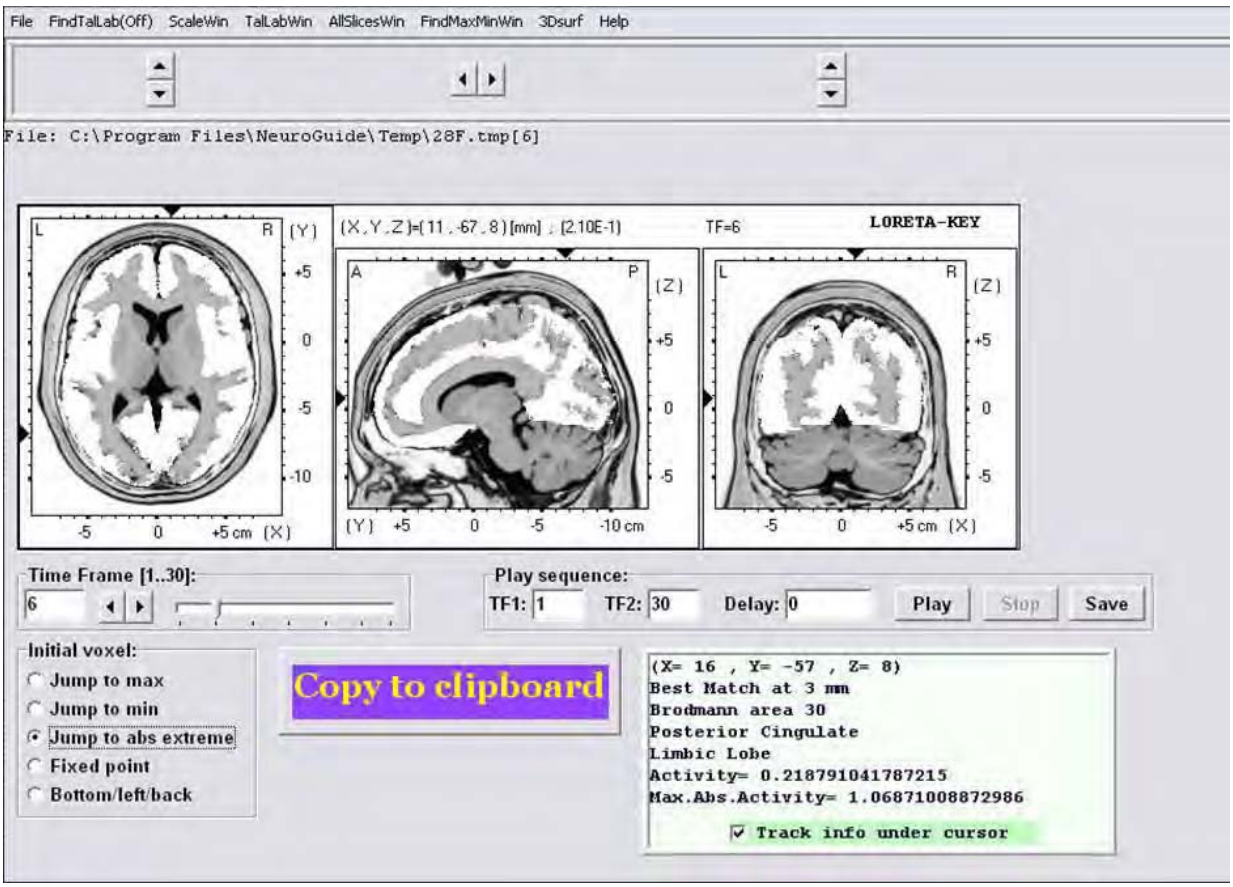


Figure 6: *Post-treatment LORETA scan, identical to the pre-treatment version, indicating the elimination of the previously seen significant increases in Theta activity for this region of interest.*

of her progress. Actually, her follow-up self report for depression showed only slight improvement, placing her barely below the range for mild depression, whereas her anxiety scale indicated a more appreciable reduction in symptoms. However her qEEG after three months of treatment illustrated a more impressive transformation, evidenced by the clear decreases in significant power values as compared to the database (see figure 5). In concordance with the surface data, LORETA analysis also showed a distinct pattern of normalization signifying major improvements from the baseline (see figure 6). Last but not least, a follow-up qEEG administered two years after the onset of treatment showed further remediation of Mary's neural dysregulation, suggesting continued efficacy even after the withdrawal of treatment (see figure 7).

Presently, Mary is still enjoying the fruits of her labor. We spoke on the phone recently and she told me that she experiences life differently now and that she was working on some of her most creative pieces of her artistic career. I informed her that her case was going to be published in *NeuroConnections*. She was ecstatic and said that she wanted nothing more than for other people to be able to benefit from her experience. Before her treatment had concluded, Mary knitted me a pair of gloves as an expression of her appreciation. As the bitter cold of Chicago winter sets in yet again, my hands are warmed by those gloves and my heart is warmed by her story.

About the Author

Corey Feinberg, MA, Clinical Director of Neurofeedback at NeuroQuest, Ltd. specializes in qEEG-based brain imaging and neurofeedback. His experience includes treating individuals with conditions such as anxiety, depression, ADHD, stroke, Parkinson's, and traumatic brain injuries.

Corey's passion for collaborative care has led him to team with specialists from the fields of neurology, biochemistry and neuro-optometry. This synergy of treatment modalities has influenced Corey to adopt a more holistic view of healing the brain and body.

Additionally, he has broadly influenced the field of neurofeedback with his extensive technical knowledge through private consultation and advanced training for leading practitioners across the country.

Corey received his Bachelor's degree from the University of Illinois in Champaign-Urbana, and his Master's degree in psychology from National-Louis University in Chicago. He continues to study a range of hybrid disciplines pertaining to brain health and performance. You may reach him at neuroquest@gmail.com.

References

Coe, C. (2013). Primate Lab History. Harlow Center for Biological Psychology. Retrieved December 3, 2013, from http://psych.wisc.edu/primatelab/primatelabhistory.html.

Frankel, H. (2013). Background. The Story of How American Families and Organizations Rescued Children of the Holocaust. Retrieved December 3, 2013, from http://www.onethousandchildren.org/background.shtml.

Kamiya, J. (1968). Conscious control of brainwaves. Psychology Today, I, 56-60.

BCIA Continued from page 10

Licensure/credentialing/registration of health care professionals serves to show how the countries regulate the treatment of diagnosed medical or psychological disorders in accordance with BCIA's perspective.

Translation of the Exam

Will the exam be translated into other languages? This question has been raised many times over the past several years and the Board has carefully considered this issue. The exam is really not the place to start the conversation. Each item on the certification exam is sourced to one of the documents listed on the Core Reading Lists, specific to the certification program. If these sources are not translated into the appropriate language, international applicants won't be able to adequately prepare. The Board has carefully watched growth and interest in other countries to see if a structure develops where this translation of selected texts can be accommodated and used as the foundation for didactic training and further study. As technology advances, this problem may solve itself. Additionally, there have been translations of some of the major reading sources, which will support international students as they learn.

The current policy is that if a country should develop a small core group of licensed/credentialed health care professionals who can fulfill all the other certification requirements and who will approach the board with a serious request, the translation process will be seriously considered. Exam translation is a huge task and the process requires two steps: *(1)* Translate the items from English into the other language and *(2)* Using a different source, translate it back to English. This must be done to ensure the accuracy of the terms that are used and that the intent of the item was not significantly changed.

BCIA looks to the future where, not only will our certification be the gold standard in North America, but worldwide.

My Experience using PEER

Merlyn Hurd PhD, QEEGD, BCN Senior Fellow

Several years ago at ISNR, Dr. Daniel Hoffman presented on the use of the raw EEG, on which the qEEG is based, to define the medications that might prove to be beneficial to a client or contraindicated. This was exciting and fit with my other work. Sufferin and Emory had been researching the EEG and the impact of drugs on the EEG for 20-25 years at that point. My enthusiasm was dampened when I learned that they would only accept a request for such an analysis from a psychiatrist. Not having any psychiatrist with whom I worked who would entertain such an analysis, I waited and waited for some client and psychiatrist who would be willing to venture into this new land.

Case Study 1

Finally, I had a client who had suffered seizures from overdosing on alcohol and he wanted to be treated for both issues. The qEEG analysis from his previous clinician (in another state in 2008) noted the following: "QEEG analyses indicated decreased delta activity over frontal-central regions and increased beta over the vertex and parietal regions. Connectivity analyses indicated communication difficulties between left mid-temporal and pre-frontal, right frontal, and central regions. These findings support difficulties with relaxation, emotional regulation, irritability, heightened information processing, sleep, and memory." His 2009 map is shown in Figure 1.

Figure 1: *Eyes Closed (left), Eyes Open (right)*

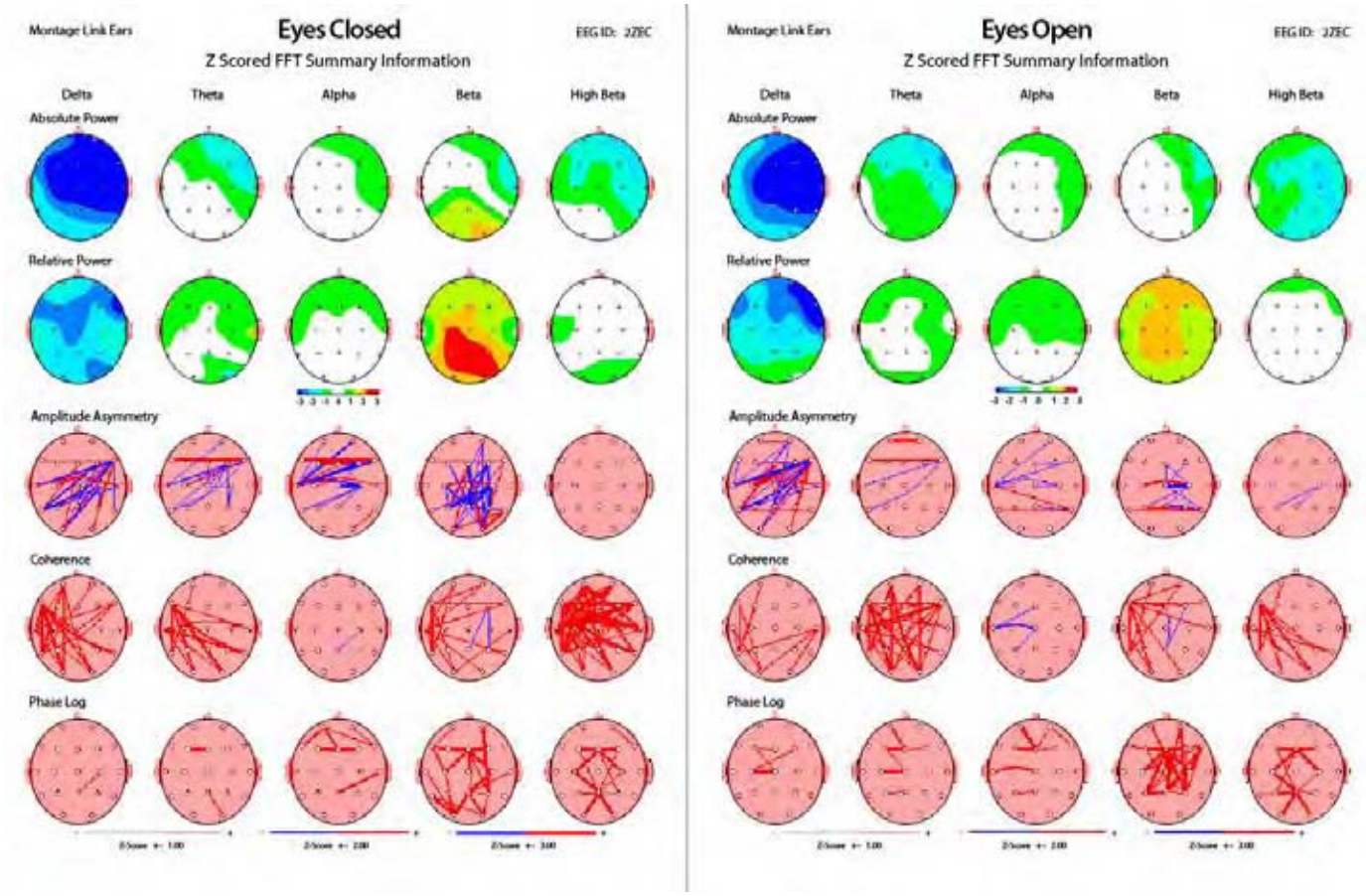
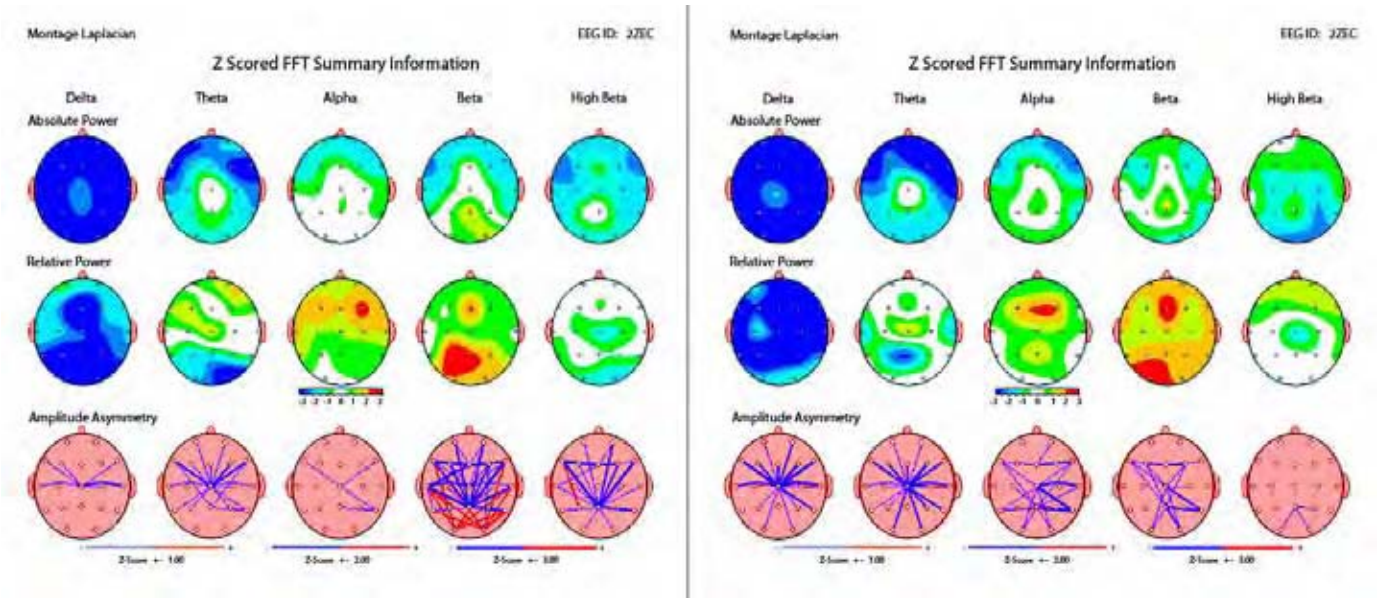


Figure 2: *The Laplacians, Eyes Closed with underactivity in Delta (left) Eyes Opened with underactivity in Delta and Theta (right)*



The underactivity in the delta in both referential and Laplacian analysis certainly looked familiar for a client who had depression issues. Even more so, the dysregulation in coherence in the left hemisphere was even more pronounced than had been seen in the previous year's qEEG analysis. We began a three times a week series of training with the seizures never surfacing again . The depression however, did not lessen and seemed to become more profound. His psychiatrist was urging more and more powerful drugs and I also wanted more information on what was going on. The psychiatrist finally recommended Lithium, at which the client resisted. At this point all agreed to have CNSResponse (now called PEER) to conduct an analysis against their database and see what drugs might help. I sent the raw eyes closed and eyes open data, and within a few days a report was generated; the psychiatrists (three) from CNSResponse held a two-way conference call with the psychiatrist and myself. Interestingly, the medication that was found to be the most resistant, meaning , the one he should not take, was Lithium. The ones that would be the most useful were anti-convulsant medication. He began taking those and there was some lift in his depression. The psychiatrist and I were urging him to have another complete physical because it seemed something else was going on. He had a physical and Lyme Disease was diagnosed. I referred him to a well known Lyme doctor in CT who began appropriate treatment with him while he continued with neurofeedback training. At this year's Thanksgiving dinner , a friend of ours had met the client in another city by chance and somehow my name came up. The client claimed he was healthy today and the "saving of his life" was due to the actions we took in 2009.

Several years ago at ISNR, Dr. Daniel Hoffman presented on the use of the raw EEG, on which the qEEG is based, to define the medications that might prove to be beneficial to a client or contraindicated.

Case Study 2

The second client is a young seven-year-old boy who has the methylenetetrahydrofolate reductase gene (MTHFR gene). He has trouble with math, spelling , writing, impulsivity, attention issues, and mood issues. He is bright and works hard at whatever is presented for him to prefom. He is an avid reader. The private school he attends suggested in July that he be put on Ritalin, because he was not staying in his seat, he was distrubing other children, and was implusive. The mother was resistant while the father was in favor of the medication. They are divorced and there is trauma between the mother, father, and child that has been viewed as contributing to the issues with the child. Several other types

Figure 3: *Eyes Closed Delta underactive (left) Eyes opened Delta underactive(right)*

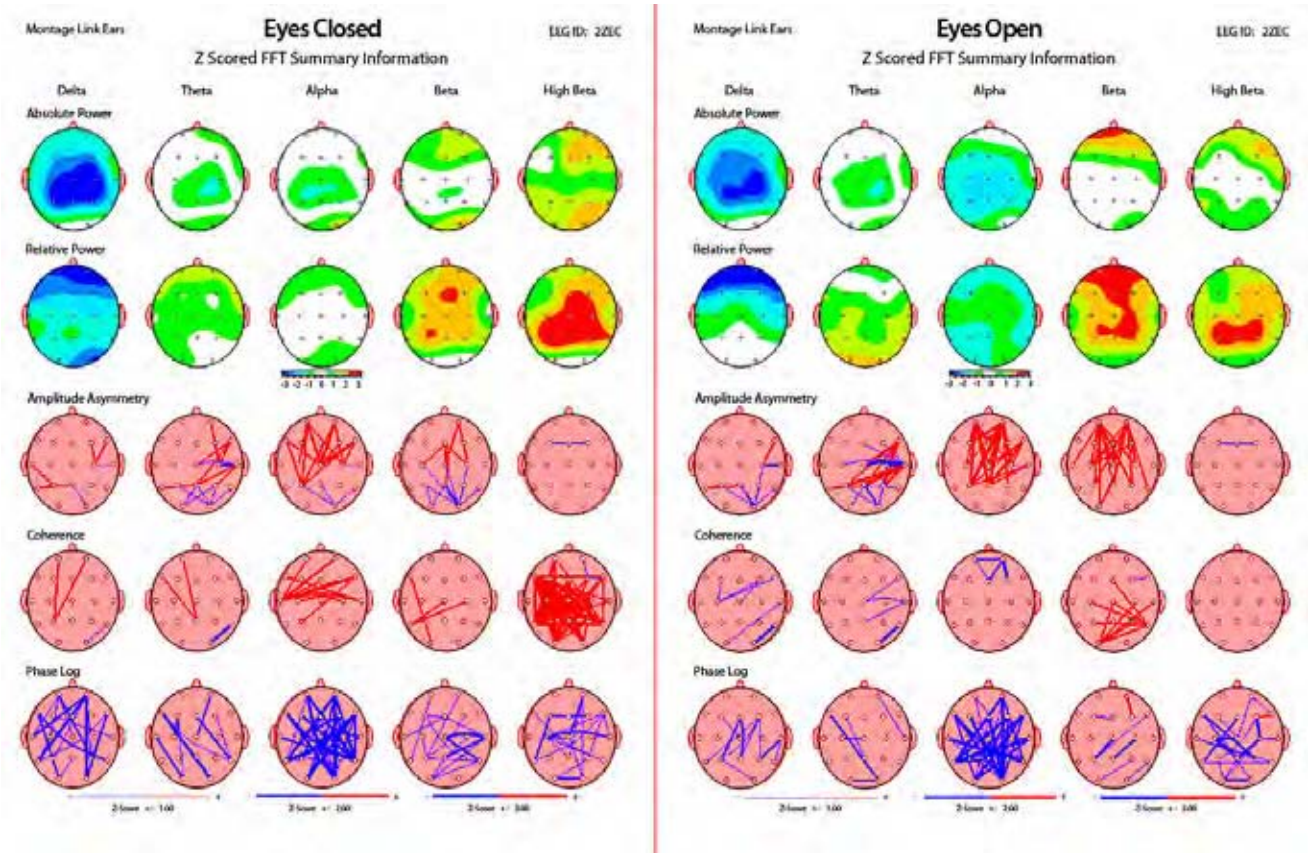
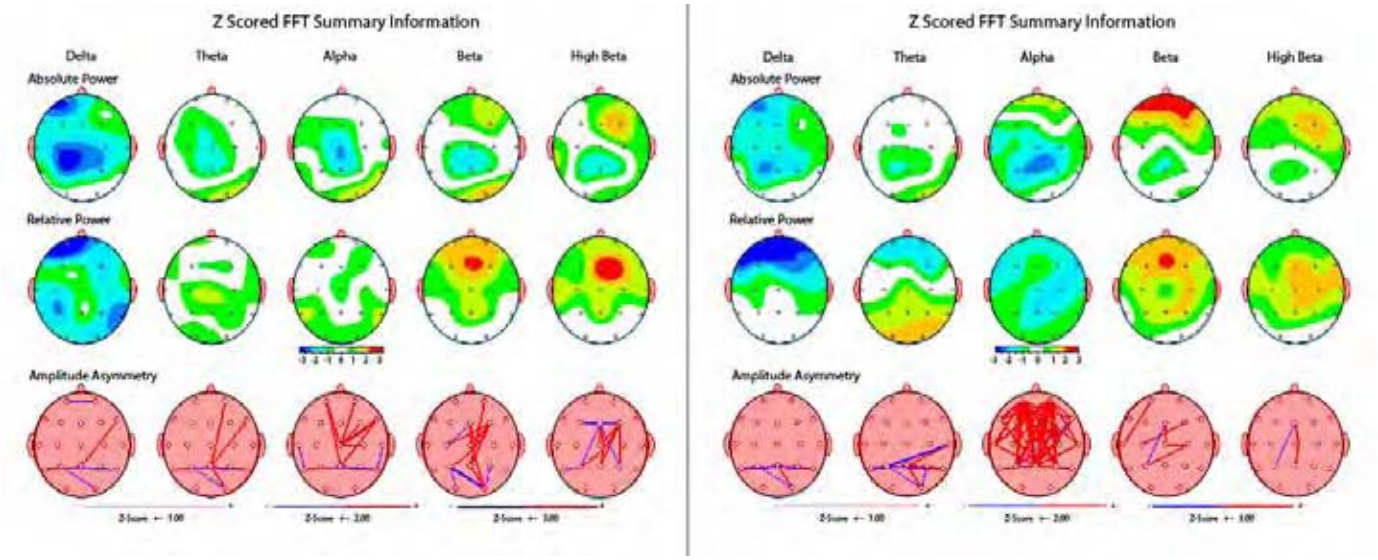


Figure 4: *Laplacian, eyes closed less Delta issue (left) eyes opened less Delta issue more Beta overactivity (right)*

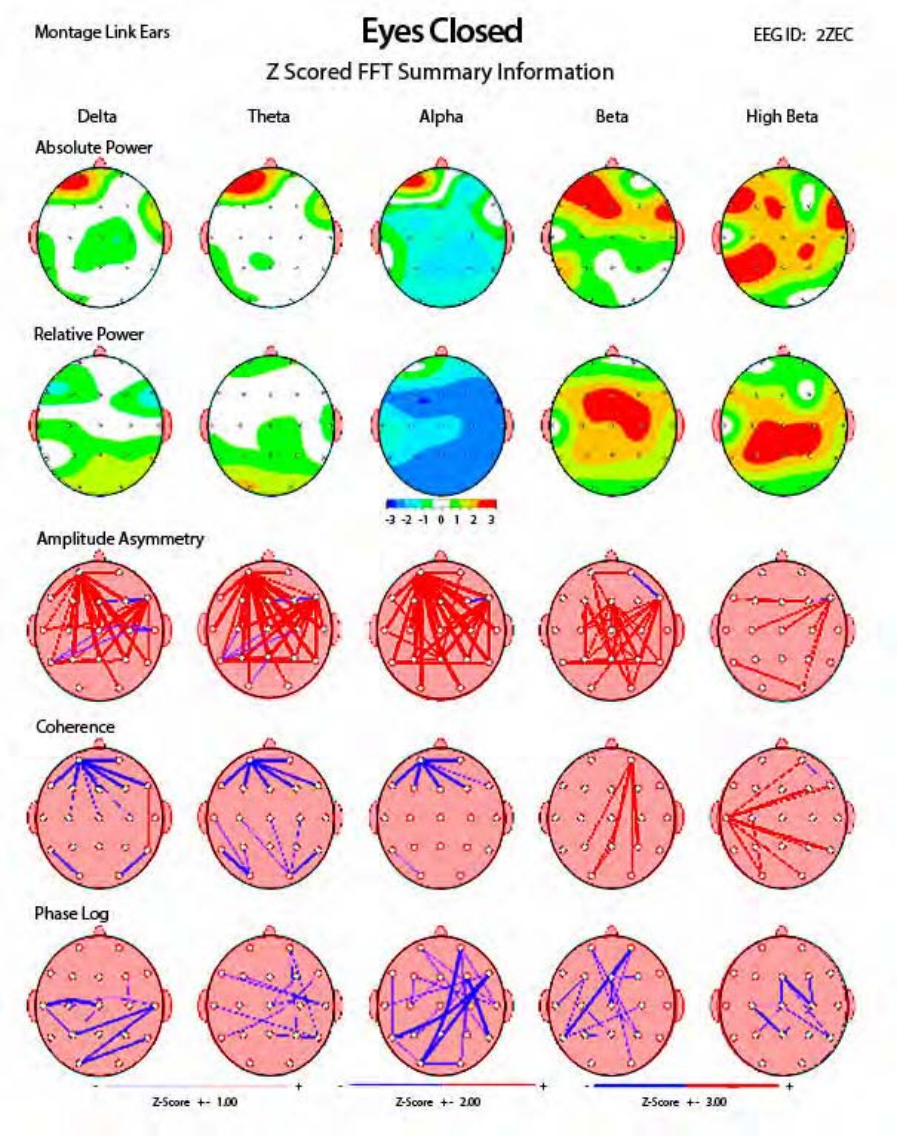


of treatment have been and are being used by the family, such as ILS (integrated listening system); a crawling program; and brain integration programs. The neurofeedback started in March with a two-times-a-week training, except for interruptions twice per month when the child was with the father, who did not think neurofeedback was useful. Then, there was a hiatus in August while the father took him to his country for a vacation. Training resumed in September for once–a-week on Saturdays which came down to twice-a-month in reality. During this time the school informed the parents they should be looking for another school since he " still does not sit still and interrupts the other children too much.". At this point, the mother entertained the idea of medication and the psychiatrist agreed to have PEER analyze the data for an indication of the appropriate medication. The findings were that stimulants were contraindicated and perhaps antidepression medication would be helpful. The changes in the qEEG showed a reduction in the underactivity in delta and some changes in high beta.

12/14/2013: Figure 5 map shows FP1 artifact that could not be removed. If one takes out the FP1 Information then the changes are quite dramatic The issue is still in the high beta, especially in the P3 and parietal areas which relate to his academic symptoms and impulsivity.

Because the mother is so worried about the child being terminated from the school , in January she started the child on focalin and one can only wait and see what happens. I have tried to help her to understand the possible side effects. Oddly enough, the father now does not want his child on medication, even though he still does not accept the changes from the neurofeedback training.

Figure 5: *12/14/2013 FP! artifact*



At the end of January 2014 the mother called to say he had a green tinge to his skin and so she was stopping the focalin.

I support the use of PEER analysis and hope that more clinicians will use their services.

Z-Score LORETA Neurofeedback as a Potential Therapy in Depression and Anxiety

J. Lucas Koberda, MD
Students: Paula Koberda, Andrew Moses, Jessica Winslow, Andrew Bienkiewicz, Laura Koberda

Major depressive disorder (MDD) is a common mental illness affecting approximately 2.5% of the general population. MDD is one of the leading causes of disability; it is projected to have the second highest burden of disease (measured in disability-adjusted life years) by 2020 (WHO). MDD has negative social consequences in terms of reduced employment and psychosocial impairment (Anderson et al. 2011). The pathophysiology of depression involves both external social stressors and internal genetic vulnerability.

Anxiety disorders are among the most common of all mental disorders (Kessler et al. 2005). The diagnostic and statistical manual of mental disorders (DSM-IV-TR)[2] includes generalized anxiety disorder (GAD; a chronic form of anxiety characterized by excessive, uncontrollable worry), panic disorder (PD; with recurrent, unexpected paroxysms of anxiety, somatic and autonomic symptoms and fear), phobic disorders [e.g., specific phobias, agoraphobia, social phobia (SP)], post-traumatic stress disorder (PTSD; characterized by unwanted, intrusive remembrances—as daytime thoughts and night-time dreams and nightmares—and avoidance of activities and other cues associated with prior life-threatening trauma) and obsessive-compulsive disorder (OCD; with recurrent obsessions and compulsions in this category.

In addition to psychotherapy and pharmacotherapy, other noninvasive modalities including neurofeedback (NFB), repetitive transcranial Magnetic Stimulation (rTMS) and transcranial Direct Current Stimulation (tDCS) have been found effective in the treatment of these conditions (Alonzo et al. 2013, Stevens, 2014). TMS has also been approved by the FDA for the treatment of medically resistant depression (Stevens, 2014). In addition to noninvasive modalities of the treatment of depression and anxiety, other invasive techniques were introduced as potential therapy including Vagus Nerve Stimulation (VNS) and Deep Brain Stimulation (DBS). DBS, especially with Subcallosal Cingulate (Brodmann's area 25), has been reported to be beneficial in depression cases resistant to conventional medical therapy (Riva-Posse et al. 2013 and Schlaepfer et al. 2013). Unfortunately, any invasive procedure carries a risk of potential complications and side effects including bleeding, infection and misplacement of an active electrode (Williams and Okun 2013). NFB (in contrast to the above invasive methods) is not associated with any major side effects or intrusive methodology and is relatively inexpensive. It has also been underestimated in the clinical arena as a potential therapeutic modality. Standard one- or two-electrode NFB has been reported as beneficial in relieving depression symptoms in several investigations, including a randomized controlled study (Choi et al. 2011).

In z-score NFB, a real-time comparison to an age-matched population of healthy subjects is used for data acquisition, simplifying protocol generation, and allowing clinicians to target modules and hubs that indicate dysregulation and instability in networks related to symptoms (Thatcher, 2013). Z-score NFB increases specificity in operant conditioning, providing a guide that links extreme z-score outliers to symptoms, and then reinforcing z-score shifts toward states of greater homeostasis and stability. The goal is increased efficiency of information processing in brain networks related to the patient's symptoms (Thatcher, 2013).

A recently introduced method called Low Resolution Electromagnetic Tomography (LORETA) z-score NFB is capable of targeting specific dysregulated anatomical structures, many of which are in deeper cortical locations (Koberda et al., 2013; Thatcher, 2013). For example, the Insula and Anterior Cingulate has been identified as potential NFB target sites to improve pain control in patients who display electrical dysregulation of these areas (Koberda et al. 2013).

Our neurology center conducted z-score LORETA NFB therapy of 31 patients with depression and associated anxiety. In addition to depression and anxiety, these patients frequently reported other coexisting problems like cognitive dysfunction, OCD, and/or chronic pain. Most patients were found to have qEEG abnormalities including alpha power increase, asymmetry, and/or LORETA electrical dysregulation in frontal areas (Figure 1).

Figure 2 shows LORETA images before (top image) and after (lower image)

completion of 15 sessions of z-score LORETA NFB of a 15-year-old female who suffered from extreme anxiety before and during horse riding competitions. Her symptoms included anxiety with palpitations, frequently associated with nausea and vomiting. Marked dysregulation of the Anterior Cingulate Subcallosal region-Brodmann's Area (BA) 25 was identified during the pre-NFB LORETA testing (Fig 2) manifesting as increased beta activity. Following 15 NFB sessions, this electrical dysregulation was corrected; as seen on the lower portion of the figure 2.

Detailed analysis of our patients diagnosed with depression and/or anxiety showed that out of 31 included in the study, 24 (77%) were found to have both subjective and objective (improvement of qEEG abnormalities) improvement of the symptoms within 10 sessions of LORETA z-score NFB. I would like to focus on just one of our representative patients who successfully completed z-score LORETA NFB with marked improvement in both depression and cognitive function. Cognitive function (which is often impaired in patients with depression) usually improves after NFB therapy.

The following report is a 40-year-old female who was previously treated for major depression and did not respond to pharmacological treatment. Prior to neurofeedback, she was treated with Electro-Convulsive Therapy (ECT), which was not successful in relieving her depression. Instead, the individual sustained major memory impairment and visual-spatial difficulties. Since she was not responding to conventional therapy, her psychiatrist referred her to my practice for NFB therapy. The patient's cognitive and depressive dysfunction caused inability to continue her employment as a pharmaceutical representative. Initial LORETA showed several areas of electrical dysregulation including (Figure 3A) BA-5 (secondary sensorimotor cortex), BA-9 (prefrontal cortex), and temporal cortex (Figure 3B).

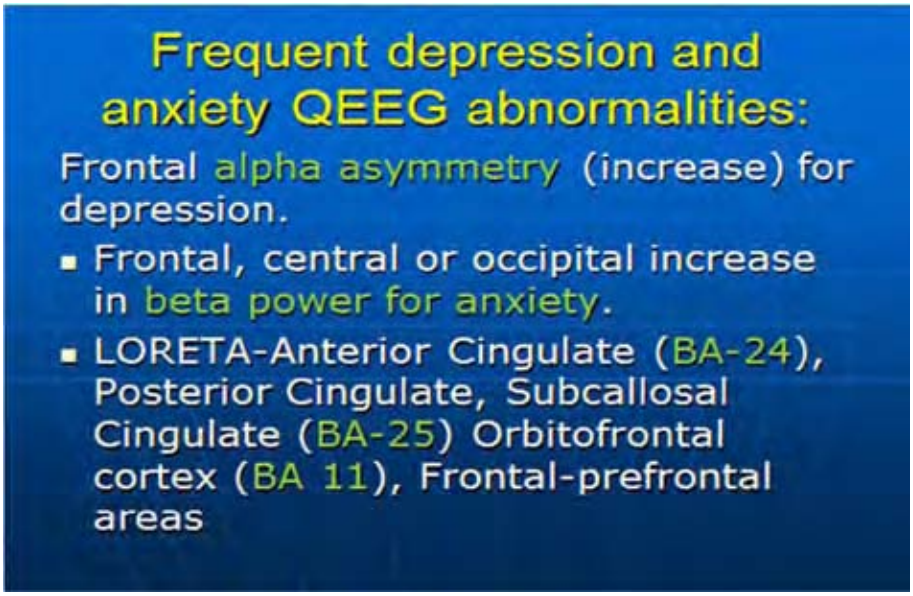


Figure 1: *Summary of frequently identified qEEG/LORETA abnormalities in patients suffering from depression and anxiety.*

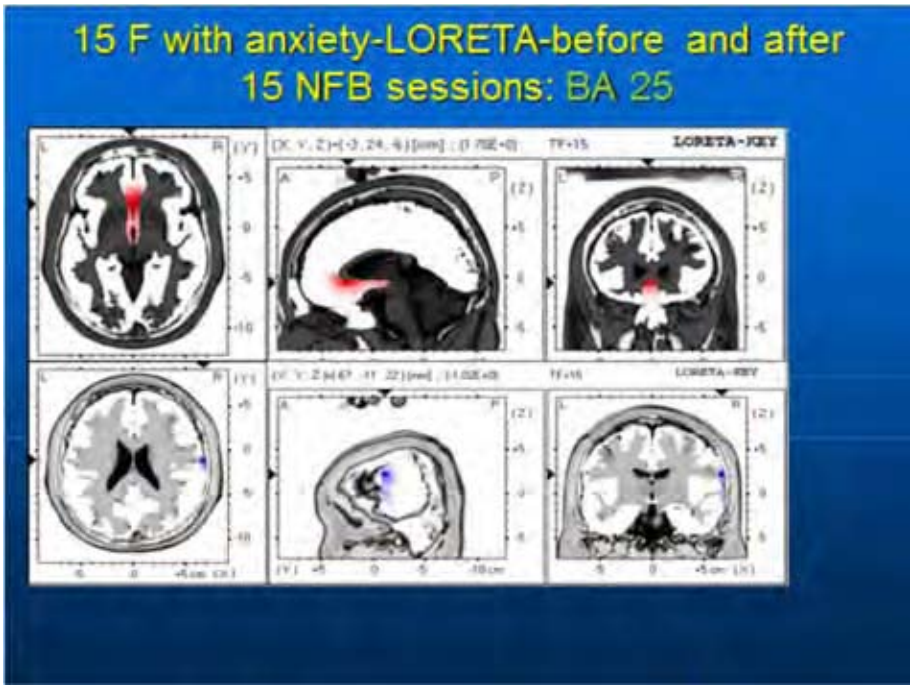


Figure 2: *15-year-old female suffering from anxiety. Upper part of the picture shows area of electrical dysregulation (area in red) in the Subcallosal Cingulate (BA-25) before NFB therapy. Lower part of the picture shows resolution of previously identified dysregulation after 15 sessions of NFB.*

Her computerized cognitive testing before NFB showed deficiency in memory, information processing speed and visual-spatial domains. After 10 sessions of z-score LORETA NFB, the patient reported major improvement

in her mood as well as mild memory improvement. Repeated computerized cognitive testing revealed marked improvement of previously deficient cognitive domains (Fig 4). Memory score increased from 85.4 to 102.8, information processing speed rose from 90.8 to 97.7 and visual-spatial domain went up from 80.8 to 100.7. Motor skills also demonstrated a one standard deviation gain in efficiency. In addition, post NFB LORETA showed an improvement of previously identified electrical dysregulation. After successful NFB therapy, the patient also was able to come back to gainful employment in the medical field (after few years of being unemployed).

This paper illustrates high effectiveness of z-score LORETA NFB therapy in complex neuropsychiatric patients, where an improvement of depression/anxiety and other associated cognitive

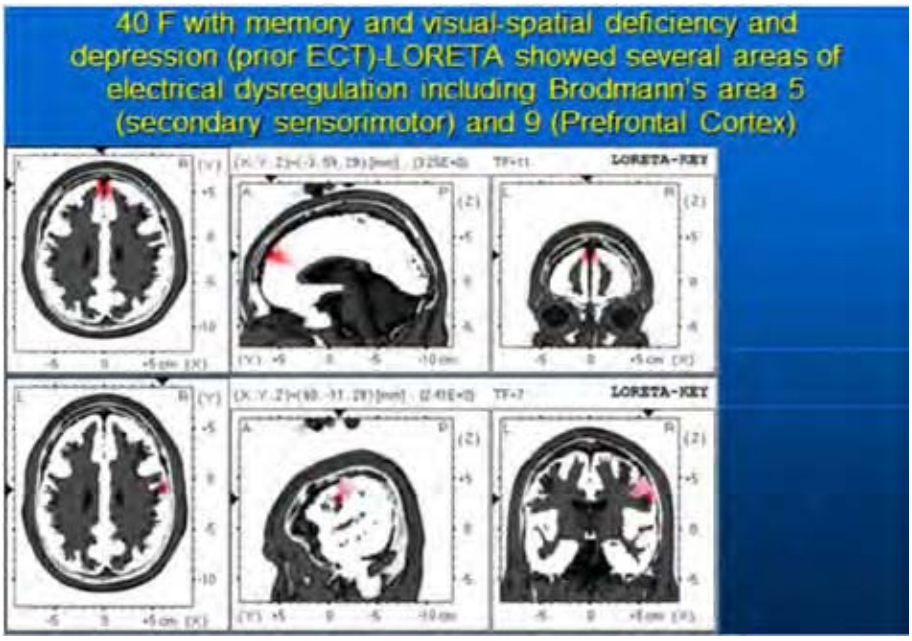


Figure 3A: *Pre-NFB LORETA of 40-year-old female diagnosed with depression associated with cognitive dysfunction. Areas of cortical dysregulation are shown in red. After 10 sessions of NFB marked improvement of previously identified LORETA abnormalities was noted.*

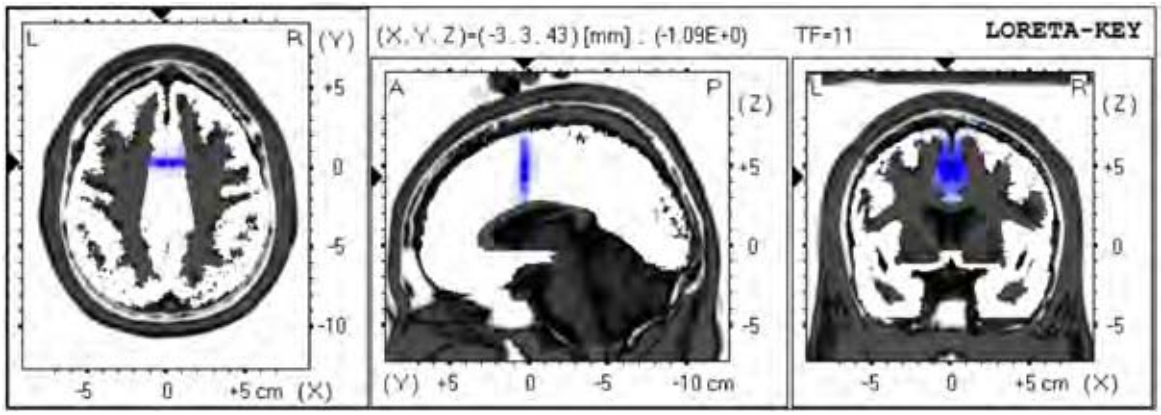
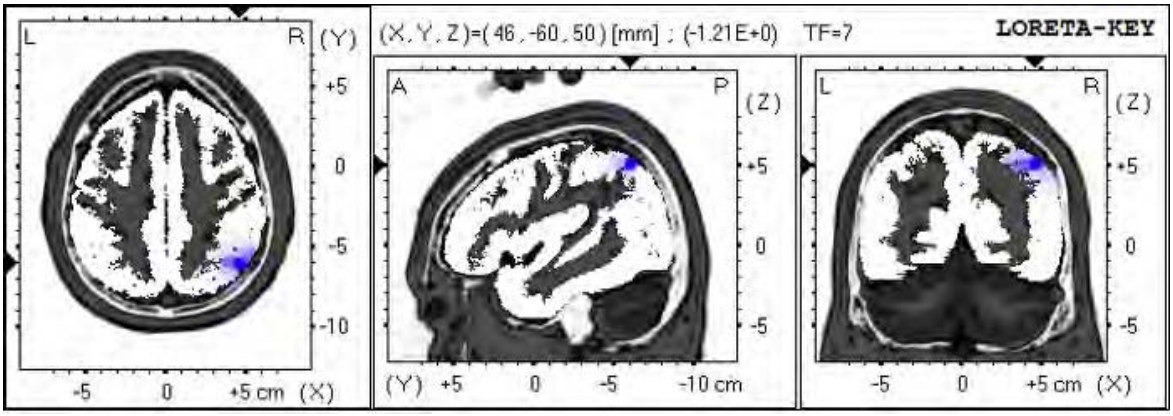


Figure 3B: *40-year-old female LORETA after NFB- shows resolution of previously electrically dysregulated BA-5 and BA-9.*



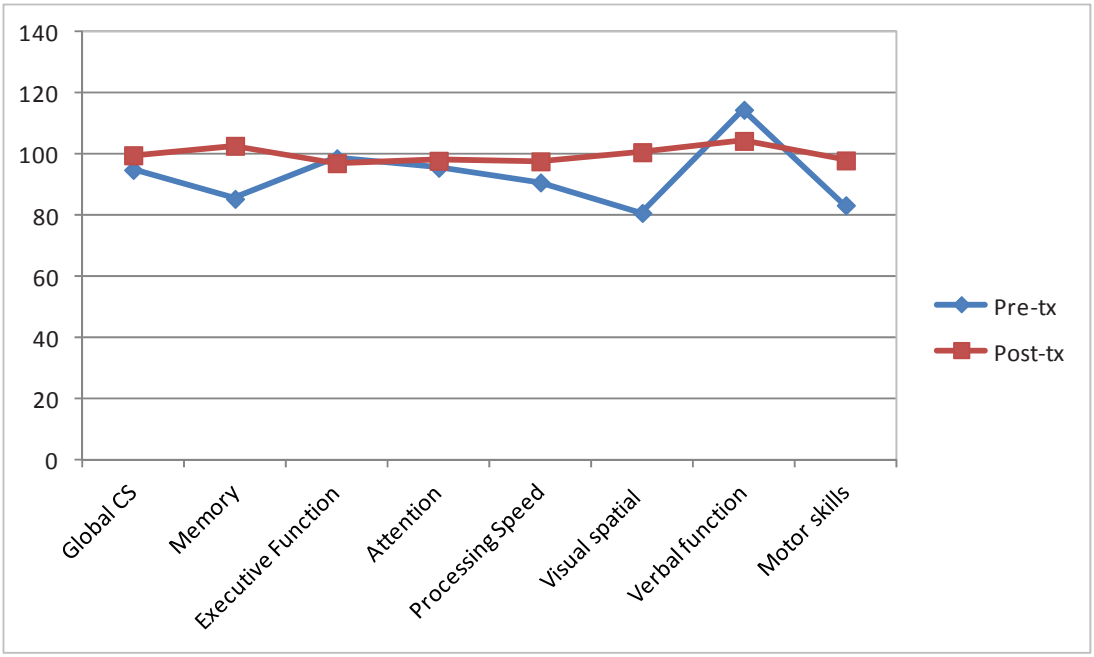


Figure 4: *The blue line shows scores from computerized cognitive testing before NFB. The red line shows scores after 10 sessions of NFB. Computerized cognitive testing (Neurotrax Corp, Bellaire TX) shows data based on comparison to age and education matched controls. Expected score is 100 with one standard deviation-15.*

domains can be achieved in most of the patients within just 10 treatment sessions. We also recommend implementation of qEEG/LORETA brain mapping testing in all patients suffering from depression, as well as anxiety. Since qEEG/LORETA NFB is a non-invasive technique and relatively inexpensive, it should be considered as the therapy of choice before other invasive modalities are contemplated.

About the author

Dr. J. Lucas Koberda is a board certified neurologist and an internationally trained physician who completed his residency in Neurology at the Oregon Health Sciences University in Portland, Oregon. Dr. Koberda is a director of the Tallahassee Neurobalance Center (www.TallahasseeNeuroBalanceCenter.com) and also affiliated with The Florida State University College of Medicine. His main interest is in neuro-psychiatry and cognitive enhancement. He uses the newest technology of qEEG and LORETA Z-score Neurofeedback to successfully diagnose and treat many medical conditions including seizures, headaches, fibromyalgia chronic pain, anxiety, depression, and prior stroke. Dr. Koberda has also effectively introduced neurofeedback protocols for a cognitive enhancement which may help students and professionals to improve their memory, concentration, verbal function, or information processing speed.

References

Alonzo A, Chan G, Martin D, Mitchell PB, Loo C. J Affect Disord. 2013 Aug 15; 150(1):91-5. doi: 10.1016/j.jad.2013.02.027. Epub 2013 Mar 15.Transcranial direct current stimulation (tDCS) for depression: analysis of response using a three-factor structure of the Montgomery-Åsberg depression rating scale.

Andersen I, Thielen K, Bech P, Nygaard E, Diderichsen F. Increasing prevalence of depression from 2000 to 2006. Scand J Public Health. 2011; 39:857–863. [PubMed]

Choi SW, Chi SE, Chung SY, Kim JW, Ahn CY, Kim HT.Is alpha wave neurofeedback effective with randomized clinical trials in depression? A pilot study. Neuropsychobiology. 2011.

Kessler RC, Chiu WT, Demler O, Merikangas KR, Walters EE. Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. Arch Gen Psychiatry. 2005; 62:617–627.

Koberda JL, Koberda P, Bienkiewicz A, Moses A, Koberda L. Pain Management Using 19-Electrode Z-Score LORETA Neurofeedback. Journal of Neurotherapy, 2013, 17:3, 179-190.

Riva-Posse P, Holtzheimer PE, Garlow SJ, Mayberg HS. Practical considerations in the development and refinement of subcallosal cingulate white matter deep brain stimulation for treatment-resistant depression. World Neurosurg. 2013 Sep-Oct; 80(3-4):S27.e25-34. doi: 10.1016/j.wneu.2012.11.074. Epub 2012 Dec 13.

Schlaepfer TE, Bewernick BH. Handb Clin Neurol. 2013; 116:235-43. doi: 10.1016/B978-0-444-53497-2.00018-8. Deep brain stimulation for major depression.

Stevens E. Treating Mood Disorders-Vlinical Neurotherapy-Applications and Techniques for Treatment. Edited by Cantor and Evans. Elsevier. 2014.

Thatcher, R. W. (2013). Latest developments in live z-score training: Symptom checklist, phase reset, and LORETA z-score biofeedback. Journal of Neurotherapy, 17, 69–87.

Williams NR, Okun MS. J Clin Invest. 2013 Nov 1; 123(11):4546-56. doi: 10.1172/JCI68341. Epub 2013 Nov 1. Deep brain stimulation (DBS) at the interface of neurology and psychiatry.

World Health Organization. Information on mental disorders management: depression. Geneva: World Health Organization. Available from: http://www.who.int/mental_health/management/depression/en/

Neurofeedback Treatments Enable the EEG-Normalization and Total Seizure Control of Epilepsy—A Case Study

Rivi Sela, MSW, Meirav Shaked-Toledano, MSW

Abstract:

Over the past 40 years, researchers have examined various non-drug approaches to the treatment of epilepsy. Neurofeedback is an approach that has been proven efficient in reducing seizure frequency. Our experience in treating children and adolescents with epilepsy shows that qEEG-guided amplitude training enables one to reach total elimination of seizure activity in a relatively short time (3–5 months of training). In addition, our patients' EEG, which before treatment included numerous appearances of spike and wave complexes, was sampled again towards the end of treatment and was found to be normal (with no spikes and discharges). This article presents two cases of epilepsy patients who were treated with qEEG-guided neurofeedback at our clinic. Both of them experienced total cessation of seizures at an early stage in the treatment and displayed substantial behavioral and cognitive improvement during the course of treatment. Both patients displayed a normal EEG at the end of the treatment.

The cases described in this article lend support to the assumption that neurofeedback treatments enable the EEG-normalization and total seizure control of epilepsy patients who do not respond (or only partially respond) to anti-convulsant medicines. Many of these patients do not have an epileptic focus, and therefore are not considered suitable candidates for neurosurgery. Other alternatives are expensive and have low efficiency. These results lend support to the idea that tailoring the neurofeedback treatment protocol specifically to each individual patient by doing qEEG tests improves treatment quality and precision in a way which enables the achievement of full control over seizures and full EEG normalization.

The cases described in this article lend support to the assumption that neurofeedback treatments enable the EEG-normalization and total seizure control of epilepsy patients who do not respond (or only partially respond) to anti-convulsant medicines.

Introduction:

Epilepsy is a seizure disorder characterized by abnormal electrical brain activity. According to the World Health Organization, about 50 million people around the world suffer from epilepsy, which amounts to 0.8% of the world population. Epilepsy has traditionally been treated with anticonvulsant drugs. However, about one third of patients do not respond to medication and are unable to control their seizures2,3. Their situation is often complicated by the drugs" aversive side effects, which are detrimental to their health. Also, women who want to become pregnant are warned against severe harm that might be caused to their fetus by the drugs. In several cases in which seizures are not controlled by medication, patients might undergo neurosurgery, whereby specific neural pathways are severed to prevent extensive epileptic activity. Seizures are caused by abnormal, excessive electrical activity in the brain. The pattern is one of oversynchronization of neuronal activity[5].

In many cases, the cause of the disorder is not clear, but its onset sometimes follows the appearance of brain cancer, brain trauma, or stroke. Abuse of alcohol and/or drugs might also be a factor[1].

Another option for treating epilepsy, aside of anti-convulsant medication or neurosurgery, is neurofeedback (or EEG Biofeedback), a non-medicinal method that has been gaining momentum and recognition over the past two decades, with high success rates in the treatment of epilepsy as well as other neuropsychiatric disorders (such as ADD/ADHD, learning disabilities, anxiety and more[6]). The method's success in reducing seizure frequency and intensity for epileptic patients is highly impressive, given the fact that most studies published have dealt with patients whose seizures were uncontrollable using orthodox medicines[7].

Neurofeedback

Neurofeedback is a form of neurotherapy that is based on the principle of operant conditioning. By this method, the brain is trained to normalize its own electrical activity through receipt of congruent feedback for its different EEG patterns (i.e. positive, reinforcing feedback for normative activity patterns

and negative, frustrating feedback for abnormal activity patterns). After a few trials, the brain starts to realize that there is a correlation between the kind of feedback it receives and the kind of EEG pattern that brings it about. Once such a realization takes place, the brain starts a slow, gradual process of changing its own activity patterns in order to avoid negative feedback and win more of the positive feedback. Such learning leads to the (partial or total) normalization of the EEG patterns. When the EEG activity starts to normalize, neuropsychiatric symptoms (which characterize non-normative EEG activity) start to diminish in size and in frequency.

One of the most common neurofeedback protocols is the up-training of Sensory Motor Rhythms (SMR; i.e. frequencies of 12-15 Hz over the somatosensory and motor strips of the brain). SMR up-training as an efficient treatment for epilepsy was first discovered in the mid-seventies by Prof. Barry Sterman of the UCLA School of Medicine[7]. Since then, neurofeedback as a whole has evolved in both form and scope, to include treatment of a host of other neuropsychiatric disorders, such as ADD/ADHD, learning disabilities, communication problems of the autistic spectrum, anxieties, depression and more. The method is widely researched in clinical and academic institutes worldwide, with high success rates reported for the treatment of various disorders.

History of neurofeedback treatment for epilepsy

Research in most institutes has focused on up-training of 12-15 Hz EEG waves (i.e. SMR) as a neurofeedback treatment protocol for epilepsy[8–10]. Most research studies report high success rates in reducing seizure frequency and magnitude for patients who do not respond to anti-convulsant drugs[10–11]. Sterman et al. have demonstrated for the first time the applicability of SMR up-training to four epileptic patients who, following treatment, experienced significant improvement in their ability to control their seizures.

Cott et al.found that 5 out of 7 patients experienced a reduction in the frequency of their previously uncontrolled seizures after three months of SMR up-training[12].

Kaplan found clinical improvement in 80% of patients treated with neurofeedback[13]. Similarly, Finley et al. found reduction of seizures and normalization of EEG in a severely epileptic patient following SMR up-training[14].

Lantz and Sterman, in a large, double-blind controlled clinical trial on SMR up-training, found a 61% seizure reduction in the experimental group only[15].

Tan et al., in their meta-analysis of 10 carefully selected research studies which answer their criteria for inclusion, determined that SMR up-training consistently decreased seizure rate among severe cases of epilepsy, which could not otherwise be controlled[16]. Andrews & Schonfeld found that out of a sample of 83 patients, 80% managed to gain control over their seizures using SMR up-training neurofeedback protocol together with other methods of intervention (such as diaphragmatic breathing) [17].

Two different studies found that SMR up-training influences epileptic EEG during sleep also, when no conscious effort is done by the patient to control it: as SMR was up-trained, night-time epileptiform activity decreased[18,19].

In a review of research studies spanning the years 1972-1996, Sterman concluded that 82% of 174 patients who participated in these studies gained clinical improvement, while around two thirds of them managed to achieve change in their EEG towards 2021 normalization[8]. Other researchers (like Lubar and Bahler, Zhao, Wu, Liang and Hu, Johnson and Meyer[22]) reported decreases in seizure activity or even total cessation of seizures in some of the patients following neurofeedback treatment. Walker and Kozlowskiclaim that a qEEG-guided coherence training improves treatment outcomes[23]. The question remains to be asked: how is the neurofeedback effect achieved?

Possible mechanisms mediating the neurofeedback SMR effect

The Sensory Motor Rhythms (SMR) seem to emanate from the thalamus (specifically, from the ventrobasal nuclei of the thalamus, which conduct afferent somatosensory information)[24]. During SMR up-training, the firing pattern of these thalamic nuclei becomes more systematic and rhythmic, which suggests suppression of somatosensory information passage[25]. The GABA neurotransmitter participates in this process. This is also influenced by nonspecific cholinergic and monoaminergic neuromodulation, which can affect excitability levels in the thalamus and in cortical areas receiving the thalamic input[11]. SMR up-training is thought to result in better control over excitation in that system. Epilepsy is characterized by over-excitation of the cortical and/or thalamocortical areas. SMR training raises excitation threshold in these areas, and thus exerts its therapeutic effect. Another structure implied in this process is the striatum complex of the basal ganglia[11 25]. Froemke discusses coincidence detection and synaptic plasticity, and his concepts might be in line with an LTP (long-term potentiation) based explanation for the neurofeedback effect. All in all, it appears that the SMR up-training neurofeedback effect is achieved through the enhancement of inhibitory mechanisms in sensorimotor pathways[26].

Case 1 Report

There are many kinds of epilepsy. Among children, the most common type is known as Rolandic epilepsy, which is characterized by spikes over centro-temporal regions of the brain (the Rolandic Strip) [27]. The seizures are considered to be partial, because they occur over the Rolandic region of the brain only[28]. This kind of epilepsy usually starts in early childhood (sometimes as early as the age of 3), and often naturally recedes in adolescence[29,30]. Other neuropsychiatric and cognitive symptoms may accompany this disorder in some cases, such as attention deficit disorder and learning disabilities (specifically, difficulties with oral and written language or with drawing and visuospatial skills[30,31]). Despite this, children with this kind of epilepsy usually have normal levels of intelligence. This kind of epilepsy is also related to acquired epileptic aphasia (Landau-Kleffner Syndrome). Oral-motor deficiencies appear during the epileptic phase in some children. Recent studies have shown that adjustment difficulties at school, experienced by some of these children, are in correlation with their epileptic electroencephalographic activity[31].

This case study involves a patient with Rolandic epilepsy, A.B., age 8 years, who suffered from neuropsychiatric symptoms, cognitive dysfunction, verbal apraxia of speech and gross motor dysfunction. She was unable to function effectively in school and in many everyday life situations.

The first seizure was observed when she was 4.5 years old. She had an event of a sudden and involuntary contraction of muscles in all limbs, without loss of consciousness (before sleep). Her EEG taken at that time at the hospital showed forms of epilepsy and recurrent seizures with Rolandic temporal spikes on both sides. She was treated with Tegretol and later on with Depalept.

Although she was treated for epilepsy, her function at school and at home was very low. At the age of 7.5 her diadochokinesis was anomalously clumsy (e.g. she could not even put her shoes on and she could not speak).

A clinical EEG test was conducted in our clinic at intake for 10 minutes, with eyes open and eyes closed (Figure 1). Her parents were asked to fill a Conners' parent questionnaire before intake and during subsequent assessments. EEGs were recorded from 19 scalp sites. The electrodes were applied according to the International 10-20 system. The EEG was recorded in the average montage. Visual inspection of raw EEG was made in order to search for paroxysmal patterns that pop out of the background EEG. The EEG was analyzed with EEG/qEEG software (WinEEG). The analysis consisted of the following steps:

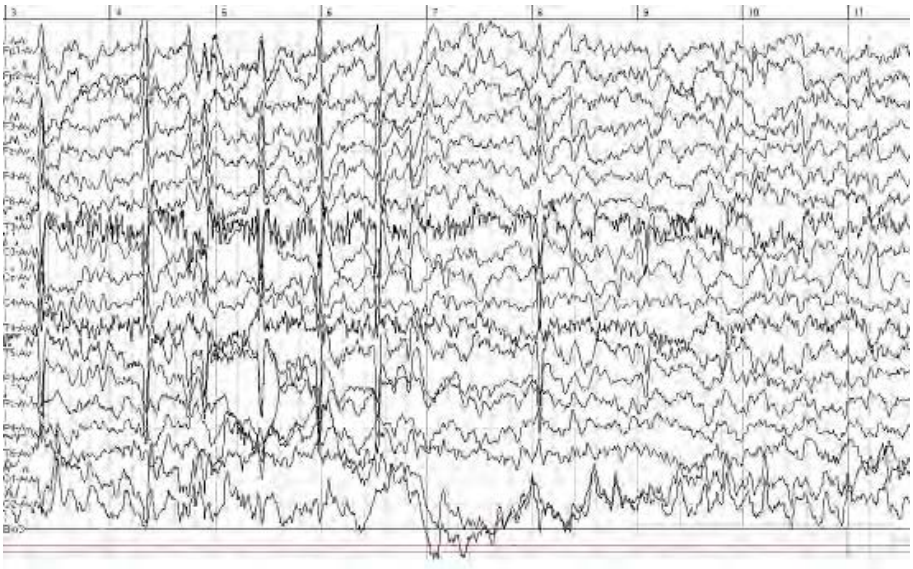


Figure 1: *complexes of spike & wave on the patient raw EEG (eyes open), at intake.*

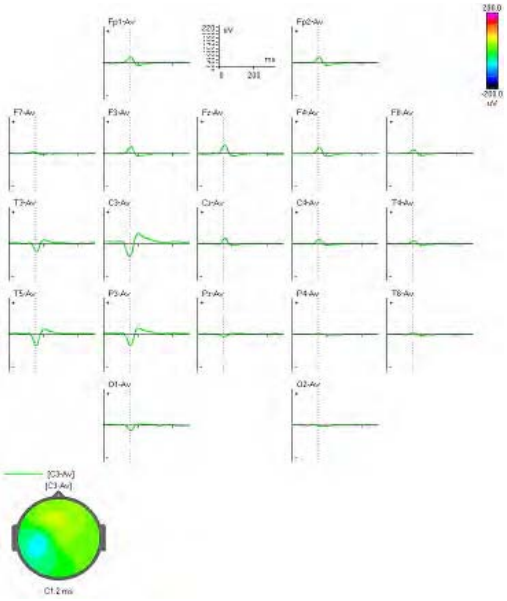


Figure 2: *Spike & wave pattern detected by the automatic spike averaging system on C3 at intake.*

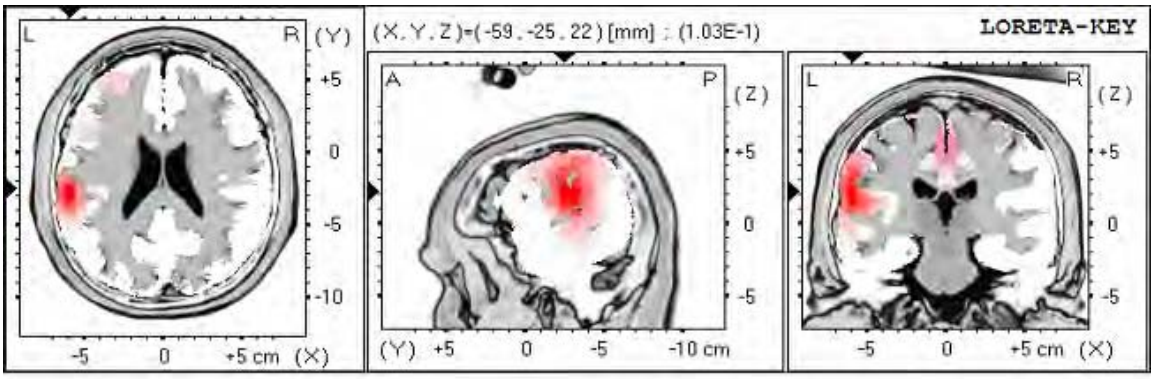


Figure 3: *Loreta analysis for source distribution indicates source in middle temporal gyrus, Brodmann area 22.*

1) Eye movement artifact correction and elimination: *(a)* using a spatial filtration technique based on zeroing the activation curves of individual Independent Component Analysis (ICA) components corresponding to horizontal and vertical eye movements, as well as *(b)* excluding epochs with an excessive amplitude of EEG and excessive faster and slower frequency activity.

   We used the automatic spike detection, analysis, and average spike calculation system. This resulted in evidence of significant paroxysmal activity consistent with the spike and wave pattern in centro-temporal regions, mostly on the left side (Figure 2). There were significant 257 events detected over 10 minutes of recording. We used low-resolution brain electromagnetic tomography (LORETA) analysis to locate the source distribution. It was located by LORETA in the middle temporal gyrus, Brodmann area 22 (Figure 3).

2) Fast-Fourier Transformation (FFT) of the corrected EEG for extracting EEG power. We computed EEG with eyes open and eyes closed average and compared to age-regressed, normative database (HBI), for absolute power (Figures 4 &5) . There were obvious power-

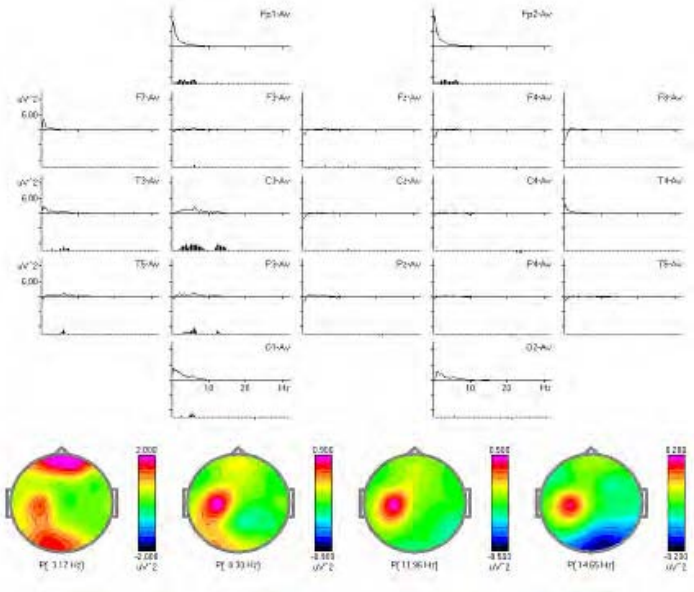


Figure 4: *Graphs of EEG power spectra (eyes open) compared to a normative database, at intake.*

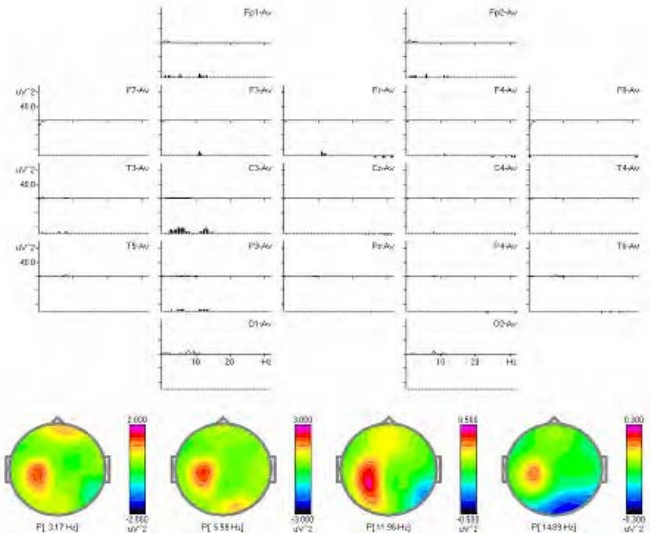


Figure 5: *Graphs of EEG power spectra (eyes closed) compared to a normative database, at intake.*

excesses in the 3-9Hz and 11-15 Hz band frequencies (Theta, Alpha and low Beta wave bands).

The patient underwent 20 guided Neurofeedback training sessions. The training involved the sensory motor strip, not directly involving the regions of the epileptiform activity. We up-trained 12-15 Hz (i.e. SMR). During the training, the patient was still receiving anticonvulsant medication. The parents reported a significant improvement in her speech comprehension and linguistic abilities.

After 17 sessions of neurofeedback we recorded and analyzed her EEG again. There were 115 events detected over 10 minutes of recording on C3 (Figure 6), and a significant change in the eyes-open and eyes-closed power spectra, compared with norms (HBI database), for absolute power (Figures 7 & 8).

The patient underwent 25 more guided neurofeedback training ses-

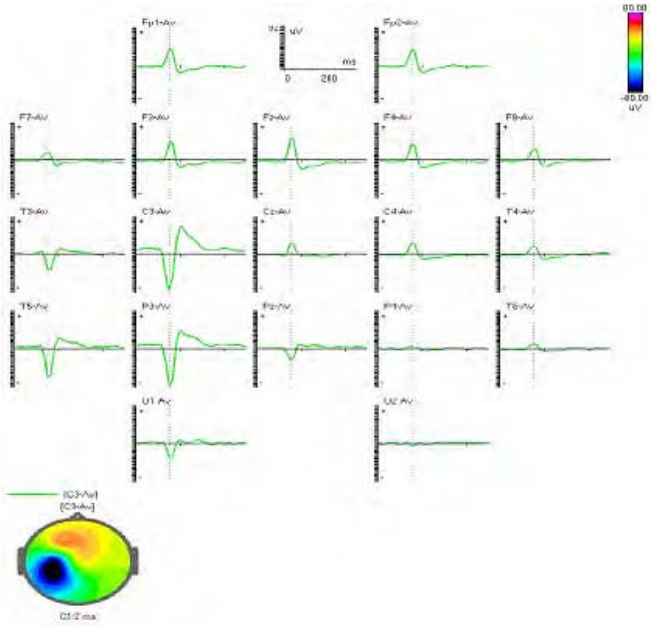


Figure 6: *Spike & wave pattern detect by the automatic spike averaging system on C3, after 17 neurofeedback sessions*

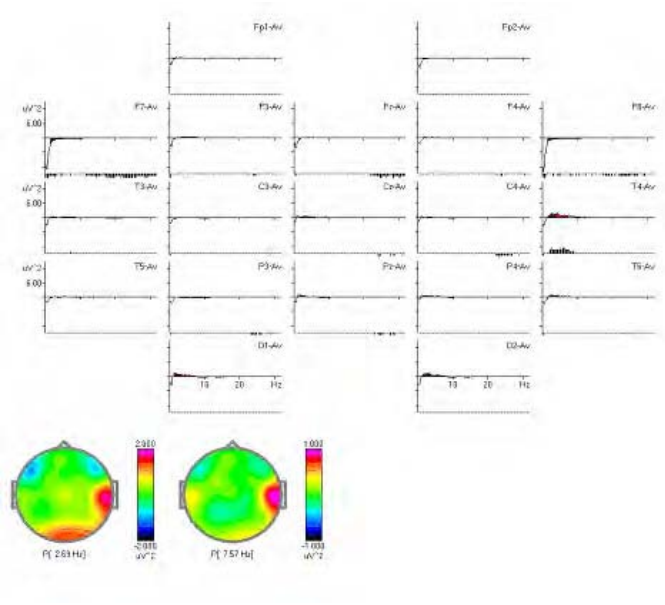


Figure 7: *Graphs of EEG power spectra (eyes open) compared to a normative database, after 17 neurofeedback sessions.*

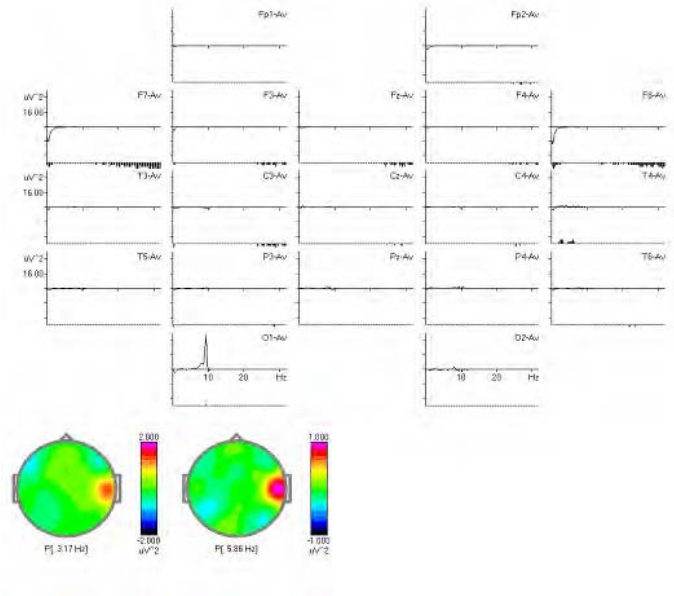


Figure 8: *Graphs of EEG Eyes closed power spectra compared to a normative database, after 17 neurofeedback sessions.*

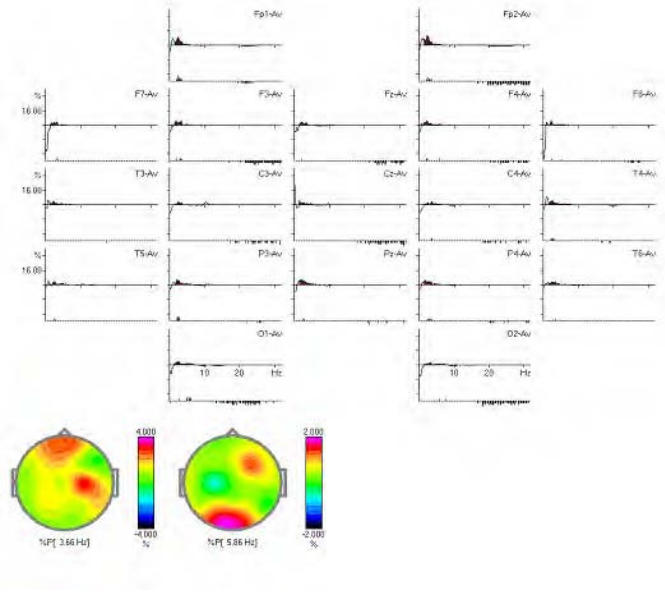


Figure 9: *Graphs of EEG power spectra (eyes open) compared to a normative database, after 42 neurofeedback sessions.*

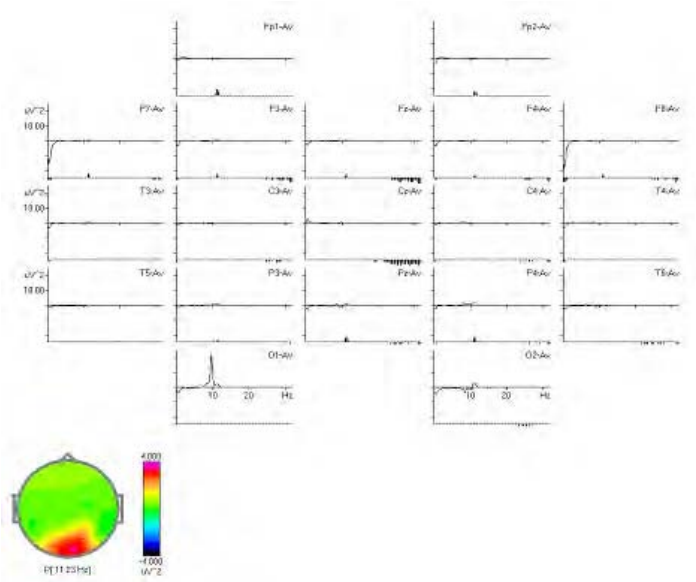
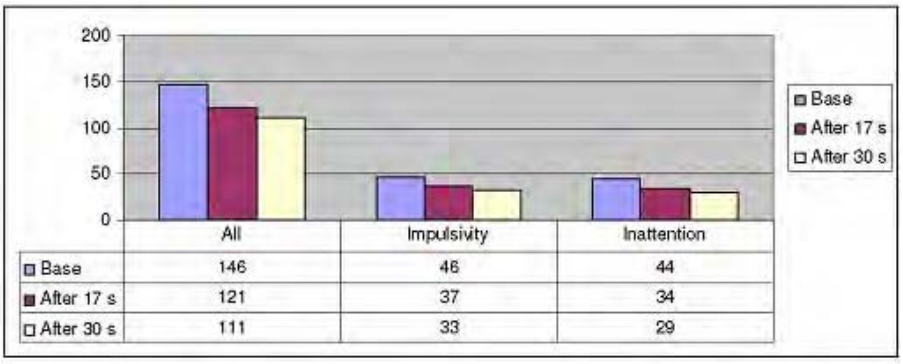


Figure 10: *Graphs of EEG power spectra (eyes closed) compared to a normative database, after 42 neurofeedback sessions.*



	All	Impulsivity	Inattention
Base	146	46	44
After 17 s	121	37	34
After 30 s	111	33	29

Figure 11: *Chart of Conners' parent questionnaires before treatment, after 17 sessions and after 30 sessions. The total rate score decreased during treatment (from 146 on the first assessment to 111 after 30 sessions). Attentiveness improved by 34% and impulsivity improved by 28%.*

sions. Around the middle of the training period there were no seizure events, so she stopped taking anticonvulsant medication. Continued improvement of language abilities and gross motor skills was observed, and she even started riding a bicycle on her own.

After 42 neurofeedback sessions we recorded and analyzed her EEG again. The patient's raw EEG was clear of any epileptiform discharges. There were zero events detected over 10 minutes of recording on C3. There were a few changes with eyes closed power spectra compared with norms for absolute power, mostly a lack of beta-power in the frontal lobe. These changes can be explained by her quitting the anticonvulsant medication (Figures 9, 10).

The Conners' parent questionnaires were taken in three different points in time, before treatment, after 17 training sessions, and after 30 sessions (Figure 11). Her total rate score decreased during the course of treatment (from 146 on the first assessment to 111 after 30 sessions). Attentiveness improved by 34% and impulsivity improved by 28%.

Case 2 Report

The patient, H.Y., a 10-year-old girl, was diagnosed with epilepsy and suffered from developmental delay, cognitive dysfunction, impulsivity, and wild behavior. Seizures were first observed at age 6. There were many observed instances of her disconnecting and gazing leftward, without limb movement. The patient was treated with anti-convulsant drugs, and it seemed like her epilepsy was controlled. At the age of 8.5 there were night-events during sleep, which were accompanied by grinding of teeth, trismus, tremors all over the body and loss of consciousness for a few minutes. Despite the drug treatment, the seizure events repeated every night. The EEG chart that was done at the hospital during wakefulness, nap, and sleep after sleep deprivation showed electrical status epilepticus during sleep (ESES). ESES is a typical childhood process of generalization of paroxysmal activity.

A clinical EEG was conducted in our clinic at intake for 10 minutes, with eyes open and eyes closed (Figure 12). Her parents were asked to fill a Conners' parent questionnaire before intake and during subsequent assessments. EEGs were recorded from 19 scalp sites. The electrodes were applied according to the International 10-20 system. The EEG was recorded in the average montage. Visual inspection of raw EEG was made in order to search for paroxysmal patterns that pop out of the background EEG. The EEG was analyzed with EEG/qEEG software (WinEEG). The analysis consisted of the following steps:

1) Eye movement artifact correction and elimination *(a)* using a spatial filtration technique based on zeroing the activation curves of individual Independent Component Analysis (ICA) components corresponding to horizontal and

vertical eye movements, as well as*(b)* excluding epochs with an excessive amplitude of EEG and excessive faster and slower frequency activity.

2) We used the automatic spike detection, analysis, and average spike calculation system. This resulted in evidence of significant paroxysmal activity consistent with the spike and wave pattern in medial frontal gyrus regions F7 and Fz (Figure 13). There were significant 121 events detected over 10 minutes of recording. We used LORETA analysis to locate the source distribution. It was located by LORETA in the middle temporal gyrus, Brodmann area 9 (Figure 14).

3) Fast-Fourier Transformation (FFT) of the corrected EEG was used for extracting EEG power. We computed EEG with eyes open and eyes closed and compared to age-regressed, normative database (HBI) for absolute power (Figures 15 &16) . There were obvious power-excesses of high Beta-waves (25-30Hz) on frontal-central-parietal areas and of Theta waves (2-6 Hz) over central areas.

The patient underwent 37 guided neurofeedback training sessions. The active electrode was on Cz. We down-

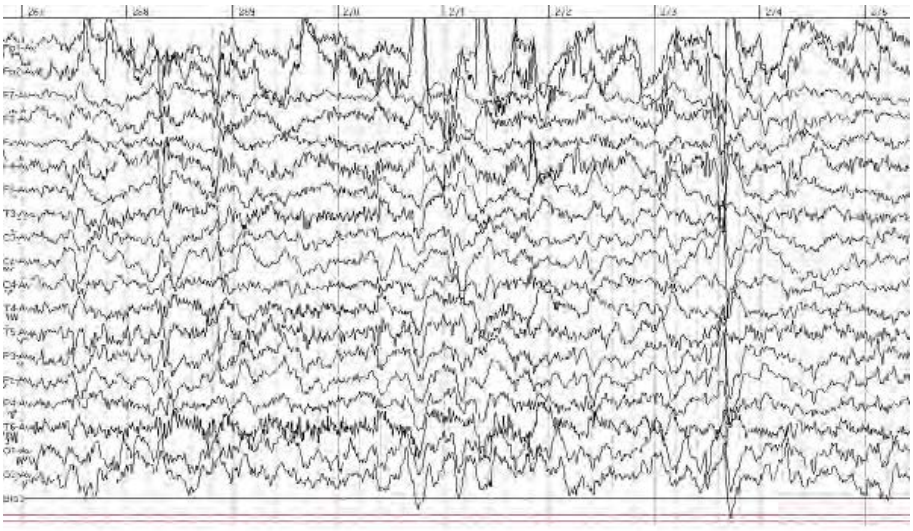


Figure 12: *complexes of spike & wave on the patient row EEG (eyes open), at intake.*

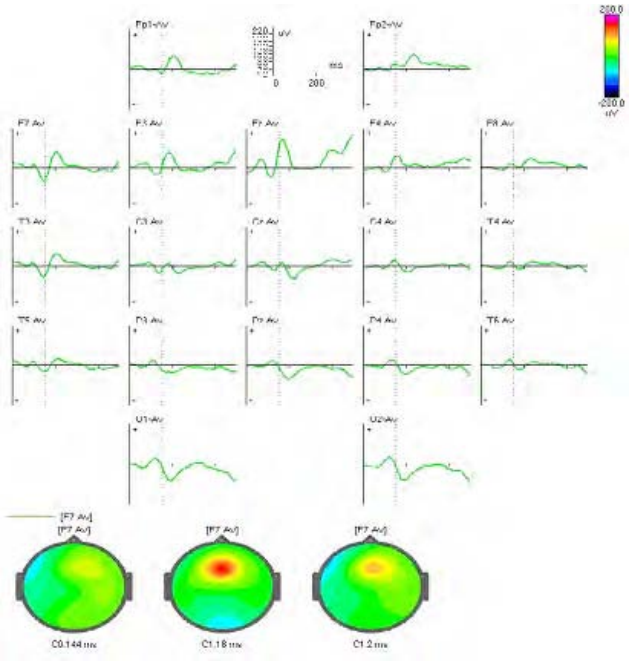


Figure 13: *Spike & wave pattern detect by the automatic spike averaging system on Fz at intake.*

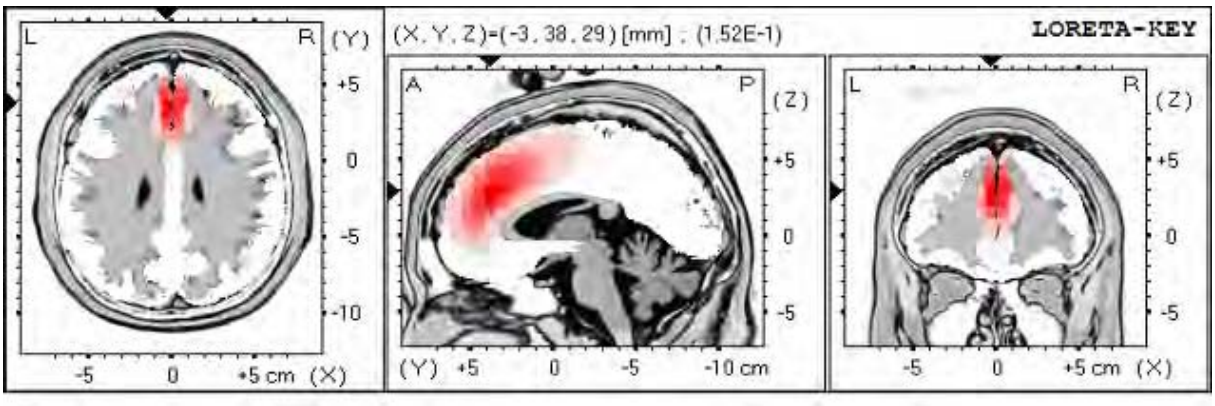


Figure 14: *Loreta analysis for source distribution indicates source location in the middle frontal gyrus, Brodmann area 9.*

trained both 2–6Hz and 20–25Hz. During the training, the patient was still receiving anticonvulsant medication as before. The parents reported a significant improvement in her behavior at school and at home. After 37 neurofeedback sessions we recorded and analyzed her EEG again. There was only 1 event detected over 10 minutes of recording on Fz (Figure 17), and a significant decrease in theta-power in the eyes-open and eyes-closed power spectra, compared with norms, for absolute power (Figures 18 & 19).

Analysis of the Conners' parent questionnaires taken before treatment, after 20 neurofeedback sessions, and after 35 sessions shows a 26% improvement in attentiveness and a 44% improvement in impulsivity (Figure 20).

Summary

Based on our previous experience and according to previous studies published, we hypothesized that qEEG-guided neurofeedback can help regulate EEG and help achieve complete cessation of seizures for patients with epilepsy that was not controlled by medications. This article reviewed two cases of epileptic patients:

1. A 7.5-year-old female patient, diagnosed with benign Rolandic epilepsy with cognitive dysfunction, verbal apraxia of speech and gross motor dysfunction. Although she was treated with anticonvulsant medication, she could not function independently at school and at home.

   We used EEG/qEEG and parent questionnaires to follow her progress before and during the training sessions. The baseline EEG that we took before starting the neurofeedback treatment showed significant paroxysmal activity consistent with the spike

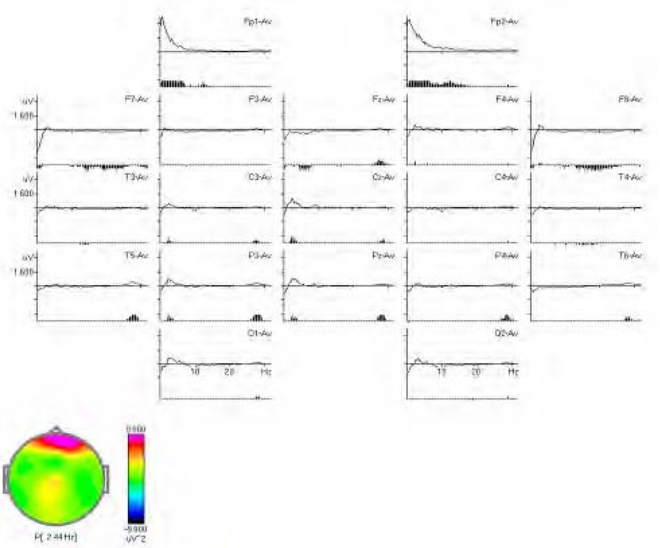


Figure 15: *Graphs of EEG power spectra (eyes open) compared to a normative database, at intake.*

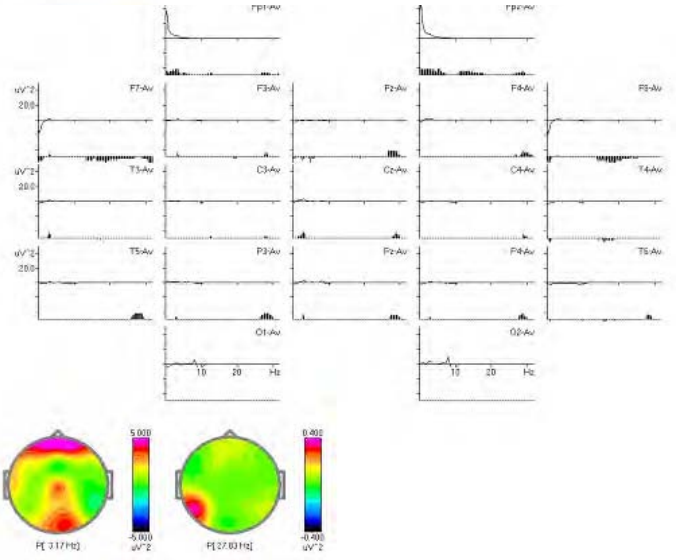


Figure 16: *Graphs of EEG power spectra (eyes closed) compared to a normative database, at intake.*

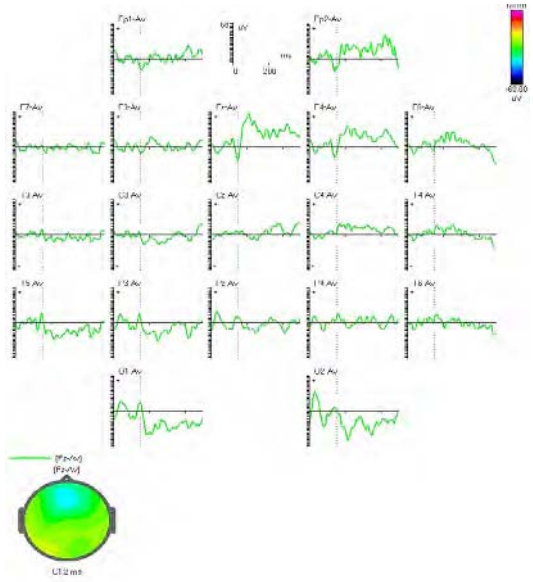


Figure 17: *Spike & wave pattern detect by the automatic spike averaging system on Fz, after 37 neurofeedback sessions.*

and wave pattern in centro-temporal regions, mostly on the left side (Figure 2). There were significant 257 events detected on C3 over 10 minutes of recording. The second EEG assessment, taken after 17 sessions of neurofeedback training, detected 115 spike and wave events over 10 minutes of recording on C3. The third EEG, taken after another 25 (total of 42) training sessions, revealed zero events of spike and wave on C3.

Over the course of neurofeedback the patient improved significantly in language understanding, speech, and gross motor skills. She speaks fluently and rides a bicycle. There is an improvement in her attention and impulsivity, but her cognitive functions remain relatively low for her age.

2. A 10-year-old female patient, diagnosed with epilepsy and developmental delay, cognitive dysfunction and wild, impulsive behavior. Although she was treated with anticonvulsant medication, the seizures repeated every night during sleep and her behavior was uncontrollable at school or at home.

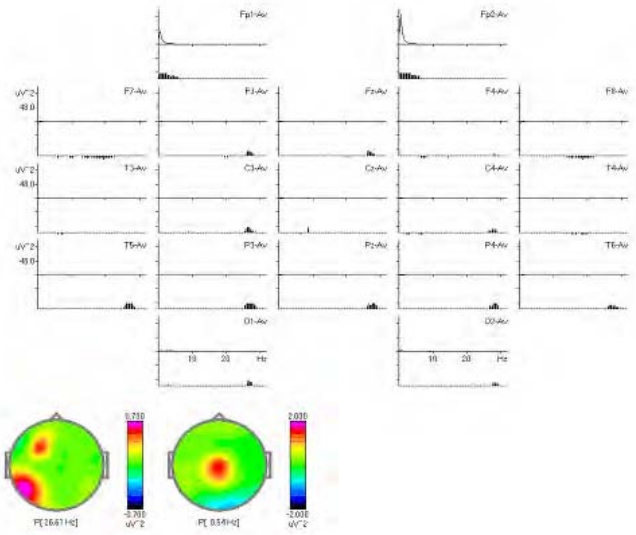


Figure 18: *Graphs of EEG power spectra (eyes open) compared to a normative database, after 37 neurofeedback sessions.*

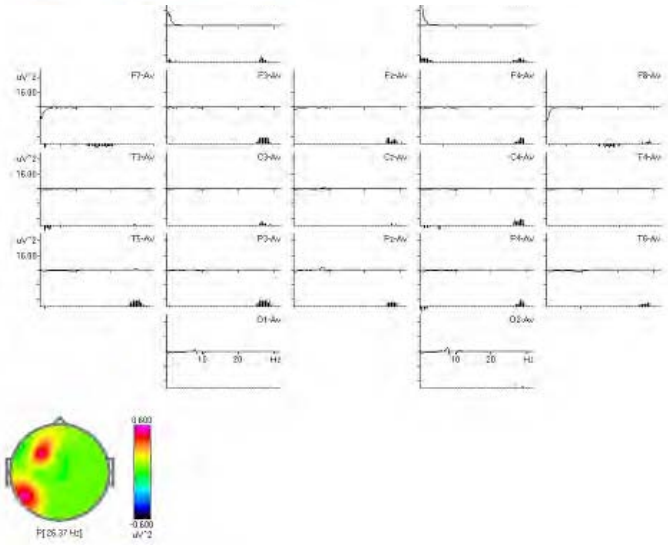
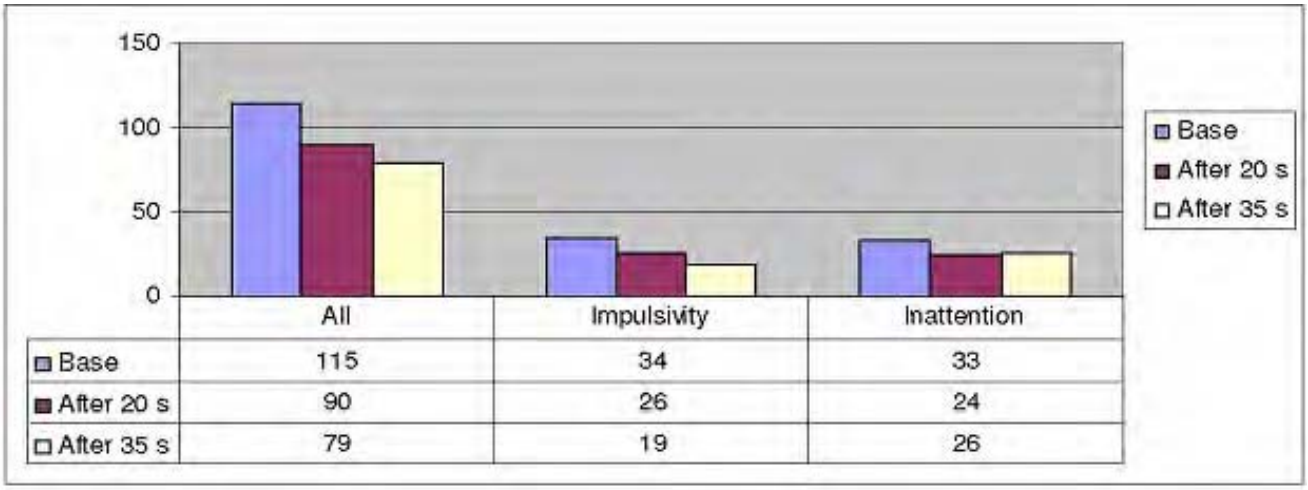


Figure 19: *Graphs of EEG power spectra (eyes open) compared to a normative database, after 37 neurofeedback sessions.*



	All	Impulsivity	Inattention
Base	115	34	33
After 20 s	90	26	24
After 35 s	79	19	26

Figure 20: *Chart of Conners' parent questionnaires before treatment, after 20 sessions, and after 35 sessions. The total rate score decreased during the treatment (from 115 on the first assessment to 90 after 35 sessions). Attentiveness improved by 26% and impulsivity improved by 44%.*

We used EEG/qEEG and parent questionnaires to follow her progress before and during the training sessions. The baseline EEG that we took before starting the training sessions showed significant paroxysmal activity consistent with the spike and wave pattern in frontal regions, mostly on Fz (Figure 13). There were significant 121 events detected on Fz over 10 minutes of recording. The second EEG assessment, taken after 37 sessions of neurofeedback training, detected 1 spike and wave event over 10 minutes of recording on Fz. Over the course of neurofeedback the patient's behavior at school and at home improved significantly.

About the Author

Rivi Sela, MSW, is a co-founder and CEO of BrainGames, two clinics in Israel which have operated for the last six years. The clinics specialize in treating ADHD, epilepsy, and autism. Under her supervision, the centers have provided diagnostic evaluations, qEEG recording and interpretation, and drug-free interventions for hundreds of children and adults.

Before that, Rivi served as the Chief Technology Director of "Sheba," the largest medical center in Israel. During her tenure there she specialized in developing and implementing clinical technologies, hardware, and software components in collaboration with companies from around the globe. She may be reached at rivi@braingamesisrael.com, Tel: +972-3-9733136.

Meirav Shaked–Toledano, MSW, is CEO of BrainGames-Israel Neurofeedback Clinics

References

1 World Health Organization. Epilepsy: etiology, epidemiology and prognosis. 2001; http://www.who.int/mediacentre/factsheets/fs165/en/index.html

2 Lasemidis, ID. Epileptic seizure prediction and control. IEEE Trans Biomed Eng 2003; 50: 549–558.

3 Witte H, Lasemidis ID, Litt B. Special issue on epileptic seizure prediction. IEEE Trans Biomed Eng 2003; 50: 537–539.

4 Rasalam AD, Hailey H, Williams JH, Moore SJ, Turnpenny PD, Lloyed DJ, Dean JC. Characteristics of fetal anticonvulsant syndrome associated autistic disorder. Dev Med Child Neurol 2005; 47: 551–555.

5 Fisher RS, van Emde Boas W, Blume W, Elger C, Genton P, Lee P, Engel J Jr. Epileptic seizures and epilepsy: definitions proposed by the International League Against Epilepsy (ILAE) and the International Bureau for Epilepsy (IBE). Epilepsia, 2005; 46(4): 470–2.

6 Hammond DC. What is neurofeedback. Investigations in neuromodulation, neurofeedback and applied neuroscience. J Neurother 2011; 15:4, 305–336.

7 Sterman MB, Egner T. Foundation and practice of neurofeedback for the treatment of epilepsy. Appl Psychophysiol Biofeedback 2006; 31(1): 21–35.

8 Sterman MB. Basic concepts and clinical findings in the treatment of seizure disorders with EEG operant conditioning. Clin Electroencephalogr 2000; 31:4555.

9 Lubar JF, Shabsin HS, Natelson SE, Holder GS, Whitsett SF, Pamplin WE, Krulikowski DI. EEG operant conditioning in intractable epileptics. Arch Neural 1981; 38:11, 700–704.

10 Sterman MB, Macdonald LR, Stone RK. Biofeedback training of the sensorimotor electroencephalogram rhythm in man: effects on epilepsy. Epilepsia 1974; 15: 395–416.

11 Sterman MB & Egner T. Neurofeedback treatment of epilepsy: from basic rationale to practical application. Expert Rev. Neurotherapeutics 2006; 6(2): 247257.

12 Cott A, Pavloski RP, Black AH. Reducing epileptic seizures through operant conditioning of central nervous system activity: procedural variables. Science 1979; 203: 73–75.

13 Kaplan BJ. Biofeedback in epileptics: equivocal relationship of reinforced EEG frequency to seizure reduction. Epilepsia 1975; 16: 477–485.

14 Finley WW, Smith HA, Etherton MD. Reduction of seizures and normalization of the EEG in severe epileptic following sensorimotor biofeedback training: preliminary study. Biol. Psychiatry 1975; 2: 189–203.

15 Lantz DL & Sterman MB. Neuropsychological assessment of subjects with uncontrolled epilepsy: effects of EEG biofeedback training. Epilepsia 1988; 29(2): 163–171.

16 Tan G, Thornby J, Hammond DC, Strehl U, Canady B, Arnemann K, Kaiser DA. Clinical EEG and Neurosceience 2009; 40(3):173–179.

17 Andrews DJ, Schonfeld WH. Predictive factors for controlling seizures using a behavioral approach. Seizure 1992; 1(2):111–116.

18 Sterman MB & Shouse MN. Quantitative analysis of training, sleep EEG and clinical response to EEG operant conditioning in epileptics. Electroencephalogr Clin Neruophysiol 1980; 49: 558–576.

19 Whitsett SF, Lubar JF, Holder GS, Pamplin WE, Shabsin HS. A double blind investigation of the relationship between seizure activity and the sleep EEG following EEG biofeedback training. Biofeedback Self-Regul 1982; 7(2): 193209.

20 Lubar JF, Bahler WW. Behavioral management of epileptic seizures following EEG biofeedback training of the sensorimotor rhythm. Biofeedback Self-Regul 1976; 1:77–104.

21 Zhao L, Wu W, Liang Z, Hu G. Nonlinear analysis in treatment of intractable epilepsy with EEG biofeedback. Proc IEEE Eng Med Biol 2005; 5: 4568–4571.

22 Johnson RK, Meyer RG. Phased biofeedback approach for epileptic seizure control. J Behav Therapy Experim Psychiatry 1974; 5: 185–187.

23 Walker JE, Kozlowski GP. Neurofeedback treatment of epilepsy. Child Adolesc Psychiatr Clin N Am 2005; 14: 163–176.

24 Howe RC & Sterman MB. Cortical-subcortical EEG correlates of suppressed motor behavior during sleep and waking in the cat. Electroencephalogr Clin Neurophysiol 1972; 32(6): 681–695.

25 Froemke RC, Poo MM, Dan Y. Spike-timing-dependent synaptic plasticity depends on dendritic location. Nature 2005; 434(7030): 221–225.

26 Sterman MB. Physiological origins and functional correlates of EEG rhythmic activities: implications for self-regulation. Biofeedback self regul 1996; 21(1): 333.

27 Clarke T, Strug LJ, Murphy PL, Bali B, Carvalho J, Foster S, Tremont G, Gagnon BR, Dorta N, Pal DK. High risk of reading disability and speech sound disorder in rolandic epilepsy families: case – control study. Epilepsia*** 2007; 48(12): 2258–65.

28 Deltour L, Barathon M, Quaglino V, Vernier MP, Despretz P, Boucart M, Berquin P. Children with benign epilepsy with centrotemporal spikes (BECTS) show impaired attentional control: evidence from an attentional capture paradigm. Epileptic Disord 2007; 9(1): 32–38.

29 Pan A, Luders HO: Epileptiform discharges in benign focal epilepsy of childhood. Epileptic Disord 2000; 2S1: 29–36.

30 Monastra V, Lubar J, Linden M. VanDeusen P, Green G, Wing W, Phillips A, Fenger TN. Assessing attention deficit hyperactivity disorder via quantitative electroencephalography: an initial validation study. Neuropsychology 1999;

EEG & qEEG Technology Identifies Neurobiomarkers Critical to Medication Selection and Treatment for Children and Adolescents with ADHD

Ronald J Swatzyna, PhD, LCSW, BCN

In early 2013, the National Institute of Mental Health (NIMH) launched the Research Domain Criteria (RDoC), in an effort to evolve the diagnostic process by incorporating a multidisciplinary approach that relies not only on symptoms, but also on genetics, neuroimaging, and cognitive science. This movement away from the traditional categorization of the Diagnostic and Statistical Manual (DSM) towards a science-based classification highlights the importance of psychiatry fully exploring the potential of available electrophysiological testing. There are previous classifications of ADHD by Joel Lubar and Daniel Amen. However, our five year research (N=386 pending publication) led to the development of a neurobiomarker profiling model which we use in our clinic. Based on clinically correlated electroencephalogram (EEG) and quantitative EEG (qEEG) findings, our model is both concise and suitable to application by neurofeedback practitioners. There is not a layman's equivalent to the names used in this suggested classification. To date, the application of clinical EEG and qEEG have been very limited in psychiatry, although studies suggest effective application in diagnosis, medication response, and treatment selection (Coburn, Lauterbach, Boutros, Black, Arciniegas, & Coffey, 2006). Neurobiomarkers specific to ADHD symptom presentation are numerous and account for the variance in treatment response (Johnstone, Gunkelman, & Lunt, 2005).

Based on clinically correlated electroencephalogram (EEG) and quantitative EEG (qEEG) findings, our model is both concise and suitable to application by neurofeedback practitioners.

Diagnosing ADHD

The diagnosis of ADHD is established when a minimum number of symptoms are identified through testing, behavioral observation, and self-report; however, the diagnostic specificity of these approaches is limited by the fact that many similar issues can cause identical symptoms. Chabot, Michele & Prichep (2005) state, "ADD represents a spectrum of disorders that may be represented by different neurobiomarkers present within the population of children with attention and learning problems" (p. 42). Although ADHD symptoms cross all subtypes, it is our experience that there are subtle significant tendencies common to each subtype. We find that there are four subtypes that are more

Prediction of ADHD Medication Response
via Neurobiomarker Profiling

Neurobiomarker	Behavioral characteristics	Recommended Medications
• **Slow Alpha Peak**	Under-aroused, maturationally lagged	Amphetamine-class
• **Frontal Midline Theta**	Distractible, requires high stimulation levels to maintain focus	Methylphenidate-class
• **Fast Alpha Peak**	Anxious, superior declarative and semantic memory and reaction time	Alpha 2 agonists, and anticonvulsants
• **Anterior Hypercoherent Alpha**	Artistic/creative; Affective regulatory difficulties	SSRI class

successfully identified, medicated, and treated. These cases usually only have an ADHD diagnosis and are on a single medication. In addition, the preliminary findings of our study of 224 children and adolescents (Swatzyna, Pillai, Tarnow, Tannous, Kozlowski & Schieszler pending publication) suggests that EEG and qEEG technology have identified four neurobiomarkers common in those more difficult to diagnose refractory cases of ADHD having multiple diagnoses and prescribed multiple medications.

Neurobiomarkers in ADHD

ADHD symptoms are common to many diagnoses and can often elude detection. However, there are four subtypes of ADHD that can be identified by four distinctly different neurobiomarkers. Neurobiomarkers (NBMs) are abnormal fluctuations in an EEG and the output of the qEEG. The following neurobiomarkers respond well to medications:

1. Slow Alpha Peak (SAP) is identified in children who lag in maturational development and whose central nervous systems (CNS) are under-aroused. Children and adolescents with this pattern should respond well to Amphetamine-type stimulants that increase norepinephrine (NE) and speed up the peak frequency of alpha.
2. Frontal Midline Theta (FMT) is most often seen in distractible children who require high stimulation such as video gaming to maintain focus. Children and adolescents with FMT have been found to respond best to methylphenidate class medicine. Methylphenidate medication increases the release of NE but more so, dopamine. Those with frontal midline theta excess (FMT) can have issues with dopamine depletion.
3. Fast Alpha Peak (FAP) is seen in

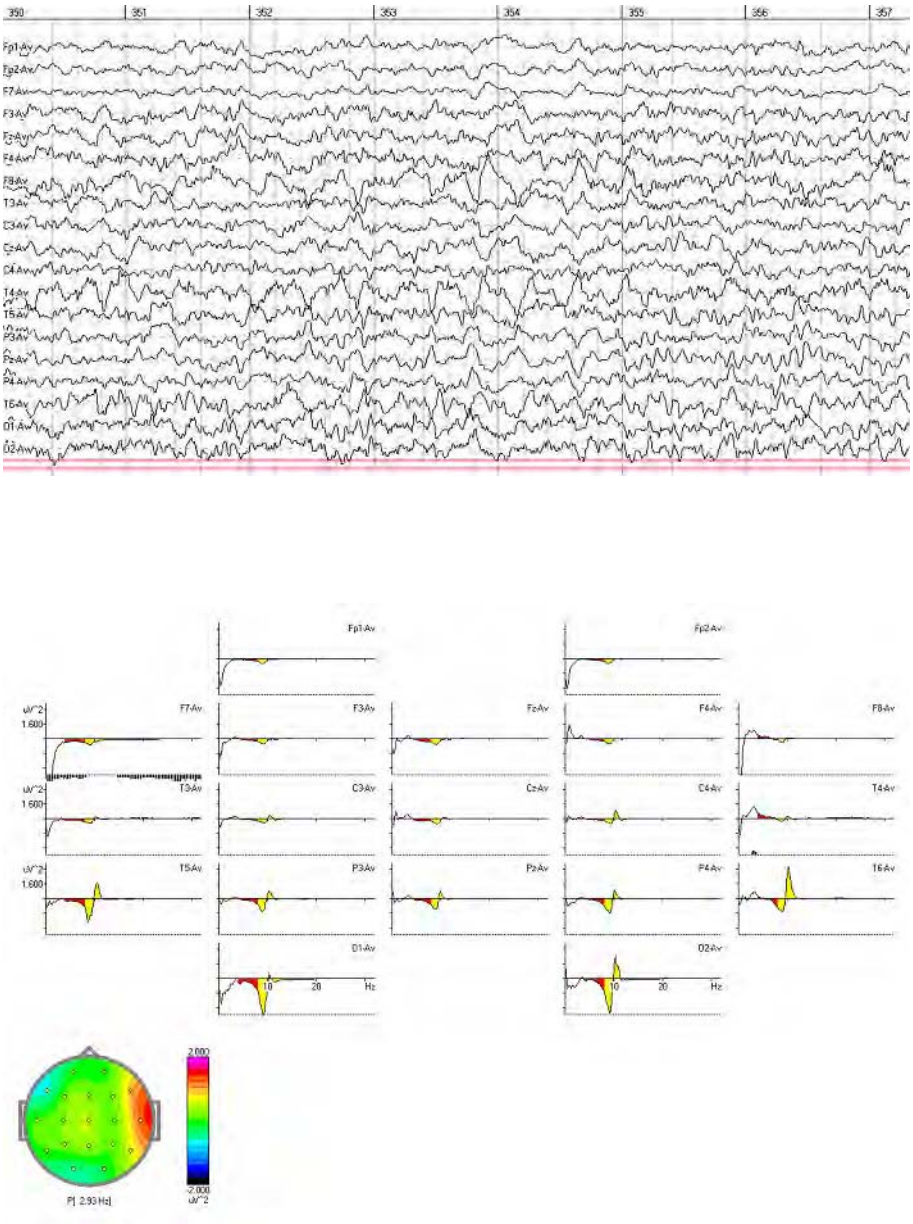


Figure 1: *Focal slowing in the EEG of a 12 y/o male, with diagnoses of Dx ADHD combined type & autism spectrum disorder.*

A) *Eyes Closed – background EEG tracings. Scale: 70 mcV/cm.*

B) *Spectral differences: patient-norms. Absolute EEG power. Slowing was thought to reflect possible white matter involvement.*

anxious ADHD children who are most often highly intelligent with superior declarative (facts & knowledge recall) and semantic memory. Alpha 2 agonists and anticonvulsants reduce NE. Children and adolescents with fast peak frequency of alpha (FAP) often have anxiety issues (CNS hyperarousal) and benefit from reduction of NE. Medications to be avoided are in the benzodiazepine class as well as any other medication that speeds up alpha such as stimulants and Selective Norepinephrine Reuptake Inhibitors (SNRI).

4. Anterior Hypercoherent Alpha (AHA) is seen in very artistic/creative children who tend to be constantly thinking. Difficulty in focus stems from their distracting internal dialogue. Children and adolescents with AHA tend to have issues with affective regulatory dysfunction. These children typically respond well to Selective Serotonin Reuptake Inhibitor (SSRI) class medications, which increase serotonin levels while reducing anterior hypercoherent alpha (AHA).

Neurobiomarkers in Refractory ADHD

In many cases, ADHD is correctly diagnosed; however, multiple attempts at psychotropic intervention may fail, often producing negative side effects. More recently, four NBMs have been identified in persons with ADHD and may account for medication failure. These NBMs are:

1. Focal slowing (FS), (Figure 1) is identified in head injury/concussions, stroke, and genetic abnormalities, to mention a few, and is characterized by electrical activity in one area of the brain that is

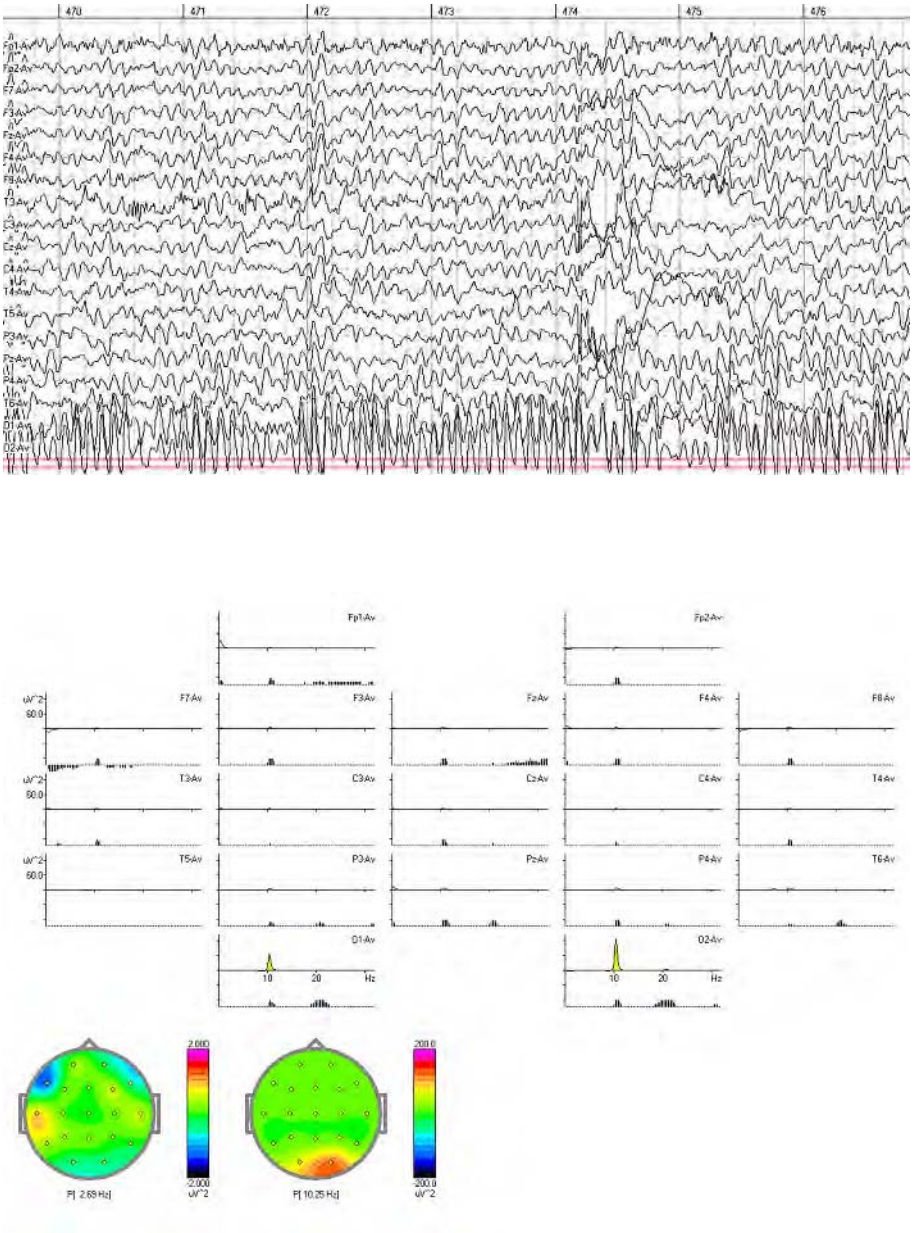


Figure 2: *Transient discharges recorded in the EEG of a 9 y/o female with history of ADHD & LD.*

A) *Eyes Closed – background EEG rhythms. Scale: 100 mcV/cm.*

B) *Spectral differences: patient-norms. Absolute EEG power. Note the significance at T3, F7, F3, P3 and O1*

firing much slower than adjacent areas. This results in suboptimal performance and poor connectivity. In the current study, 59% of children and 57% of adolescents with FS also had diagnoses of ADHD. These anomalies can have many cognitive deficits similar to ADHD (e.g., poor attention, distractibility, impulsivity) and do not typically respond well to medication. Attempts to speed up the area that is focally slowed results in the rest of the brain becoming pathologically fast with intolerable side effects. Since traumatic brain injuries typically occur in focal regions, the FDA has yet to approve any medications for their treatment. Other neuromodulation interventions such as neurotherapy, transcranial direct current stimulation (tDCS), and transcranial magnetic stimulation (TMS) can isolate and speed up the slow area, affectively reconnecting the neural network.

2. Transient discharges (TD), (Figure 2) are erratic bursts of electrical activity. These are considered normal variants in most EEG patients. However, if the functional area having the discharges is symptomatic, that area becomes noteworthy and should be treated (Asokan, Pareja, & Niedermeyer, 1987). In the current study, 61% of children and 56% of adolescents with transient discharges also had diagnoses of ADHD. Depending upon the severity and location, TD can account for many of the ADHD and learning disability issues. TDs occur more often with insufficient sleep, high sugar/high carbohydrate intake, and high stimulation which increase transient cognitive impairment.

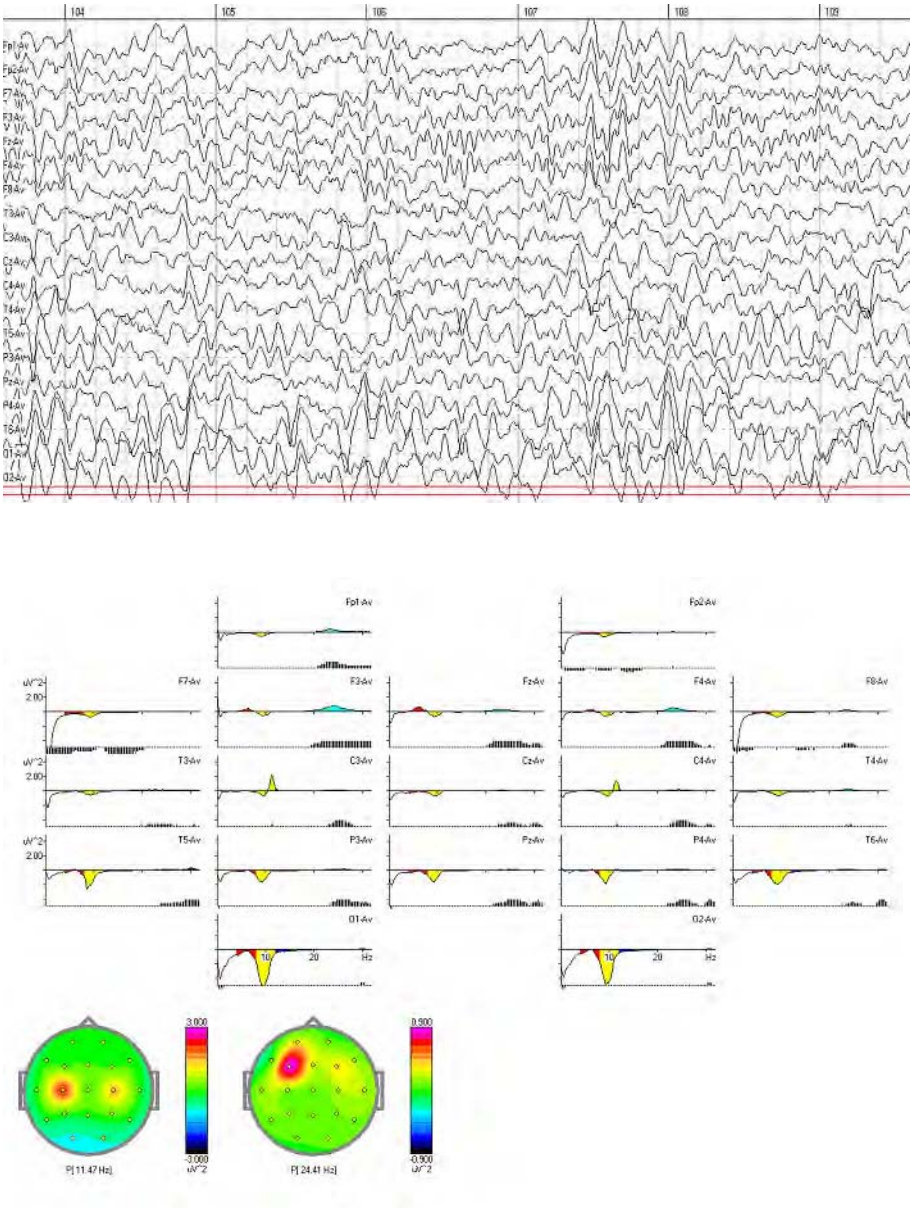


Figure 3: *Beta Spindles in the EEG of a 13 y/o male; Dx: ADHD combined type & generalized anxiety disorder.*

A) *Eyes Closed – background EEG rhythms. Scale: 70 mcV/cm. Note muscle artifacts at: Fp1, F3, T3.*

B) *Spectral differences: patient-norms. Absolute EEG power. The bins with statistically significant (t-test) differences are marked by bars at the bottom of each curve. The smallest ones correspond to p<0.05 (z-score>2), the largest ones - to p<0.001 (z-score>3), the medium ones – to p<0.01 (z-score>2.6). Topographies of significant deviations from normality are presented at the bottom.*

Consideration of stabilizing with anticonvulsant is recommended prior to prescribing a short acting methylphenidate for the ADHD slower activity (Millichap, Millichap & Stack, 2011).

3. Beta Spindles (BS), (Figure 3) are high frequency synchronous activities associated with cortical irritability, having an easily kindled cortex. This activity is also identified in excessive use of benzodiazepines (sedatives). Our current study finds 43% of children and 50% of adolescents with BS also have a diagnosis of ADHD. In addition to the comorbidity of ADHD and BS (Clarke, Barry, & Selikowtiz, 2001), other anxiety disorders commonly coexist. Those with BS have excessive excitatory neurochemistry. Medications such as Neurontin, Lyrica, Intuniv or Clonidine all work to reduce BS; however, studies have found that SSRIs often produce unacceptable side effects.
4. Encephalopathy (EN), (Figure 4) is described as a damage, disease, or malfunction of the brain and is commonly identified in children having metabolic (thyroid), electrolytic, anoxic (obstructive sleep apnea) etiology. In the current study, 74% of children and 63% of adolescents with EN also had diagnoses of ADHD. In some cases there are developmental delays in academics and behavior. These cases should be treated medically prior to any psychotropic or neurostimulation intervention.

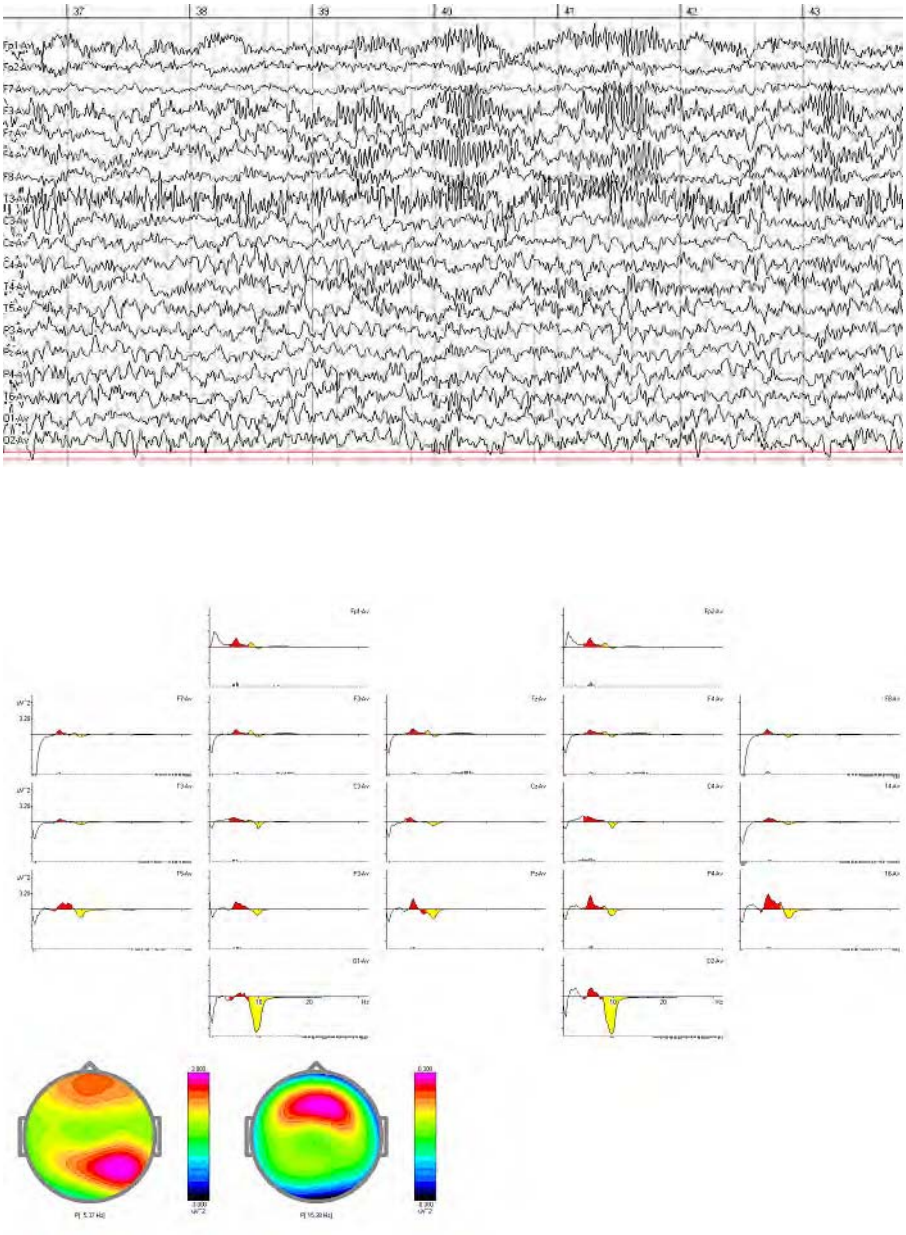


Figure 4: *8 y/o Female with suspected toxic encephalopathy. Dx: ADHD combined type and Mood Disorder, NOS.*

A) *Eyes Closed – background EEG rhythms. Scale: 100 mcV/cm. Note electrode movement artifacts at: Fp1, Fp2, O2.*

B) *Spectral differences: patient-norms. Absolute EEG power. The bins with statistically significant (t-test) differences are marked by bars at the bottom of each curve. The smallest bars correspond to p<0.05 (z-score>2), the medium bars to p<0.01 (z-score>2.6) and the largest bars - to p<0.001 (z-score>3). Topographies of significant deviations from normality are presented at the bottom.*

Summary

We have learned in the past eight years that there is much room for improvement in the selection of medication and treatment of ADHD in children and

Neurobiomarkers Associated with Refractory ADHD		
Neurobiomarker	**Etiologies**	**Recommended interventions**
• **Focal slowing**	Head injury/concussions, stroke, genetic disorders	Focal application of neurofeedback, tDCS, rTMS
• **Transient discharges**	Normal variant; ADHD, learning disability	Regulate diet, sleep, stimulation anticonvulsant and, once regulated, methylphenidate
• **Beta Spindles**	ADHD; anxiety disorders; Benzodiazepines	Neurontin, Lyrica, Intuniv or Clonidine and titrate off all benzodiazepines
• **Encephalopathy**	Metabolic, electrolytic, anoxic, post-traumatic	Treat underlying medical condition first; hyperbaric oxygen and Interactive Metronome have shown promise

adolescents. EEG and qEEG technologies provide identification of neurobiomarkers and are proving to be valuable tools for experienced physicians who: *(1)* know how to interpret the findings, *(2)* use published research suggestions to avoid medications that are likely to make their patients worse, and *(3)* consider empirical trials of research-supported medications. Lastly, neurobiomarker profiling is only a tool to provide information and is never intended to replace an experienced physician's wisdom and judgment. Psychiatrists and neurologists familiar with the use of EEG and qEEG technology have a distinct advantage.

About the Author

Dr. Ronald J. Swatzyna received his Masters of Science and Doctorate of Philosophy degrees from The University of Texas Arlington. Currently, he is the Director of Electro-Neurophysiology Research and Director of Neurotherapy at the Tarnow Center for Self-Management®, and is an associate of Brain Science International. Dr. Swatzyna is a licensed clinical social worker and board certified in neurotherapy and biofeedback by the Biofeedback Certification International Alliance (BCIA). For 17 years, Dr. Swatzyna has analyzed and treated the most diagnostically challenging cases in both inpatient and outpatient settings. As a researcher, he has presented and/or published over 40 peer-reviewed papers on brain dysfunction and other related topics at national and international conferences and is a Special Editor for WebmedCentral plus, an online British medical journal. In 2011, Dr. Swatzyna was inducted into Sigma Xi: The Scientific Research Society: Rice University/Texas Medical Center Chapter and in 2013, he accepted an appointment to the board of directors. Dr. Swatzyna is a veteran of both Vietnam and the first Gulf War, and his personal battle with a traumatic brain injury and posttraumatic stress disorder has motivated him to become a leading expert in brain dysfunction.

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References:

Asokan, G., Pareja, J., & Niedermeyer, E. (1987). Temporal minor slow and sharp EEG activity and cerebrovascular disorder. Clinical electroencephalography 18(4), 201–210

Chabot, R.J., Michele, F.D. & Prichep, L. (2005). The Role of Quantitative EEG in Child and Adolescent Psychiatric Disorders. Child and Adolescent Psychiatric Clinics of North America 14(1), 21–53.

Johnstone, J., Gunkelman, J., & Lunt (2005). Clinical database development: Characterization of EEG Phenotypes. Clinical EEG and Neuroscience 36(2), 99–107.

Millichap, J.G., Millichap, J.J. & Stack, C.V. (2011). Utility of the electroencephalogram in Attention Deficit Hyperactivity Disorder. Clinical EEG and Neuroscience 42(3), 180–183.

Swatzyna, R.J., Pillai, V.J., Tarnow, J.D., Tannous, J., Kozlowski, G.P. & Schieszler, C. (pending). EEG/QEEG technology identifies neurologic biomarkers critical to medication selection and treatment: A preliminary study.

Clinical versus Normative Databases: Case Studies of Clinical Q Assessments

Paul G. Swingle, PhD

For clinicians, the most accurate databases are clearly clinical. Normative databases are far less accurate. The fundamental organizing concept of the normative database for the clinical practitioner is, simply stated, logically incorrect.

The organizing concept for normative databases is that one can identify a group of individuals who are symptom free and therefore have "normal" functioning neurology. This group of symptom-free individuals then serves as the comparative database to identify those who are statistically discriminant. The statistical departures from the normative database define the anomalous neurological condition that is associated with the client's clinical condition. This concept is also logically incorrect.

The reason that normative database treatment recommendations are so often incorrect is because the fundamental premise is wrong. Symptom-free individuals may well have predispositions to conditions that have not manifested. The data are quite clear and we have definitive evidence for this that spans decades.

Let us simply take the example of heritability data for schizophrenia (similar data are available for other conditions as well, such as vulnerability to PTSD and Bipolar Disorder). As the data in Table 1 indicate, if one monozygotic twin has diagnosed schizophrenia the probability that the second identical twin will have schizophrenia is about 50%. So, the schizophrenic ends up in the ClinicalQ database. But, the interesting statistic is that 50% will not! Where do we find the 50% without manifested schizophrenia, but obviously with the same genetic load? In the normative databases. So, clearly, the organizing concept for normative databases, at least for clinicians, is incorrect. Normative databases so constituted ignore basic psychopathology and basic biology. Every person has predispositions. Predispositions to anxiety, depression, emotional volatility, and the like. However, many of these predispositions are not manifest at any particular time. In general, clinicians understand that one needs a trigger to "turn-the-key" to manifest a neurological predisposition.

Table 1: Heritability Statistics on Schizophrenia

Genetic Predispositions	
Monozygotic Twins	30-50%
Dizygotic Twins	15%
Siblings	15%
General Population	1%
Adopted-Biological Relatives with Schizophrenia	
Adoptee with Schizophrenia	13%
Adoptee without Schizophrenia	2%

Source: **Gottesman, I. I. (**1991**)** *Schizophrenia Genesis: The Origin of Madness. New York: Freeman.*

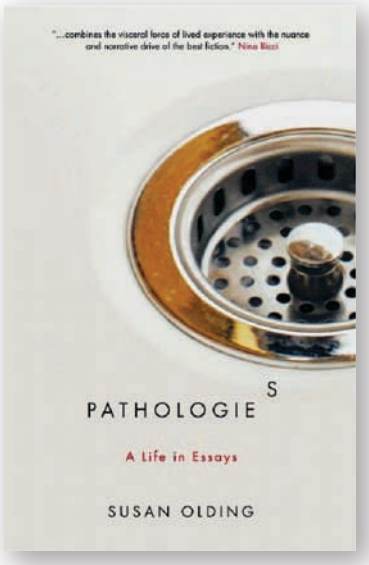
These logic considerations are well known and, surprisingly, ignored by the developers of the normative databases. If in the normative database one has subjects with non-manifested predispositions, then statistically one can expect very poor discrimination. The normative databases are going to be statistically blind to manifested predispositions that bring clients into our offices. Related to this problem is that the data collection procedures ignore conditions that expose clinically relevant information. An example of this is ignoring the Alpha blunting response that is the cardinal marker for exposure to severe emotional stress (Swingle, 2013).

Clinical databases permit quite remarkable accuracy in revealing the fundamental causes and exacerbating factors contributing to a client's morbidity. We, of course, ask clients about their condition, but these reports are often quite inaccurate as to causality. A good example is the client report of depression that is more accurately despair associated with debilitating anxiety. Treating "depression" instead of "anxiety" with antidepressant medications or left frontal Alpha brainwave amplitude suppression is therefore likely to be marginally effective because the wrong condition is under treatment.

The accuracy of the ClinicalQ database is nicely captured in the following

excerpt:

Susan Olding, from her book *"Pathologies"* Freehand Press.



Desperate, determined, undeterred by cost or lack of insurance coverage, undismayed by the doubts of conventional physicians … I switched off my cell phone at the threshold of Dr. Swingle's office and carried my daughter (into the office)...

I had brought a medical and developmental history—the long litany of concerns that had brought us to his door—but Dr. Swingle waved the papers aside without even looking at them. Instead, he ushered Maia toward a computer screen… [and] …fixed a couple of delicate wires to her ears…

Then Dr. Swingle sent Maia to the "treasure chest" in the waiting room. He stared at the printout in his hand. "Here," he said, and he pointed to an outline of the brain, "these numbers imply trauma." He shrugged, palms up, waiting for my response. I nodded. "And here," he continued, "too much Theta. This is the hyperactivity people associate with ADHD… There was more: extreme stubbornness, a tendency to perseverate, lapses of short-term memory, attachment disorder, inability to read social cues, emotional reactivity, tantrums, explosions. One by one he read the ratios, divining my daughter's character more quickly, more accurately than any professional I'd yet encountered.

The assessment described by Susan Olding is the ClinicalQ that is based on 6½ minutes of recording time. The clinical database then o ers direction for the clinician's probing of the client regarding anomalies in functioning that may be the bases for the clinical condition.

The Case of the Kelly Family:

Mrs. Kelly brought in her two children, Jane who was seven years of age and Martin who was nine years of age, for treatment of what her family physician thought was Attention De cit Disorder with both children. Fortunately for Mrs. Kelly, her family physician was strongly opposed to medicating children for ADHD, unless absolutely necessary. It may well have been that this very vigilant physician was suspicious that the problem with the children resided in problems with the family and that medicating this problem would be totally inappropriate.

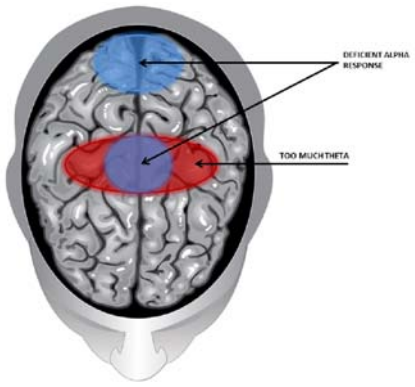
The following are the data summaries for the ClinicalQ evaluations. The data critical to this discussion have been highlighted in red and on the schematic brain diagrams those areas have been highlighted. There are other areas in these summaries that are of clinical relevance but will not be addressed for the purposes of this discussion.

Figure 1 shows the initial intake clinical EEG assessment of Jane. Although Jane shows the EEG feature associated with ADHD (Theta/Beta ratio of 2.86 at location Cz), the feature of particular concern is the blunting of the Alpha response at both locations Cz and 01 (circled numbers—increase in Alpha should be at least 30% at location Cz and at least 50% at location O1). As the data show, the Alpha response was 18.3% at Cz and slightly negative at location O1. These are the markers for exposure to severe emotional stress (Swingle, 2013).

One EEG feature we often nd with children who have severe attention problems is that they show the trauma marker. It is possible that the trauma is associated with fear of failure and humiliation in school associated with their attention and/or learning problems. However, whenever we see this pattern in children, we always determine if the child is being exposed to marked emotional stressors. This can be bullying, it could be family strife or it could

Figure 1: *ClinicalQ of seven year old Jane*

Cz	xΘ/β	RΘ/β	α↑%	α↓%	TA
	2.86	3.12	18.3	1.6	58.5
O1	α↑%	α↓%	EO	EC	
	-2.0	NA	2.33	1.69	
	Θ/β	Θ/α			
F4	2.53	1.87			
F3	2.98	1.91			
F=F%	β	α	Θ		
	11.6	10.2	4.9		
Fz	Dz	Hβ/β	∑	L/H	Θ/β
	17.8	0.70	15.3	1.98	3.0



be some form of abuse. So in addition to neurotherapeutic treatment for the ADHD, we have to determine the cause of the Alpha blunting.

Mrs. Kelly had brought in both of her children at the same time for back-to-back appointments for the brain assessment. She was told by her physician that both children may have an ADHD problem.

As we can see in Figure 2, Martin's EEG looked remarkably similar to Jane's. Both had the marker for ADHD (Theta/Beta ratio of 2.98 at location Cz). In addition, both had markers for exposure to severe emotional stressors. The Alpha blunting was in both locations Cz and O1 (Alpha response of zero at Cz and 39.6% at O1), just as with Jane.

There are several important issues to consider here. First, given that we are seeing this marker with both children, it is possible that we are dealing with a genetic factor. Although Alpha blunting is highly correlated with exposure to severe emotional stressors (Swingle, 2013), nonetheless, although rare, we do find it in situations in which there is no apparent present or historical exposure to emotional trauma.

The second issue is how we approach the mother in a manner that is not going to make her bolt from the therapeutic situation or make her severely distraught about her children's well-being. If there is no context in which this parent is aware of severe emotional stress, this kind of information can be clearly distressing. Parents immediately think about bullying, sexual predators, and other forms of abuse to which children might be exposed. It is extraordinarily important for the therapist to be able to deal with this situation in a manner that is rational and systematic.

The third issue is that healthcare providers have an obligation to report to the authorities any potential harm to a child. However, we have no direct evidence of this other than the EEG data. Recognizing that the parent may be the perpetrator, careful and prudent probing of the parent regarding the various conditions under which the emotional stress may occur, or have occurred, is required.

When I broached the subject of the children showing signs of being exposed to severe emotional stress, Mrs. Kelly broke down and admitted that there were severe problems in the family. According to Mrs. Kelly, her husband vacillated between severe depression and severe emotional abuse. He "flew off the handle" with minimal provocation, was heavily medicated, and she felt that the children were severely disturbed by her husband's behavior. Mrs. Kelly agreed to let me measure her brainwave activity. Her ClinicalQ is shown in Figure 3.

As can be seen in Figure 3, Mrs. Kelly's EEG shows the marker for exposure to severe emotional stress, just as her children's. Her brain assessment also shows mild markers for problems with attention, again, just as her children, so she may be the source of the ADD markers that we find in her children. There are several other features of Mrs. Kelly's EEG that are important to note. The first is that she has a major marker for predisposition to depressed mood states; the amplitude of Beta activity is

Figure 2: *ClinicalQ of nine year old Martin*

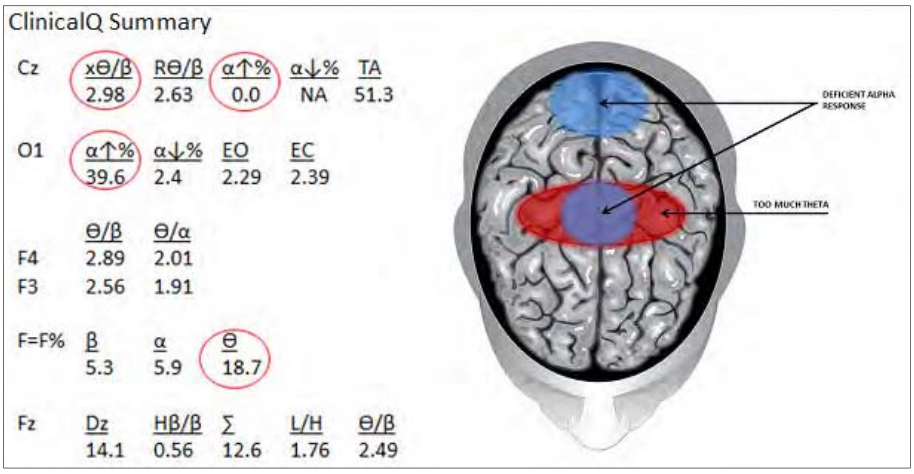
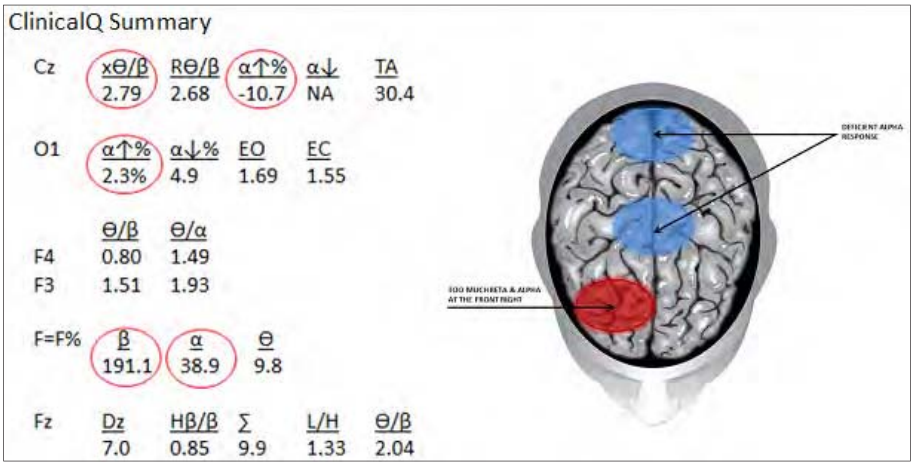


Figure 3: *Mrs. Kelly's ClinicalQ*



markedly greater in the right prefrontal cortex relative to the left. Whether the depression of Mrs. Kelly contributes to the family dynamic issue or whether it is the result of her exposure to the abusive behavior of her husband, nonetheless, children whose mother is severely depressed are profoundly more likely to have emotional behavior problems.

The second feature in Mrs. Kelly's ClinicalQ is that there is a marked elevation of Alpha amplitude in the right prefrontal cortex relative to the left. In children, we find this imbalance is often associated with oppositional and defiant behavior. In adult populations, we often find this disparity with individuals who are going through severe interpersonal problems such as marital discord, divorce, conflict in the workplace, and so forth.

It seems obvious that we are dealing with a family in crisis. Both of the children and Mrs. Kelly show markers for exposure to severe emotional stress (the blunted Alpha trauma markers). Mrs. Kelly shows a major marker for predisposition to depressed mood states and, on her intake self-report assessment, she describes herself as being one who falls into depression easily. Mrs. Kelly's description of her children's behavior, likewise, suggests that these children have some emotional difficulties. She describes Jane as easily upset, quick to anger, and unable to engage in cooperative play because she always must win. The latter condition, a child who must always win or they will refuse to play, is a cardinal marker for children who feel insecure and have negative self-regard. This is a characteristic often found with adopted children.

Mrs. Kelly describes Martin as being very anxious, unresponsive to others' feelings and, importantly, she describes him as having behaviors associated with Internet addiction (addiction to video games). Internet addiction is an extraordinarily serious problem that is largely unrecognized by parents.

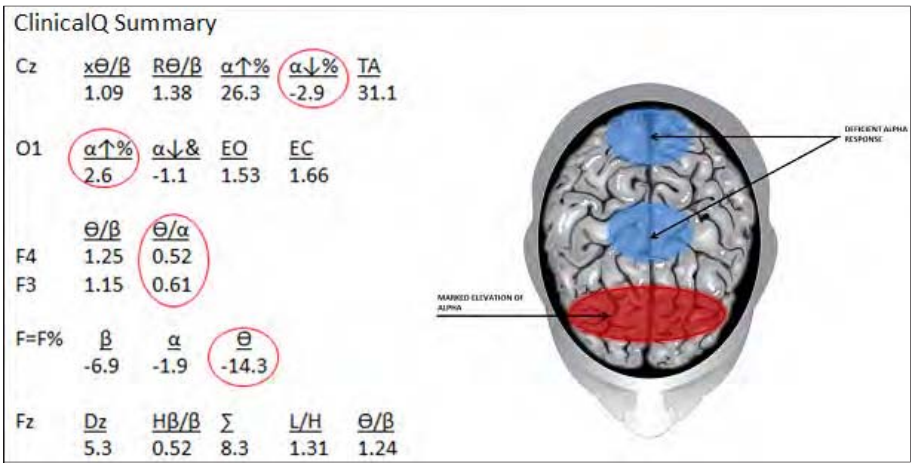
Although both children show the neurological pattern associated with common ADD, the central problems here are emotional and appear to result from family strife as opposed to being associated with attention deficit disorder. It is, of course, very likely that the ADD is contributing to the family strife. Such children require more assistance and more monitoring to complete their homework assignments and they are usually experiencing difficulties in school, which puts further pressure on the family.

Our ability to diagnose the problem with the Kelly children as being primarily a problem with family strife testifies to the remarkable facility of the ClinicalQ EEG as a diagnostic instrument. Without any input from the parents, we were able to determine that family difficulties were giving rise to the problems that were affecting the children. Recall, the children were brought in for treatment because of difficulties in school. The assumption was that the children had some form of ADHD or other learning problem. This testifies to the accuracy of the EEG diagnostic procedure. Most importantly, however, it points out that other therapeutic strategies must be put in place to assist this family. Changing the neurology of the situation will be important but it will be a minor component associated with the treatment of these children. It is extremely important to understand that family therapy and treatment of the parents will be equally as important as any kind of neurological work that we might do with the children.

We were most fortunate that Mr. Kelly not only recognized that he had serious problems but also recognized and acknowledged that his behavior was likely to be seriously affecting family functioning in a negative way. He further admitted that he thought his psychological problems were very likely interfering with the children's ability to perform efficiently in school. Mr. Kelly willingly came in for the ClinicalQ EEG assessment, the results of which are shown in Figure 4.

Mr. Kelly described himself as follows: "I fly off the handle at minor problems. I'm anxious, depressed and fatigued. I am on major medications including Wellbutrin, Cipralex and Ativan that are not very effective. And I've been on other mixes of medications, all of which may have helped somewhat, but eventually lost their effectiveness.

Figure 4: *Mr. Kelly's ClinicalQ*



I know that my behavior has seriously affected my marriage, my children, and my wife."

Although this situation is severe and complex, I am really tempted to take out the "Cured" stamp at this point! Whenever one has clients who are willing to present themselves for treatment, are open and candid about their problems and their potential influence on other individuals, the prognosis is extraordinarily good for a favorable outcome. We will have challenges in dealing with this situation, of course. The challenges are not only neurological but behavioral in nature. Martin, for example, has developed a dependency on video games. This provides an escape and stimulation for this child and it will be very difficult to wean him from this addictive behavior.

Mr. Kelly has a long history of dysregulated behavior and a long history of being medicated. Titrating him off the medications will also be a challenge. Nonetheless, given the data that we have on the neurological condition of each family member and the willingness of both parents to be candid about their condition and enthusiastic about presenting themselves for treatment, the prognosis bodes well for positive outcome.

Mr. Kelly's ClinicalQ indicated a number of anomalies that are interacting and exacerbating each other. That is, they are synergic in a negative sense. Mr. Kelly does not have the marker for the form of ADHD that the children show and, to some extent, Mrs. Kelly also shows. That form of ADHD is characterized by elevated slow frequency, primarily over the central part of the brain.

However, Mr. Kelly has an ADHD condition and a particularly nasty form, at that. He has marked elevation of Alpha amplitude in the front part of the brain (locations F3 and F4). The high frontal Alpha form of ADHD is characterized by problems with planning, organizing, sequencing, and following through on things (Swingle, 2008). However, more importantly in this case is that high amplitude frontal Alpha is associated with emotional dysregulation. These individuals can have marked emotional volatility, problems with emotional impulse control, and difficulty sustaining emotional stability. Clients with this neurological condition are often diagnosed with bipolar disorder, personality disorder, and anxiety disorders in addition to ADHD.

Mr. Kelly also has a mild marker for depressed mood states in which the amplitude of slow frequency Theta is greater in the left front part of the brain as opposed to the right. The predisposition to depression involves a number of conditions that result in the right prefrontal cortex being more active (aroused) than the left. Slow frequency (Theta) amplitude was greater in the left relative to the right in Mr. Kelly's situation. When slow frequency amplitude is greater in the left relative to the right, then the right frontal cortex will be more active than the left, a cardinal marker for depression.

Mr. Kelly has a few other situations that are giving rise to some difficulty. There is a deficiency of slow frequency amplitude relative to fast frequency amplitude (the Theta/Beta ratio) in the occipital region of the brain. Low ratio of the strength of Theta relative to Beta is associated with poor stress tolerance, predisposition to anxiety, sleep quality problems, fatigue, and often leads to self-medicating behavior such as excessive use of alcohol or prescription medications. Mr. Kelly's description of himself included many of the above. He described himself as "flying off the handle" at minor provocation, being anxious, depressed and fatigued. Although individuals with low Theta/Beta ratios in the occipital region of the brain have a predisposition for self-medicating behavior, Mr. Kelly denied that he had any difficulty with alcohol. His wife substantiated this. He did comment, however, that he had a very long history of use of prescription medications.

Finally, we note that Mr. Kelly also has the marker for exposure to severe emotional stress. It is not an uncommon finding that the individual whose behavior is the fundamental cause of strife in the family also shows a marker for emotional trauma. It is difficult to know whether Mr. Kelly's trauma markers are associated with his present situation (family in turmoil) or whether this is an historical condition. Mr. Kelly's emotional difficulties may be associated not only with neurological conditions but also with the fact that he had been exposed to severe emotional trauma earlier in his life. Mr. Kelly did admit that he came from an extraordinarily violent household. During his early childhood, he lived in a constant state of fear and anxiety. Hence, it is not unlikely that Mr. Kelly's trauma markers are associated with childhood exposure to severe stress, whereas the markers we find in the brain assessments of the children and Mrs. Kelly reflect strife within the family, caused largely by Mr. Kelly's behavior.

Consider the child shown in Figures 5a and 5b. This child was brought in for treatment of an attention problem. He was having significant problems in school and was judged to have many of the symptoms associated with ADHD (inattentive type). The figures are the actual output from the ClinicalQ for this child. Again, there are many features of this profile that are clinically important but we will limit the discussion to those associated with the suspected bullying. This child does show a minor marker for ADHD. The Theta/Beta ratios at Cz are

a bit elevated. However, this child is showing a trauma marker at Cz (indicated in red), a marker for reactive depression at F3/F4 (Alpha is 45.2% greater in the left relative to the right), and a marker for emotional volatility (F4 Theta is considerably greater than at F3). So, the hypotheses are that this child has been

Figure 5a: *Nine year old male child—potential bully victim*

CZ	VALUES	% CHANGE
EO Alpha	8.61	
EC Alpha	10.23	
% Change EO to EC Alpha > 30%		18.78%
EO Alpha Recovery	9.27	
% Change EO – Alpha Recovery		7.63%
Theta Amplitude EO	15.76	
Beta Amplitude EO	6.5	
EO Theta/Beta	2.47	
Theta Amplitude Under Task (UT)	13.69	
Beta Amplitude UT	5.89	
UT Theta/Beta	2.32	
% Change T/B EO to T/B UT		-6.45%
% UT Beta Increase		-10.29%
Total Amplitude	30.65	
Theta Amplitude preceding Omni	14.42	
Theta Amplitude with Omni	13.15	
% Change in Theta with Omni		-9.68
Alpha Peak Frequency EC	10	
Alpha Peak Frequency EO	9.8	
Theta/SMR EC	3.15	

01	VALUES	% CHANGE
Alpha EO	6.16	
Alpha EC	12.06	
% Change in Alpha EO to EC		95.84%
EO Alpha Recovery	5.7	
% Change EO – Alpha Recovery		-8.16%
Theta Amplitude EO	10.09	
Beta Amplitude EO	5.17	
Theta/Beta EO	1.95	
Theta Amplitude EC	10.46	
Beta Amplitude EC	6.99	
Theta/Beta EC	1.5	
% Change T/B EO to T/B EC		-30.21%
Alpha Peak Frequency EC	10	
Alpha Peak Frequency EO	9.9	

Figure 5b: *Nine year old male child—potential bully victim*

F3 & F4 (ALL EC)	VALUES F3	VALUES F4	% Difference F3–F4
Theta Amplitude EC	10.10	16.93	
Beta Amplitude EC	6.37	7.06	
EC Theta/Beta	1.59	2.41	
% Diff F3 T/B – F4 TB EC			50.93%
Theta Amplitude EC	10.10	16.93	
Alpha Amplitude EC	13.47	9.28	
EC Theta/Alpha	0.75	1.83	
EC Total Amplitude	29.95	33.26	
*F4><F3 Beta	6.37	7.06	10.74%
*F4><F3 Alpha	13.47	9.28	-45.20%
*F4><F3 Theta	10.10	16.93	67.56%

FZ (ALL EC)	VALUES
Delta (2Hz)	10.05
HiBeta Amplitude	3.89
Beta Amplitude	6.15
HiBeta/Beta	0.63
Sum HiBeta + Beta	10.04
LoAlpha Amplitude	5.20
HiAlpha Amplitude	3.61
LoAlpha/HiAlpha	1.44
Alpha Peak Frequency	9.40

or is being exposed to significant emotional stressors, that he is experiencing a reactive depression (perhaps related to the emotional stressors) and that he is emotionally volatile (and hence a "sitting-duck" for a bully). This child, if these hypotheses are correct, cannot pay attention or do well in school because he is afraid! Probing the child and the parents revealed that the child was being severely bullied, was afraid to tell his parents because of the bullies' threats, and he was emotionally volatile (cried frequently over minor issues). The parent corrected the bully issue at school and we did some minor braindriving neurotherapy to improve the minor ADHD problem.

Summary and conclusions

As the above cases indicate, for those of us who actually treat patients/clients, qEEG statistical discriminations based on normative databases are not adequate for clinical practice. Discriminations based on the normative databases are simply statistically blind to many of the important neurological features associated with the clinical condition of clients. Clinical databases, such as that used in the ClinicalQ, are far more efficient for

identifying manifested predispositions and experiential factors that are fundamental to the efficient neurotherapeutic treatment of our clients. Clinical databases are also far more efficient at identifying conditions that require therapies other than neurotherapy.

The ClinicalQ identified that the cause of the Kelly children's academic difficulties were, if not caused by, certainly markedly exacerbated by, not their ADHD, but Mr. Kelly's ADHD! Mr. Kelly was being pharmaceutically treated for the wrong condition. Hence proper treatment for Mr. Kelly turned out to be the effective treatment for his children's academic difficulties. The children did have some neurotherapy to correct the minor excesses of slow frequency amplitude associated with the inattentive form of ADD. And, weaning Martin off of his addiction to Internet gaming was difficult and required some parenting assistance.

Likewise, in the case of the bullied child, the normative databases are completely blind to this condition. Treating this child either pharmaceutically or with neurotherapy for ADHD would have been largely ineffective because the primary cause of this child's academic difficulties was that he was afraid.

The normative database qEEG provides very useful and important information. For efficient clinical practice, however, it must be augmented by discriminative comparisons with clinical norms.

About the Author

Paul G. Swingle, PhD, RPsych, was Professor of Psychology at the University of Ottawa prior to moving to Vancouver. A Fellow of the Canadian Psychological Association, Dr. Swingle was Lecturer in Psychiatry at Harvard Medical School, and during the same time period was Associate Attending Psychologist at McLean Hospital (Boston) where he also was Coordinator of the Clinical Psychophysiology Service. Dr. Swingle was Chairman of the Faculty of Child Psychology at the University of Ottawa from 1972 to 1977 and Clinical Supervisor from 1987 to 1997. He has also taught at McGill University, Dalhousie University and McMaster University. He is a Registered Psychologist in British Columbia and is certified in Biofeedback and Neurotherapy. Since 1997 he has been in private practice in Vancouver, British Columbia. His newest book "Biofeedback for the Brain" was published by Rutgers University Press.

References

Gottesman, I. I. (1991) Schizophrenia Genesis: The Origin of Madness. New York: Freeman.

Olding, S. (2008) Pathologies. Calgary, AB: Freehand Press.

Swingle, P.G., (2013) The effects of negative emotional stimuli on alpha blunting Journal of Neurotherapy, 17(2), 133-138.

Swingle, P.G. (2010) Biofeedback for the Brain. Revised paperback edition. Rutgers University Press. New Brunswick, NJ.

Swingle, P.G. (2008). Basic Neurotherapy: The Clinician's Guide. Vancouver, BC. Soundhealth.

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pires to explore the relationship of neural networks and their function using techniques in brain imaging

References

Anokhin, A., & Vogel, F. (August 1996). EEG Alpha rhythm frequency and intelligence in normal adults. Intelligence, 23, 1, 0160–2896.

Baker, S.N. (December 2007). Oscillatory interactions between sensorimotor cortex and the periphery. Current Opinion in Neurobiology, 17, 6, 649–55.

Barry, R.J., Clarke, A.R., Magee, C.A., & Rushby J.A. (December 2007). EEG differences between eyes closed and eyes open resting conditions. Clinical Neurophysiology, 118, 12, 2765–2773

Bazanova, O. M., & Vernon, D. (May 1, 2013). Interpreting EEG alpha activity. Neuroscience & Biobehavioral Reviews, 2.

Castelhano, J., Rebola J., Leitao B., Rodriquez E., & Castel-Branco M. (June 13, 2013). To Perceive or Not Perceive: The Role of Gamma-Band Activity in Signaling Object Percepts. Plos One, 8, 6, e66363.

Downar, J., & Daskalakis Z.J. (May 2013). New targets for rTMS in depression: A review of convergent evidence. Brain Stimulation: Basic, Translational, and Clinical Research in Neuromodulation, 6, 3, 231–240.

Grandy, T. H., Werkle-Bergner, M., Chicherio, C., Lovden, M., Schmiedek, F., & Lindenberger, U. (October 1, 2013). Individual alpha peak frequency is related to latent factors of general cognitive abilities. Neuroimage, 79, 10–18.

Jensen, O., Kaiser, J., Lachaux, J. (May 17, 2007). Human gamma-frequency oscillations associated with attention and memory. Trends in Neuroscience. 30, 7, 317–324.

Johnstone, T., van Reekum, C. M., Urry, H. L., Kalin, N. H., & Davidson, R. J. (August 15, 2007). Failure to Regulate: Counterproductive Recruitment of Top-Down Prefrontal-Subcortical Circuitry in Major Depression. The Journal of Neuroscience, 27, 33, 8877–8884.

Kirschfeld, K. (January 01, 2005). The physical basis of alpha waves in the electroencephalogram and the origin of the "Berger effect." Biological Cybernetics, 92, 3, 177–85.

Lutz, A., Greischar, L. L., Rawlings, N. B., Ricard, M., Davidson, R. J., & Singer, B. H. (November 16, 2004). Long-Term Meditators Self-Induce High-Amplitude Gamma Synchrony during Mental Practice. Proceedings of the National Academy of Sciences of the United States of America, 101, 46, 16369–16373.

Ros, T., & Gruzelier, J.H. (2011). The immediate effects of EEG Neurofeedback on cortical excitability and synchronization. (1 ed., p. 381–402). Burlington, MA: Academic Press.

Spronk, D., Arns, M., & Fitzgerald, P. B. (2011). Repetitive transcranial magnetic stimulation in depression: protocols, mechanisms, and new developments. (1 ed., p. 257–291). Burlington, MA: Academic Press.

Sauseng, P., Klimesch, W., Gerloff, C., & Hummel, F. C. (January 01, 2009). Spontaneous locally restricted EEG alpha activity determines cortical excitability in the motor cortex. Neuropsychologia, 47, 1, 284–288.

Wyrwicka, W., & Sterman, M. B. (December 26, 1968). Instrumental conditioning of sensorimotor cortex EEG spindles in the waking cat. Physiology & Behavior, 3, 5, 703–707.