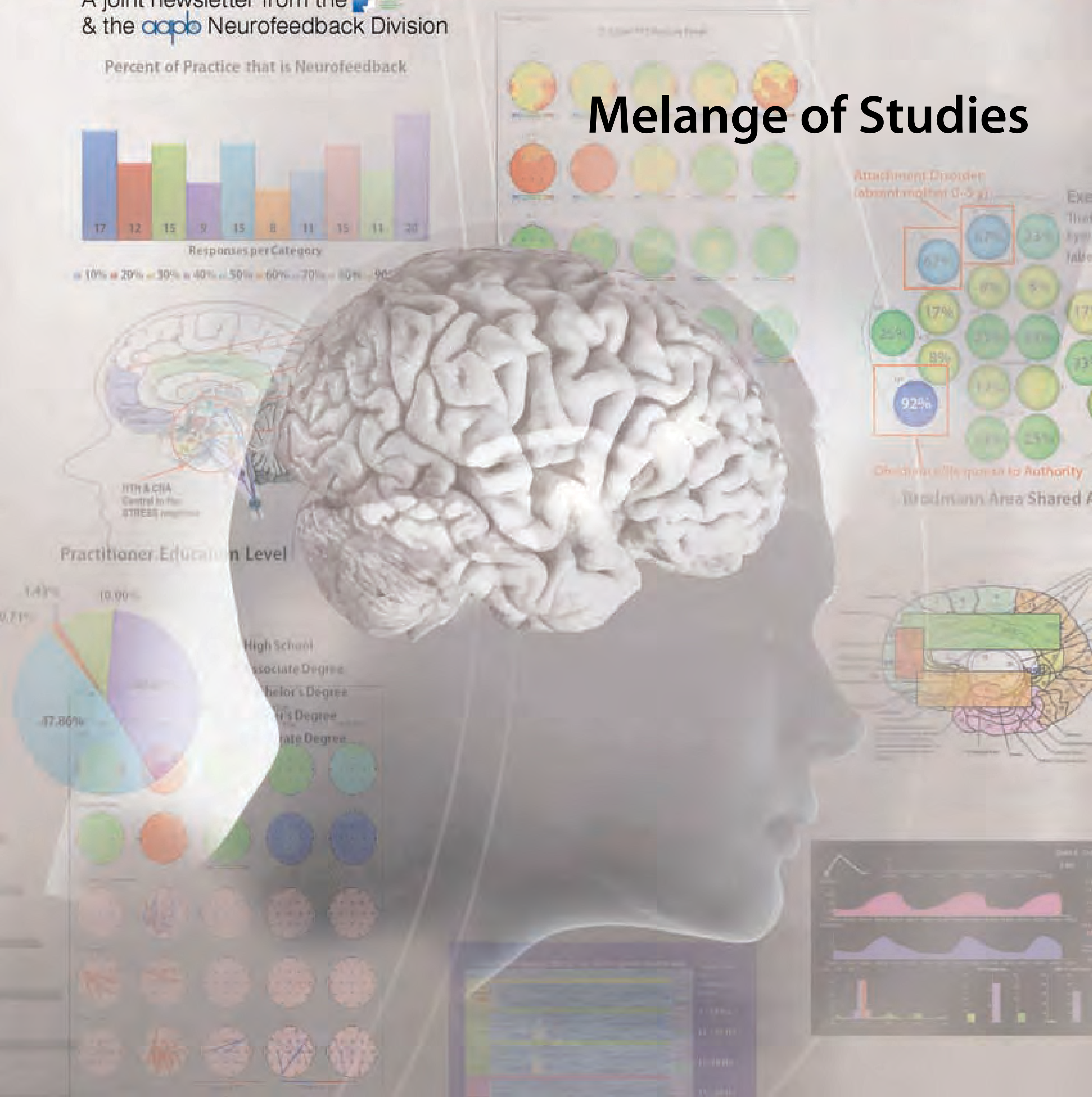
NEURO CONNECTIONS

Winter 2013 **Newsletter**

A joint newsletter from the ISNR
& the aapb Neurofeedback Division

Melange of Studies



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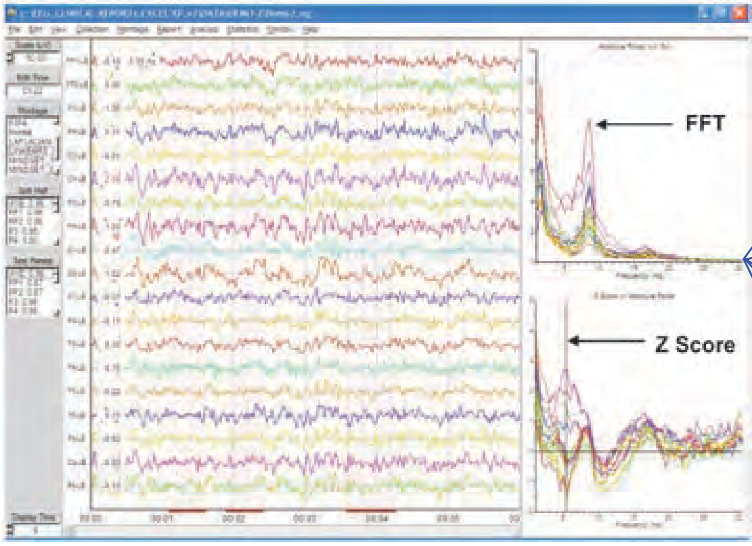

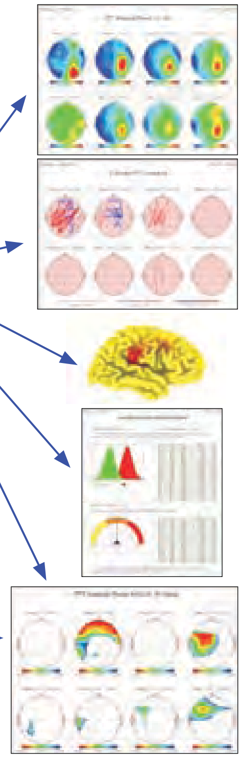

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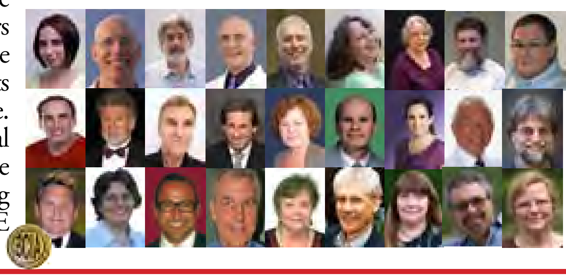

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NEURO CONNECTIONS

A joint newsletter from the ISNR & the aapb Neurofeedback Division

Contents

ONLINE SUPPLEMENT

Online Supplement

References cited in the following papers may be found in the *NeuroConnections* Supplement at http://isnr.org/neurofeedback-info/neuroconnections-newsletters.cfm.

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Letter from ISNR President

Rex Cannon, PhD

Salutations to all of the ISNR members. It was great to see many of you at the 21st annual conference in Dallas, TX. For those not able to attend, we missed you and hope to see you in Pittsburgh (or San Diego, hopefully) next year. Despite a transitional state for the board and conference committee with the addition of a conference vendor, although lacking in some areas, the conference turned out well because of its members. It is true that we were missing many components seen in previous years, such as symposia, panels, debates, and so forth. We apologize for this development and it is being corrected. We will be seeking a relationship with a new vendor for future conference fundamentals, while maintaining a committee devoted to oversight, program compliance, and efficiency. We want to obtain the best educational (research) and clinical presenters for our membership, as well as visitors to our conference.

Dr. Leslie Sherman stepped down from his role as conference chair. We wish to thank him for his noted efforts for ISNR and its continued growth. We are assembling a conference committee for the upcoming year and will advise the membership once this group is in place.

I would like to take the opportunity to thank past president, Randall Lyle, for his efforts in the initiation of a strategic plan for our organization, in addition to his efforts for ISNR during our growth and transition. Randy also devoted much time and effort to the *Journal of Neurotherapy* and the formation of our new open access journal ***Neuroregulation*** which is scheduled for its inaugural issue in January of 2014. This gratitude also extends to those board members who completed terms of service this year—Richard Davis, Sarah Prinsloo, and John Davis. I am excited to welcome our incoming board members, President Elect, *Rob Coben*; Secretary, *Joseph Barr*; Member at Large, *Kirk Little*; and International Member at Large, *Pedro Delgado*. This leads into another important issue for your Board and the membership in general, we have numerous committees that are not populated. Service is a great way to give back as well as provide valuable feedback to your Board and the membership body on important topics such as insurance reimbursement, marketing strategies, fundraising efforts, directions for growth, and numerous others. Committee service is also a wonderful way to expand your network of colleagues, and make professional friendships grow. We want members involved and would greatly appreciate your involvement. It is important to keep in mind that the ISNR Board is directly responsible to the body it serves (its members). If you want to be involved in service to ISNR please contact any Board member or the executive director (ED) to be advised of our needs.

Often the ISNR Board has limited knowledge about the activities, needs, and successes of local neurofeedback organizations. We would like to keep apprised of what is happening at the local level to be able to support and help, if needed, as well as report important activities to our members. For example, we were unaware of work by the Biofeedback Society of California in working with state legislators to draft a bill including applied psychophysiology (biofeedback and neurofeedback) as part of an EEG technologist's certification process. Jay Gunkelman is working with Bradley A. Hix, governmental and grassroots Advocacy Manager for ASET, The Neurodiagnostic Society, to bring this important designation to life. ISNR will

As we grow it is important to remember that we are ONE organization with a common purpose (the research and clinical advancement of applied neuroscience and neurofeedback).

ISNR Mission Statement

To promote excellence in clinical practice, educational applications, and research in applied neuroscience in order to better understand and enhance brain function. Our objectives are:

- Improve lives through neurofeedback and other brain regulation modalities
- Encourage understanding of brain physiology and its impact on behavior
- Promote scientific research and peer-reviewed publications
- Provide information resources for the public and professionals
- Develop clinical and ethical guidelines for the practice of applied neuroscience

AAPB Neurofeedback Division Mission Statement

To improve human welfare through the pursuit of its goals. The specific goals are:

- The encouragement and improvement of scientific research and clinical applications of EEG technology and neurofeedback.
- The promotion of high standards of professional practice, peer review, ethics, and education in neurofeedback.
- The promotion of neurofeedback and the dissemination of information to the public about neurofeedback.
- The division is organized for the purpose of carrying on educational and scientific objectives and is not to be operated for profit.

do what it can to be of assistance to this important process.

ISNR has grown since my first visit to a conference in 2004. As a student, I found the collegial atmosphere refreshing and inviting. The research and clinical presentations were extremely valuable in my academic pursuits. Of particular note, over the course of years, I have found all members to be inviting and willing to share their experience with encouragement. As we grow it is important to remember that we are ONE organization with a common purpose (the research and clinical advancement of applied neuroscience and neurofeedback). There is a place for healthy competition between our members. However, we need to hold fast to principles before personalities. We must continue to grow and expand outside of our organization. We know the potential of neurofeedback and the scientific basis behind it. It is now time for the world to share in our knowledge. We must put our focus on this growth and increasing our presence in other professional organizations. It is important to maintain our collegial atmosphere by encouraging, teaching, and supporting each other across all personal or financial interests.

I would encourage any member with an issue (positive or negative) or great ideas to contact me or the Board directly. It is important to maintain an open dialogue with our members and we shall abide by this principle. If you have seen the new brochure, thanks to Kathy Abbott and the marketing committee. They have worked extremely hard on this project and turned out a great product! We hope to continue on with numerous projects in the coming year to make your ISNR better than ever. If you were present at the banquet, then you felt the electricity in the air given Joe Kamiya, Barry Sterman, and Joel Lubar were in the room at the same time. This was a fantastic experience!

In case you are not aware, ISNR is on Twitter (ISNRORG), Linkedin (ISNRORG) and Facebook. Follow and like us! It is my hope to inspire ISNR members and those boards that follow. We are a great society of scientist practitioners with much to offer the rest of the world. We will no longer sit idly by while poor research defames our methodology. We will not be intimidated by larger societies or professional organizations. We will not tolerate contempt prior to investigation. We will not accept opinion or questionable science as truth. Our members are a bright light that will outshine every dimming candle. We are ISNR!

Rex Cannon, PhD

Letter from AAPB NFB Division President

Richard Soutar, PhD

As neurofeedback emerged and distinguished itself from biofeedback and the ISNR was formed, there was both hope and conviction that NFB would be even more powerful than biofeedback with respect to a wide variety of disorders. It was expected by many that somehow changing the brain could also change the biochemistry related to disorders at many levels, or at least Sterman's research suggested it could have a very powerful impact. In my readings of the literature in the '90s, I noted NFB was widely characterized and embraced as a unique standalone solution to a wide range of disorders. That theme appears to maintain a strong foothold within the field today, despite growing evidence to the contrary. Although there are many sophisticated practitioners who are keeping pace, my experience as an international mentor for the past decade has confronted me with evidence that there is a large body of practitioners who are not aware of the emerging literature on this topic. Having been a professor of psychology, I am also very aware of how focused academic training in counseling and psychology is on psychological causes and how it underestimates the importance of other biological sources of mental disorder. In the 2006 Sand Diego Conference I did a workshop on qEEG and behavior; over 65 people showed up. The following year I did a workshop in San Antonio on qEEG and nutrition and only 8 people showed up. This year over 35 people showed up for our workshop on qEEG and metabolics. So awareness may be growing.

Recently, research has revealed to us that gastrointestinal disorders can result in anxiety and depression and that psychological disorder can issue from the bottom up. New models of top-down psychogenic vs. bottom-up physiogenic anxiety and depression are emerging in the scientific literature. Indeed, many of us have found it easier to have a client obtain thyroxine to reduce high amplitude slow alpha than to endlessly train alpha down with minimal results or chase down new protocols and equipment that can't do the job either. It is difficult to train against the physiological consequences of hypothyroid and the associated depression. It would seem as time passes that clinical experience indicates NFB is not as standalone

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.

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as we might like. Many are being driven by clinical experience toward the more bio-psycho-social paradigm of psychophysiology. As a consequence, more and more clinicians are adding HRV to their toolbox and finding it a powerful adjunct. I have been using it for over fifteen years and can't imagine working without it. It has replaced many of the other peripheral measures I used in the past because I believe it gave me clear indicators of both eustress and distress. Likewise, I have felt compelled to use amino acid tests and hair analysis along with my qEEGs to help identify and resolve metabolic limitations to NFB. I have been doing workshops on this topic at ISNR since 2008.

Today, I work with a team of other professionals who assist my clients in having the maximum resources available to allow them the optimal response to the NFB challenge of operant conditioning. I know of hundreds of other clinics around the country who are following in this path. The best responses to NFB seem to occur when family systems and physiology are optimally managed. I think the recent special issue in Biofeedback Magazine on HRV dovetails nicely with the highlighted article on HRV in this issue. I believe that my training in biofeedback from Marjorie Toomim provided me with a much deeper understanding of NFB than if I had entered the field without it. It changed the way I did NFB at the very most basic level. It helped me understand the fundamental importance of Barry Sterman's arousal model of NFB as well. I have kept my ear to the rail at AAPB conferences ever since. Many of us have been doing biofeedback and neurofeedback for many years, and the Thompsons have championed and developed this well-rounded approach more than most. I believe it is very important for all of us in NFB to try to integrate these emerging biofeedback technologies into our practice. Clients can even take them home and compliance improves outcomes!

Richard Soutar, PhD, BCN

Letter from ISNR Executive Director

Cindy A. Yablonski, MBA

An Invitation to Join or Renew Your ISNR Membership In 2014

I would like to thank everyone who attended the 21st Annual ISNR Conference in Dallas, Texas this September for their continued support. This year's annual conference included over 50 general sessions, and 44 workshops, along with an impressive array of speakers, topics and networking opportunities. I would especially like to recognize the Conference Committee for its hard work in designing and implementing an exciting and informative agenda. Attendance this year exceeded our expectations in sheer numbers and in diverse backgrounds. The gathering also brought together an exceptional mix of vendors showcasing the latest technology and trends.

Throughout 2013, ISNR has undertaken many positive changes on behalf of our members. . .

Throughout 2013, ISNR has undertaken many positive changes on behalf of our members including:

- With the hard work of the Public Relations and Outreach Committee along with the Board of Directors, we have just published an ISNR brochure for members' use with patients that explains (in laymen's terms) neurofeedback, its benefits, and outcomes based on current scientific research. Purchase yours today from the ISNR store.
- We've been able to advance our mission of promoting excellence in applied neuroscience with the publication on our web site of *The Evidence-Base for Neurofeedback as a Reimbursable Healthcare Service to Treat Attention Deficit/Hyperactivity Disorder* by H. Edmund Pigott, PhD, Lindsay De Biase, PhD, Eugenia Bodenhamer-Davis, PhD and Richard E. Davis, MSd.Additionally, the ISNR Tool Kit for NFB as an Evidence-Based Treatment for ADHD, also available on our web site, is one of our most downloaded; helping practitioners file clients' claims with insurance providers.
- ISNR will launch its new journal: *NeuroRegulation* in the first quarter of 2014. The editor of *NeuroRegulation* encourages the submission of papers from all fields—including cognitive science, neuroscience, and social sciences—that are relevant to neurofeedback research, theory, or applications.
- Finally, we continue to work on expanding our membership outside of North America and are conducting outreach efforts to neurofeedback professionals in Europe, Asia, and South America, as well as Australia and New Zealand.
- Please mark your calendars for next year's conference to be held in San Diego, CA, at the Wyndham San Diego Bayside, October 16 through October 19, 2014, with pre-conference workshops held October 13–15. Wyndham San Diego Bayside has an excellent location directly across the street from the bay hosting the Maritime Museum and USS Midway aircraft carrier/museum, and will be newly rennovated as of May 2014.

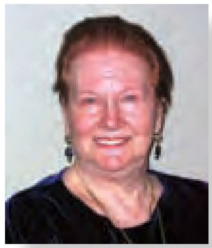
As we begin to wind down 2013, I would like to remind everyone to renew their ISNR membership. Remember ISNR membership is annual (running from January 1 to December 31). Your 2013 membership will expire on December 31, 2013 so please renew before December.

Membership applications are available electronically at www.isnr.org. Please encourage colleagues both within and outside of your organization to join ISNR. Membership in ISNR brings exceptional value for neurofeedback professionals at all career stages, providing a wide range of world-class programs, publications, and services for members around the world. The field of neurofeedback is growing and globalizing rapidly with the pace of biomedical research. ISNR members have access to high-quality venues to share great science, to network with peers, and to develop resources for their career advancement. Don't wait. Renew your ISNR membership today.

I welcome your comments, feedback, and suggestions. Please feel free to contact me anytime at cyablonski@isnr.org.

Cindy A. Yablonski, MBA

Letter from ISNR Editor



Merlyn Hurd, PhD

Dear All,

Welcome to the Winter 2013 edition with the theme of Mélange of Articles. Since the wintertime gives us a mélange of activities to celebrate, we are in keeping with the traditional spirit. The first article is by Michael and Linda Thompson, et al., who give us an incredible journey through the implementation of bio/neurofeedback. I believe many of you will download this article to have at your side to help yourself, those you mentor, and your clients, to understand these approaches even better. Next, the wonderful scientist David Kaiser illuminates for us the use of qEEG and its importance with very tough cases. The newly reorganized QEEG Board follows with a clearer article about the ins and outs of certification. The newest Dipolmates are listed with our congratulations. Dr. Koberda gives us a case history on Alzheimer's and his effective treatment of this disease. Dr. Mascaro provides a discussion of the quick resolution of stuttering via neurofeedback; before and after Qs tell the story effectively. Then, getting into the world of business, and especially the neurofeedback business, Dr. Larson reviews the advantages of continuing education. The edition ends with a very unusual look at the EEG signal in an article titled *Spike Train EEG (STEEG): A New Understanding of the Basic Neurophysiological Mechanisms of the Brain*. It will provoke you to do some thinking, because, just as we used to say we knew everything about the brain by just assessing the activity at CZ, we may be missing information because we have not had the technology to see it.

Aside from all the good information in this edition, I hope you have a wonderful holiday and see you in 2014!!!

Merlyn Hurd, PhD, BCN Senior Fellow

Heart-Brain Connections Underlie Effective Neurofeedback plus Biofeedback Interventions: Complex Case with Depression

Michael Thompson, MD, Lynda Thompson, PhD, Andrea Reid, MA, James Thompson, PhD, BCN, David Hagedorn, PhD

Introduction

In 2006 we took on a client who had been under medical treatment for depression since 1988. A conflict with his business partner in 1996 resulted in the loss of his half-ownership in a lucrative franchise operation he had been dedicated to for over fifteen years, and his depression worsened; indeed, he had not worked for a decade. Over the course of a year, he completed 82 sessions of training, stopped taking antidepressant and anxiolytic medications, and reduced his sleep medication. He discontinued his training when he returned to work, becoming CEO of a mid-size company in another city. Clinical experience with this client and others has shown that for a wide range of conditions, people can decrease symptoms and better manage their response to stress when neurofeedback (NFB) is combined with biofeedback (BFB) treatment. This paper gives an overview of this client's assessment and training and then provides the rationale, based on neuroanatomy and neurophysiology, regarding why the intervention was successful and achieved long-term results.

Case Study

Presenting Problems: JB was 55 years old when his wife brought him for an assessment in spring 2006. He was passive, amotivational, and not keen on learning self-regulation skills, preferring to let his doctors (family practitioner and psychiatrist) manage his treatment. He was taking medication for depression (an SSRI), for anxiety (Ativan), and for sleep (Imovane). For diabetes (diagnosed 5 years earlier) medications (insulin and Metformin) were combined with watching his diet and getting regular exercise. He additionally had started acupuncture treatments twice a week, which improved his mood for the rest of the day but did not have lasting effects. A few times each week he would feel panic and experience tightness in his chest and he kept two Ativan tablets in his pocket to alleviate these panic attacks. The family doctor and psychiatrist had recently ordered a PET scan and a 3D brain scan. There was mildly reduced glucose uptake in the parietal and temporal lobes relative to frontal lobe uptake and a "subtle relative decreased uptake in the left frontal lobe compared to the right." But, the main finding turned out to be a spot on his lung. Two weeks after his assessment at the ADD Centre, a biopsy indicated it was malignant, further increasing his anxiety and depression. He subsequently underwent surgery a month after he started his neurofeedback plus biofeedback treatment. Fortunately, the tumor had not metastasized.

Background Information: History was positive for a traumatic brain injury (TBI) but JB denied any lasting effects from a concussion suffered when a drunk driver hit his car when he was 21. His head went through the driver's side window and hit the side mirror. He was briefly unconscious and spent four days in the hospital under observation and being treated for facial lacerations and other injuries. His next medical crisis was a severe case of malignant hyperthermia after surgery in 1986. (They thought it was appendicitis but it turned out to be diverticulitis.) Thereafter he experienced memory problems for about a year and word finding difficulties that persisted. About two years later, he was diagnosed with depression. After the unfavorable business buyout in 1996, his depression worsened and he was additionally diagnosed with anxiety disorder. Various medications were tried over the years. Prozac, Ciprolex, Zoloft, Lorazepam, Clonazepam, and Amitriptyline all produced side effects. In addition to medications, there was extensive cognitive behavior therapy and periods of day treatment at a psychiatric hospital. The latter helped get him through the periods when medications were being changed, a slow and discouraging process, because the dose would gradually be increased and then there would be slow weaning off when side effects were intolerable. On Amitriptyline he had a reaction of racing heart and they were unsure if he was having a cardiac incident or a panic attack. They stopped that drug but a feeling of tightness in his chest did not go away, so Ativan started to be used more frequently. On Clonazepam he could not get off the sofa and his speech was slowed. After the diagnosis of diabetes in 2001, it became clear that fluctuating blood sugar levels further complicated his low energy and resistant depression. At the time of assessment he was taking Effexor (37.5 mg) because he had tolerated that better than the other antidepressants that were tried.

There had been long-term sleep problems and, after sleep studies identified sleep apnea, he began using a C-Pap mask. Imovane (15 mg) had been taken for 9 years, prescribed at the same time as the C-Pap. After eight years, a second opinion regarding sleep was sought and the physician said JB had to be weaned off the Imovane, though that process had not started. Sleep had been very restless with nightmares for the last ten years. One psychiatrist said he had Post Traumatic Stress Disorder after the loss of his business. He awakened early with a lot of negative thoughts and felt agitated to the point that it was hard to sit and he would sometimes walk for hours.

Assessment Procedures and Results: The initial assessment at the ADD Centre involved history-taking, a com-

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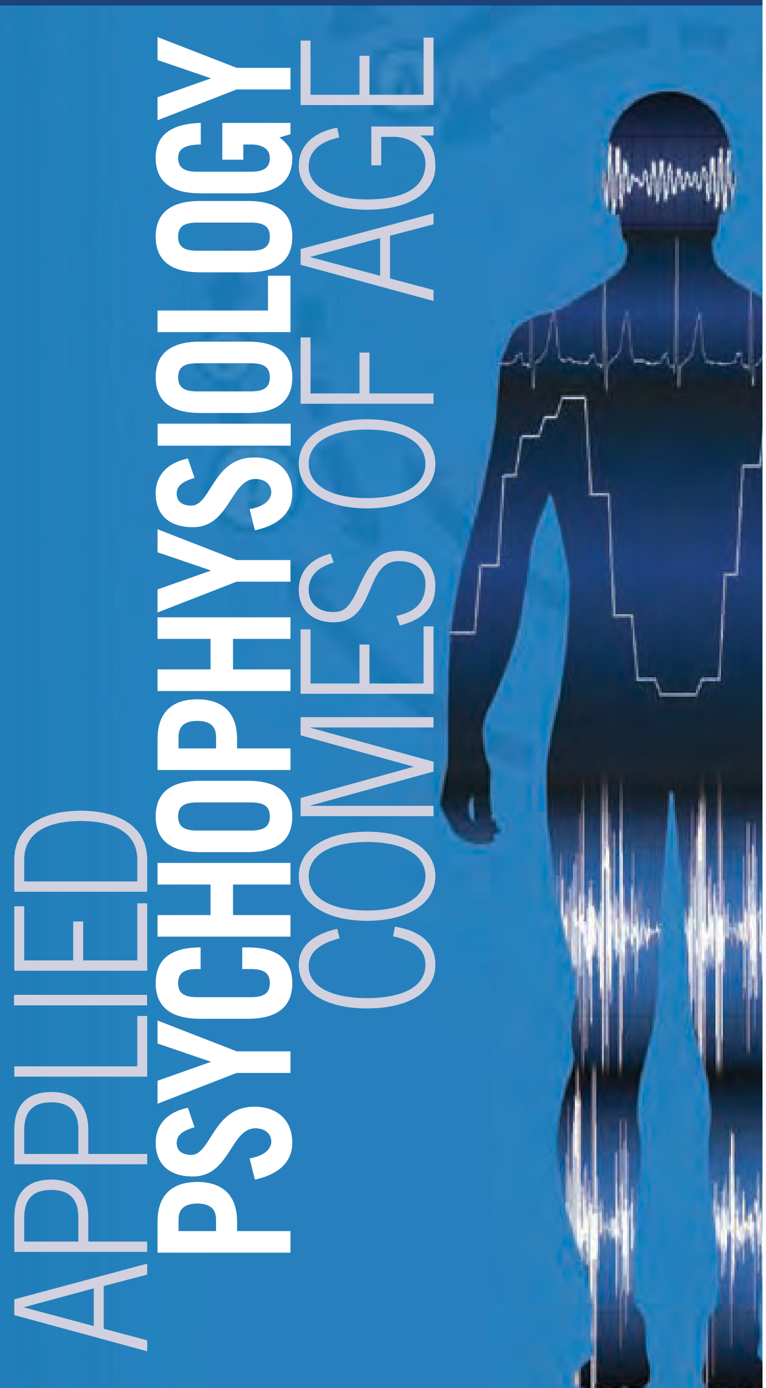

Biofeedback and Neurofeedback Applications in Clinical Practice and Optimum Performance.

puterized continuous performance test (the IVA), a psychophysiological stress assessment, and a single channel (CZ) EEG profile. Three days later, 19-lead EEG data collection and quantitative analysis was done as well. He performed well on the IVA, showing vigilance (missed no targets) without impulsivity. The only scores outside the normal range were for reaction time. He was, however, very slow, below the 1st percentile rank for males age 55, when responding to both visual and auditory targets. (TOVA, which is usually also done as part of the initial assessment, was not administered as no problems related to attention were found on the IVA.) The stress assessment indicated poor recovery after stressors. The Stroop Color Test and a mental math task are the stressors used in the Thompson & Thompson stress assessment script[1]. Given the finding of shallow and fairly rapid respiration, it was clear that practicing diaphragmatic breathing and getting synchrony between his respiration and his heart rate changes through heart rate variability training would be helpful for stress management. The EEG profile indicated some spindling beta in the 23-27 Hz range (a "busy brain" pattern) at the central location (CZ), and the 19-channel analysis using NeuroGuide showed some other differences that would be consistent with depression (more alpha activity in the left frontal area and with 11 Hz activity 2.1 SD above the database norms in LORETA images with source in the uncus, amygdala, and parahippocampal gyrus) and with his problems related to memory and integration of information (excessive alpha seen in the right temporal-parietal area on brain maps).

The conclusion was that neurofeedback plus biofeedback training could play a role in improving JB's mood and overall functioning because areas that were outside database means matched with symptoms. It was explained that the training would be complimentary to other interventions already in place: medications, regular exercise, healthy diet and sufficient sleep. It was expected that sleep quality would improve with training. Learning self-regulation skills and reducing his dependence on medications were the main goals agreed upon. Improved mood would be a bonus. Regaining a feeling of control in his life and engaging in productive work, whether in a volunteer capacity or using his business and accounting skills in paid employment, were long term goals. JB himself did not think the latter was realistic, given his age, lack of energy, and being ten years out of the workforce, but his wife and the second author (LT) agreed it should be in the "Objectives" section of the Initial Assessment Report.

Training Methods and Procedures: Training proceeded with the first couple of sessions showing gains coming slowly because of JB's apathy and depression. As expected, the heart rate variability training showed progress before the brain wave changes were apparent and, by the end of April (one month of training, nine twice-weekly sessions) he was doing well using diaphragmatic breathing to calm himself. He had stopped using Ativan except

1 Available from the Biofeedback Foundation of Europe for use with Thought Technology Biograph-Infiniti equipment

for two occasions in the week preceding his lung surgery. He returned to training in May, two weeks after surgery, and was able to achieve synchrony between his breathing and heart rate changes by the end of his first five-minute session of HRV training. Training that day followed what had been done in his first nine sessions and consisted of baseline recording followed by seven five-minute training segments as follows: two HRV segments, four NFB segments, and a final HRV screen. Between each segment his statistics were reviewed and he would be praised for the scores that moved in the right direction. Breathing at baseline was at 14 breaths per minute (Brpm) and then dropped to an average of 6.23 Brpm during feedback on the first HRV segment and was maintained at 6–7 Brpm for the remaining segments. Mean finger temperature was 33.82 Celsius at baseline and rose to 35.03 degrees during the first segment; it was then maintained at an average 35 degrees (range from 34.66 to 35.08). EEG training parameters, with placement between F3 and F7 and referenced to the right ear, were to reduce 4—8 Hz activity while enhancing 13—15 Hz and inhibiting beta in the 23—25 Hz range. EMG influence was tracked by monitoring 52—58 Hz and that activity was < 2 microvolts throughout (range of 1.28—1.83). JB was in great spirits. He was proud of being off the Ativan and felt he could soon start to reduce the dose of Effexor. His surgeon had proclaimed him a model patient and prognosis was excellent because he felt the entire tumor had been excised and no further treatment would be needed, just monitoring for any recurrence.

This single-channel amplitude training to increase left frontal activation continued untill August and then progress testing was done because he had discontinued his SSRI medication and we wanted to update his qEEG. JB had continued on the same dose of Imovane and it was targeted next for reduction, which had to be done with medical supervision. The physician at the sleep clinic who initially prescribed the medication almost a decade before refused to see him again, explaining that he was a sleep specialist and this was now an addiction problem. The psychiatrist at the Centre for Addictions and Mental Health who subsequently saw him for a consultation would not prescribe any further drug to alleviate withdrawal effects and suggested a very, very slow reduction in dose. A plan was developed to very gradually wean off that highly addictive sleep medication, which he had taken for almost a decade, and it was to be done in conjunction with his pharmacist, who would shave a bit off each pill at the next refill and shave a little more off with subsequent refills.

The next stage in his training at the ADD Centre, starting mid-August, was to continue with the biofeedback (HRV training and monitoring finger temperature) and change the neurofeedback to focus on a series of new parameters that would include coherence training, all of it based on an updated qEEG. A sequence of steps was planned for NFB:

1. Decrease 22 and 23 Hz activity at CZ and then at FZ. (It was >2 standard deviations above the database norms in the eyes open condition.)
2. Decrease 19–22 Hz at FCZ.
3. Do a few sessions of alpha/theta training (eyes closed) at PZ for deep relaxation.
4. Do coherence training, starting with the most deviant pairs. First, increase alpha coherence (8–12 Hz) between T4–CZ, then T4–PZ. Then the following:
    - Decrease 12–15 Hz coherence FP1–FZ, FP1–F4, FP2–FZ, FP2–F3
    - Decrease 18–25 Hz coherence FP1–T3
    - Increase 15–18 Hz coherence T5–F3 and T5–F7

Steps 1–3 were completed in August through October and coherence training began in November. Approximately 4–6 sessions for each pair of sites was done, though this could vary depending upon success. In December, the IVA continuous performance test was re-administered and JB's response time was now above average. The standard scores for speed had increased from just 47 and 50 respectively for auditory and visual targets, to 113 and 118! The faster response time is what one would expect in a bright person. He spoke of how much he appreciated the ADD Centre staff and how much they cared. He noted that he always felt good after a session, though if he went too many days without training (more than a week), his chest would start to feel tight, his motivation would drop and his head "doesn't feel right," so he wanted to keep training. JB had surprised himself by taking the car to get gas for the lawn mower, realizing afterwards that this was the first time he had driven for over a year. He was starting to think about learning more about the daily operations of a company in which he had been a silent investor, because the operating partner had suffered a minor heart attack and was considering working less. JB was still taking Imovane nightly, but slightly less. He also continued on his insulin and Metformin to control his diabetes, prescribed and monitored by his family doctor. He was no longer seeing a psychiatrist. He was exercising regularly.

Outcomes: On April 9, 2007, a year after he started training, further progress testing was done; he had completed 82 sessions of training. This time the TOVA was used as the continuous performance test and he had spectacular results with an ADHD score of +5.54. His standard scores were 110 (75th percentile rank and the ceiling for men his age) for Attention, 123 (94th percentile) for Impulsivity, 145 (>99th percentile) for Response Time, and 127 (96th percentile) for Variability. The WISC-III was administered and he scored at the 97th percentile rank on his Full Scale Score. He had been tested in 2000 on the WISC-III as part of a neuropsychological test battery and had also scored in the Superior range (96th percentile) and the increase was due to improved scores for Processing Speed from the 66th to the 73rd percentile rank. On academic testing, scores on the Wide Range Achievement Test (WRAT3 in 2000 and WRAT4 in 2007) showed arithmetic was still his strong suit (82nd percentile rank and down a bit from the 91st percentile), which is not surprising in an accountant. Reading and spelling had improved from mid-average to the 73rd percentile rank (top of the average range). I noticed a big increase in his sense of humor as we did the lengthy testing session together. Staff had also remarked on this during his recent training sessions. It was just after Easter and his wife told me that their two grown children were home for that holiday and remarked that they were delighted with the way their Dad took charge of things. They said that their old Dad was back. With respect to medication, the Imovane was down by 1/3 and they were still going slowly with respect to reducing it. Best of all was the news that he was going to take over management of the company in which he had been a silent partner because the managing partner was retiring. This meant a move to a new city and discontinuation of training.

Rationale for Why the Training Worked

Overview: This client's history was positive for both TBI (concussion with loss of consciousness at age 21) and long-standing depression. Though a more severe case than others, with possible hypoxia in conjunction with his episode of malignant hyperthermia after surgery and with diabetes diagnosed five years before he started training, he was like the majority of clients seen post TBI, in that his symptoms related to both affect networks (depression, anxiety, irritability), and cognitive functioning. There was also a change in the ability to handle stress and less resilience. There were abnormal EEG findings that included ones related to depression (less left frontal activation than right) and ones that related to cognition, such as coherence abnormalities between frontal and temporal-parietal areas. Areas of the brain involved in these different systems and networks are neuroanatomically linked and the manner in which they are linked means that they respond to a combination of NFB plus HRV training. Since different networks are involved, there are improvements in multiple areas across thinking, feeling, and behavior, as discussed below.

Brain affects heart, and vice versa: As outlined in detail elsewhere (Thompson & Hagedorn, 2012; Thompson & Thompson, in press), TBI can affect not only brain function but also cardiac function, as reflected in reduced heart rate variability (HRV). It is also true that HRV training can change the EEG; in particular, improved HRV is found in conjunction with increased sensorimotor rhythm (Reid, Nihon, Thompson, & Thompson, 2013). These heart-brain connections have a basis in functional neuroanatomy. Fortunately, the relevant neuroanatomical structures can be influenced by NFB that changes activation patterns in the affect network (the emotional brain) using either single channel or, perhaps more directly, LORETA NFB. The rest of this paper will outline the main neuroanatomical connections and structures affected by HRV training, which influences the same affect network(s) as does NFB. This naturally also involves the areas of the brain that are involved in the normal and the chronic stress response. The conclusion is that a combination of NFB training done over central midline structures (CMSs), when combined with HRV training, can be expected to influence response to, and recovery from, stress and also reduce symptoms of depression while enhancing cognitive functioning.

Heart changes after TBI: Thompson & Hagedorn (2012) show data that demonstrate clearly that even healthy athletes can show major cardiac changes post concussion. Instead of heart rate variations that are even, with high amplitude and steady rhythmicity, the picture becomes one of low amplitude and poor rhythmicity of heart rate variations. In a study concerning HRV after a traumatic brain injury (TBI), the TBI group revealed significant differences in HRV parameters compared to controls (Baguley et al., 2006).

Depression also affects cardiac functioning in a negative way (Grippo et al., 2009). Depressed patients show changes in autonomic regulation of the heart; namely, activation of sympathetic drive, decrease in vagal tone with a resulting increase in heart rate (HR), decrease in heart rate variability (HRV), and reduced baroreceptor reflex function. Reduced HRV is found in depressed patients both with and without cardiovascular disease and there is a correlation between reduction in HRV and severity of depression. The converse is that exercise can significantly improve HRV, baroreflex sensitivity, blood pressure, and vascular function (Zoeller, 2007). Exercise also relieves depression (Beniamini et al., 1997). Therefore, not only good diet and sleep, but also exercise, should be discussed with clients doing biofeedback training.

The right and left insulae—key areas that influence HRV: A stroke affecting the right insula has dramatic effects on autonomic balance and heart functioning (Nagai et al., 2010; Shah, 2009). There will be reduced HRV, tachycardia, and premature ventricular contractions. Nocturnal blood pressure (BP) will increase, as will norepinephrine levels. Left insular cortex damage will increase baroreceptor sensitivity (BRS), one of the influences on HRV. The insulae are important regulators of autonomic balance with the right insula affecting the sympathetic and the left the parasympathetic system, in part through connections to the nucleus accumbens, which balances the sympathetic and parasympathetic systems (Nagai et al., 2010). There are reciprocal connections between the insula and the anterior cingulate gyrus, amygdala, entorhinal cortex, orbitofrontal cortex, and the hippocampus. There are also reciprocal connections with subcortical autonomic structures, including the lateral hypothalamic area, nucleus tractus solitarius, and the parabrachial nucleus. These areas are also reciprocally connected to each other.

Of significance for biofeedback practitioners, the above observations link healthy brain function with healthy heart function and suggest that heart rate variability training has effects on many of the same basic neural structures that are also directly influenced by EEG biofeedback training. These observations imply that one can achieve a synergistic effect by combining the two modalities when treating a range of clients, from those with head injuries or problems like depression through to athletes wanting to optimize functioning.

Neuroanatomical structures affected by NFB that relate to affect network(s): A network consists of interconnected, functionally related, groups of neurons. In the cortex, these networks typically involve adjoining Brodmann Areas. (For an explication of Brodmann Areas, their primary functions, and their relation to International 10-20 electrode placement sites, a booklet can be ordered from the ISNR Research Foundation) Each Brodmann Area (BA), in addition to its primary function, can be involved in many different functional networks. A network serves to synchronize the functions of groups of neurons in widely distributed but functionally related areas of the cerebral cortex. Networks also involve cortical–subcortical connections (Thompson & Thompson, 2007b). This perhaps explains why NFB practitioners have obtained good results when doing NFB over a single site, such as Cz, that influences not just that site and the area with a radius of 6 cm around that site from which the EEG being measured is generated but, rather, whole networks. The cingulate gyrus that lies below the central midline (FZ, CZ, PZ) is involved in many networks, including the executive, affect, salience, and default networks. Influencing the cingulate gyrus when the patient's affect network is actively engaged may be assumed to have *indirect* effects on many deeper structures that cannot be directly affected by NFB at the surface site.

Areas deeper in the cortex can, alternatively, perhaps be directly trained using LORETA NFB. This is a variation on regular NFB which uses a 19-channel cap and a process by which the mathematics of low resolution electromagnetic tomography (Pasqual-Marqui et al., 1994; 2002) are applied so that a number of parameters are simultaneously trained. LORETA is used to identify the source deeper in the cortex of activity measured on the scalp and, with LORETA NFB it is hypothesized that one can influence these areas more directly.

Cortex-Striatum-Thalamus-Cortex Connections: When you work to influence one network, say the affect network, other networks need to be inhibited. This inhibition of non-relevant areas and activation of the relevant network is achieved through connections from the cortex to the basal ganglia, then to the thalamus and back to the cortex (Thompson & Thompson, 2011). When a cortical area, such as the anterior cingulate gyrus in our example of the affect network, is activated it will stimulate a specific area of the striatum (putamen or caudate). That area will then inhibit all other areas of the striatum so that they do not fire and thus do not inhibit other functional areas of the globus pallidus (GP). The globus pallidus is like a functional map of the cortex and it fires rapidly, inhibiting the thalamus. However, the one area of the GP that is inhibited by the putamen will then not inhibit the related, functionally specific area of the thalamus. This is easier to understand if you think of it as a double negative: when you inhibit inhibition, you activate. Thus you are opening one particular thalamic gateway so that a particular rhythm is then pulsed to all functionally related areas of the cortex (Kropotov, 2009). This may intensify a selected program of action (or inhibition) that involves many, often widely separated, areas of the cortex. These functionally related areas constitute a neural network.

The key structure for us to consider in the central midline structures is the anterior cingulate cortex (ACC) (Devinsky et al., 1995). As noted in the following discussion of anxiety and the stress response, the **ACC** has direct links to other key cortical areas that are involved in affect networks, including the medial and orbital prefrontal cortex and the insulae, in addition to key basal ganglia areas such as the amygdala, as well as to the hippocampus. Changes in the ACC will therefore also affect the hypothalamus and, through it, the autonomic nervous system and the hypothalamic-pituitary-adrenal (HPA) axis. These connections have a major role in the human stress response. All of these structures are involved in depression and influencing them using a combination of NFB + HRV, especially when combined with appropriate therapy, can alleviate depression and improve stress management (Thompson & Thompson, 2007; Paquette et al., 2009). They are also involved to different degrees in all the major neural network systems, affect, executive, salience, and default networks.

Central midline structures involved in depressive disorders: Patients who are depressed, like our case study, JB, demonstrate symptoms that relate to both executive and affect networks. Figure 1 illustrates three networks involved in different aspects of depression based on a presentation by neurosurgeon Dirk de Ridder (2010 ISNR presentation). Areas in the **green** box relate primarily to the Executive network involved in depression. More specifically, this dorsal compartment modulates attention and sensory-cognitive symptoms, such as apathy, attentional, and executive deficits. It includes the dorsolateral prefrontal cortex (BA 9 & 46), the dorsolateral anterior cingulate (area 24b), posterior cingulate (BA 31), the inferior parietal

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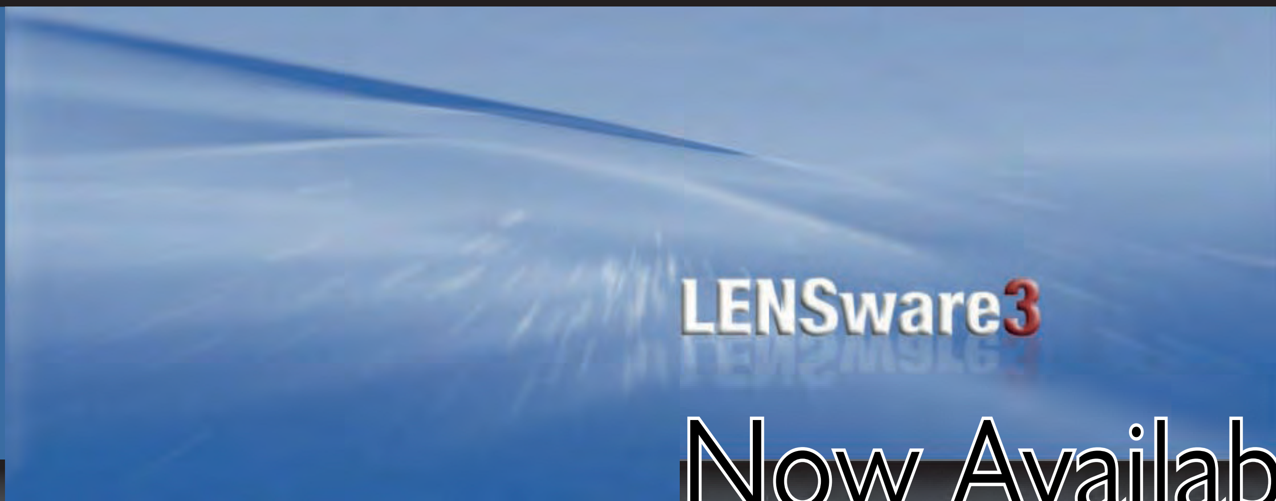

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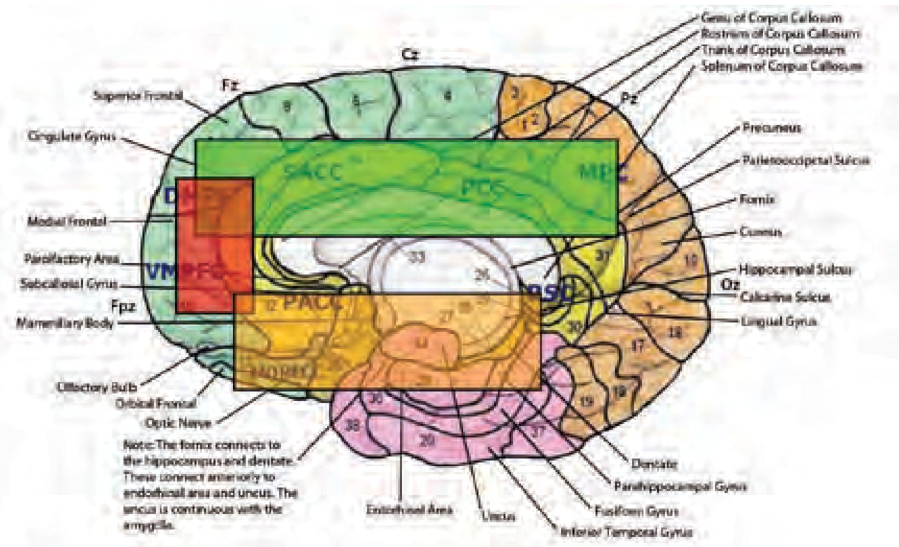

Drawing from The Neurofeedback Book (AAPB, Wheat Ridge, CO) & ADD Centre Brodmann Areas Booklet

Figure 1: *Areas involved in depression*

lobe (BA 40), and the striatum. The dorsal prefrontal components, including the AC, are also involved in the cognitive control of emotion including reappraisal, evaluation, and explicit reasoning concerning emotional stimuli. Areas in the **orange** box relate primarily to the affect network involved in vegetative symptoms. These vegetative/autonomic nervous system symptoms involve a ventral compartment that relates to things like sleep disturbance, loss of appetite and libido. Here we find the hypothalamic-pituitary-adrenal axis, hippocampus, insula (BA 13), subgenual cingulate (BA 25), and the brain stem. In **red,** is an area involved in the integration and connection of the other two areas. Information concerning cognition and emotion from the two compartments (dorsal & ventral) is integrated by the rostral anterior cingulate (BA 24), medial frontal cortex (BA 9 & 10), orbital frontal cortex (BA 11), and frontopolar areas. Also recall that selection of activation of particular pathways can involve the cortex–basal ganglia–thalamus–back to cortex connections, as previously described.

Different treatments influence different affect networks. Cognitive behavior therapy appears to have its effects on the more dorsal and executive aspects of depression by increasing activity in the ACC, BA 24. It may also decrease activity in the "connecting" areas including the medial prefrontal Cortex (MPFC), BA10, and the orbital frontal cortex (OFC), BA11 (Goldapple, 2004).

Medications can increase the activity of the more ventral, vegetative areas, such as the prefrontal cortex (PFC), BA 9, and the brain stem. They decrease activity in BA 25 and the hypothalamus according to one research group studying the regional metabolic effects of fluoxetine in major depression and the relation to clinical response (Mayberg et al., 2000**).** Deep brain stimulation can directly affect BA 25. It requires a surgical procedure to implant leads in the ventral anterior cingulate gyrus connected to a pacemaker placed under the skin near the collarbone. Helen Mayberg and collaborators successfully treated a number of depressed people, individuals virtually catatonic with depression despite years of talk therapy, drugs, even electroconvulsive shock therapy, using deep brain stimulation in Brodmann Area 25 (Mayberg et al., 2005, Kennedy et al., 2011)**.** BA25 is said to be metabolically overactive in treatment-resistant depression and stimulation; once it has been properly adjusted (which can takes months), it has been shown via a PET scan follow-up to decrease activity in BA 25 and OFC BA 11. It will also increase activity in the dorsolateral prefrontal cortex (DLPC) BA 9, 46 and the ACC BA 24 and parietal cingulate cortex (PCC) BA 31. In addition, it will increase parietal activity in BA 40. These effects are seen in responders, about half of the patients.

Interestingly, precisely the same areas that respond to deep brain stimulation showed changes after combined NFB and psychotherapy in a group of patients who had been medication non-responders (Paquette, 2009). One month after the end of treatment, responders showed significantly reduced absolute power of high-beta (18–30 Hz) in the orbitofrontal cortex (BA 11/47), insula (BA 13), amygdala/parahippocampal cortex (BA 36/37), temporal pole (BA 38), lateral prefrontal cortex (BA 10 and BA 6/8), and subgenual cingulate cortex (BA 25). In addition, responders showed an increase of high-beta activity in the bilateral precuneus/posterior cingulate cortex (BA 40/31), which makes sense because other research has shown abnormally low amplitude of high-beta activity (>20 Hz) in these cortical areas (BA 40/31) in individuals with major depressive disorder (Pizzagalli et al., 2002; 2004). Increased activity in precuneus/posterior cingulate cortex has additionally been shown to correlate with symptom remission following pharmacological treatment (Mayberg, 2003) and with interpersonal therapy (Martin et al., 2001). Since the highest level of cortical glucose metabolism during resting state occurs in these brain regions in healthy participants (Raichle et al., 2001), it is a plausible hypothesis that pharmacological treatment, interpersonal therapy, and the neurofeedback plus counseling utilized by Paquette all contribute to restoring the appropriate functioning of the default mode of the brain.

NFB+ HRV training for depression with anxiety: We have previously de-

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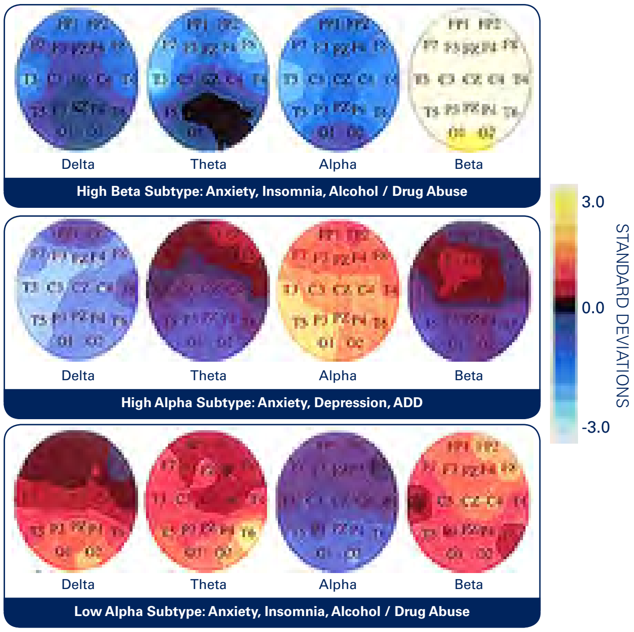

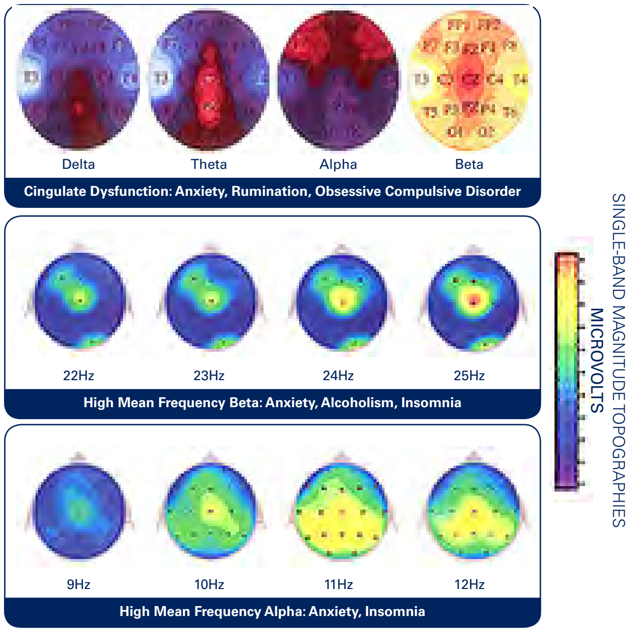

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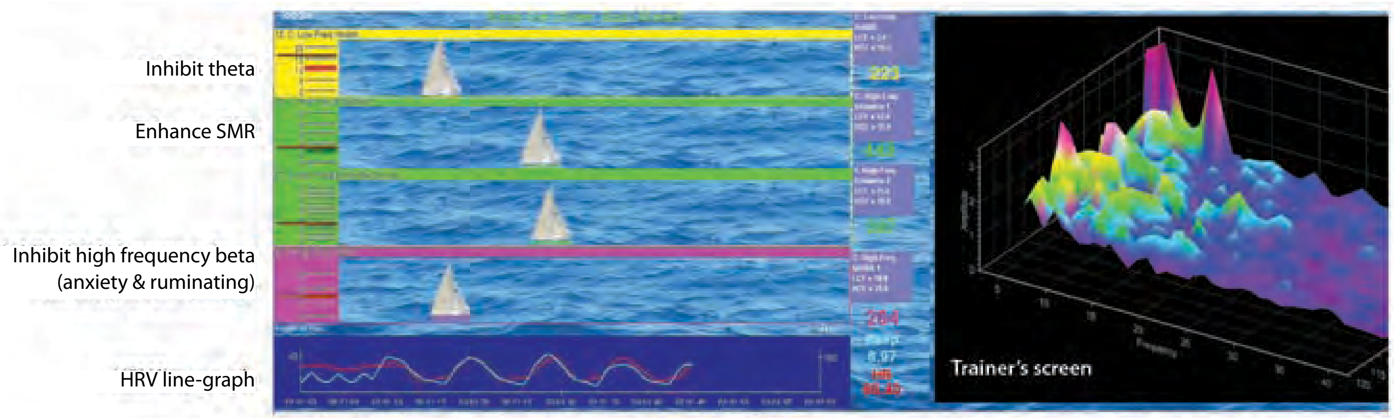
scribed the importance of BA 25 in the ventral rostral anterior cingulate cortex in very serious, often intractable, depression. Brodmann Area 25 is also called the subgenual area (literally "below the knee" referring to its location below the bend in the corpus callosum), area subgenualis or subgenual cingulate. NFB may influence affect networks that involve the more superior aspect of the cingulate gyrus under Cz and the medial aspect of the frontal lobe under Fz but BA 25 is very deep in the midline cortex. Thus LORETA NFB may, theoretically, have a much more direct effect than single channel training at one site. BA 25 is rich in serotonin transporters and is considered to influence the following areas: hypothalamus and brain stem, which are involved in changes in appetite and sleep; the amygdala and insula, which affect mood and anxiety; the hippocampus, which plays an important role in memory formation; and some parts of the frontal cortex responsible for self-esteem. Of importance to the practitioner who uses a combination of NFB + BFB, the solitary nucleus in the medulla (nucleus solitarius) has direct connections to BA 25. Since HRV training strengthens the vagal response and will send vagal afferent feedback to the solitary nucleus in the medulla, it can also influence BA 25 and thus, perhaps, the symptoms of depression related to dysfunction in this area of the cortex.

Integrating this Knowledge into Training

At the ADD Centre/Biofeedback Institute of Toronto, both single and two-channel EEG biofeedback plus HRV and 19-channel LORETA NFB plus HRV are carried out. Other biofeedback modalities, such as electromyogram (EMG), finger temperature, or electrodermal response (EDR) are added based on the stress assessment results for a particular client. Interventions are customized according to assessment findings. Conventional single-channel neurofeedback at FCz combined with HRV training has been extremely effective in the majority of our clients who have problems with mood. The screen below is a favorite because it induces a sense of competition between the boats with the client attempting to have a green boat win. To do this they must focus, eliminate all unnecessary thoughts and ruminations, relax, and concentrate. In addition, they are asked to maintain synchrony between the variations in heart rate (red line) and their breathing (inspiration and expiration shown by the blue line).

In depressed patients (and often in our patients with TBI or with autistic spectrum disorders, too) LORETA analysis often indicates that significant deviations from the normative database have their source in the anterior cingulate (BA24), the medial frontal area, the rostral-ventral cingulate gyrus (BA 25), uncus, parahippocampal gyrus and other central midline structures. These areas are deep in the midline and surface NFB can only influence them indirectly based on influencing part of the network that includes them. LORETA NFB, on the other hand, allows us to choose up to 24 areas and measurements (amplitude, phase, coherence) in the cortex for feedback. It is more time consuming because LORETA mathematics requires input from 19 channels to determine source localization and, to be accurate, the data should be artifact free. The hope is that faster results can be obtained and that will justify the more complex procedure. Controlled research has yet to be done to investigate the merits of LORETA as compared to regular neurofeedback. In the meantime, both can be used and both can be combined with HRV training. When doing LORETA NFB, HRV training can be done at the beginning of the session when the cap is being put on and impedance checked, which takes upwards of ten minutes.

Heart rate variability refers to the constantly oscillating variations in heart rate that are observed in healthy individuals. These variations may be measured in terms of their frequencies, amplitudes, the range of heart rate changes in each cycle,



Band widths are adjusted on the screen to fit the individual client.

Trainer adjusts thresholds so it's a real race!

Figure 2: *Screen for simultaneous EEG and HRV feedback*

and the standard deviations of the interbeat intervals (IBI). The standard deviation of these interbeat intervals (IBI), computed after all artifacts have been removed, is called SDNN. We are concerned when SDNN falls below 50 ms and prefer to see it above 80 to 100 ms (Gevirtz, 2010). Another statistic is the ratio of low frequency (LF)/high frequency (HF), which we like to see between 1.5 and about 4. Heart rate changes are being measured using a plethysmograph which measures red light reflected from blood vessels in a finger or thumb. Measuring pulse to pulse in the thumb gives the same figures at rest (though may not do so during tasks) for SDNN as do EKG sensors on the chest (Giardino, 2002). SDNN is calculated using the Cardiopro program from Thought Technology, which was developed to meet research criteria and provides the statistics used internationally for heart rate variability measures.

The training screen shown in Figure 3 is from the Thompson suite of screens developed for the Biograph-Infiniti. Changes in respiratory are shown in blue and changes in heart rate are in red. Note the synchrony between the two, which is what one wants to achieve. The heart rate in this athletic young woman is oscillating 6 times in 60 seconds so the frequency is therefore 6/60 or 0.1 oscillations/second. Her heart rate changes from about 60 to 108 beats per minute and more than 60% of the power is in the low frequency (LF) range. This is excellent variability and having the highest power in the LF range indicates a good balance between sympathetic and parasympathetic systems. This example shows an unusually good pattern with total power above 8000 ms^2 /Hz (good is >3000) and SDNN >120 ms.

The healthy heart is constantly changing its rate. Every person's heart has its own unique resonance frequency and, for adults, it is often close to 6 variations per minute. The range is between 5 and 7. It will be less for men than women and less for tall persons than those who are shorter because it is partly dependent on total blood volume. It will thus be higher for children than adults. Breathing is the simplest way to pace the heart in order to achieve higher variability and you can find a person's resonance frequency by checking which breathing rate achieves almost perfect synchrony between breathing and heart rate changes and the highest amplitude of variations in heart rate. Good synchrony between breathing and heart rate changes is seen in both Figure 2 (below the sailboats) and Figure 3. With inspiration there is an increase in the heart rate and with expiration a decrease in heart rate (Lehrer, 2007). The goal in HRV training is to increase the heart rate variations (HRV). Breathing diaphragmatically at a person's resonance frequency will result in the highest amplitude of HRV**.**

Vagal Control of HRV

The vagus, that wandering nerve, has many connections with the heart. The right vagus nerve primarily innervates the sino-atrial (SA) node whereas the left vagus primarily innervates the atrial-ventricular (AV) node. The aortic arch contains baroreceptors and, when the blood pressure rises, the resulting expansion of these baroreceptors sends afferent signals via the vagus nerve X to the nucleus of the solitary tract in the medulla. This nucleus sends signals to many areas in the brain with the final result for heart rate being that the dorsal nucleus of the vagus and the nucleus ambiguus send parasympathetic vagal efferents to the heart that slow the heart rate, resulting in a decrease in blood pressure. This feedback additionally causes inhibition of the sympathetic innervation of arterioles resulting in vasodilation due to a decrease in the vasoconstriction that is caused by sympathetic stimulation. This also decreases blood pressure.

The nucleus of the solitary tract (nucleus tractus solitarius or NTS) in the medulla of the brain stem is a key player when it comes to the beneficial effects of HRV training. The NTS has efferent connections to nuclei in the midbrain/diencephalon and the forebrain. These links include connections to the basal ganglia and, in particular, to the amygdala, the hypothalamus (and thus the Hypothalamic-Pituitary-Adrenal axis), diencephalon (with thalamic connections to the insula), cingulate gyrus, and the orbitofrontal and medial prefrontal cortical areas. Of particular interest regarding the above-mentioned observation of increase in the amplitude of SMR during HRV training, the NTS con-

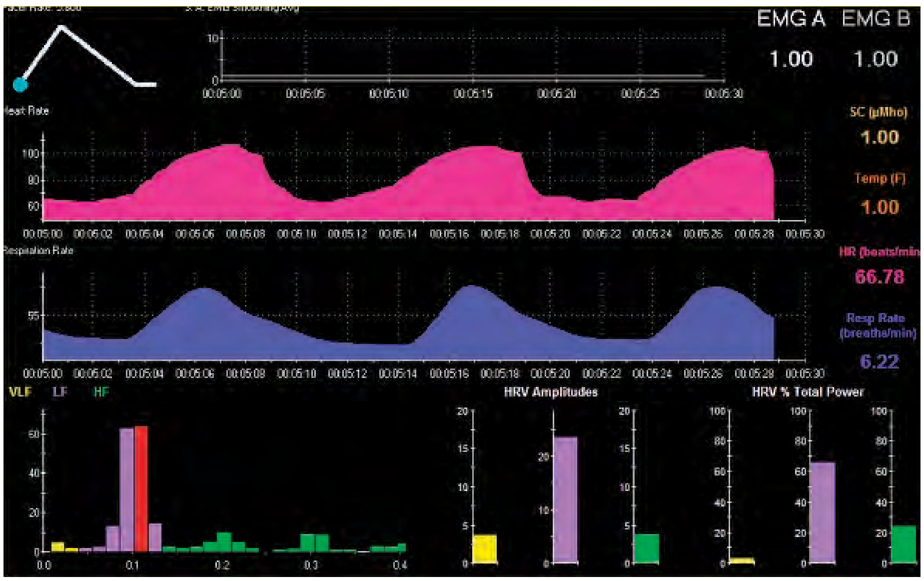


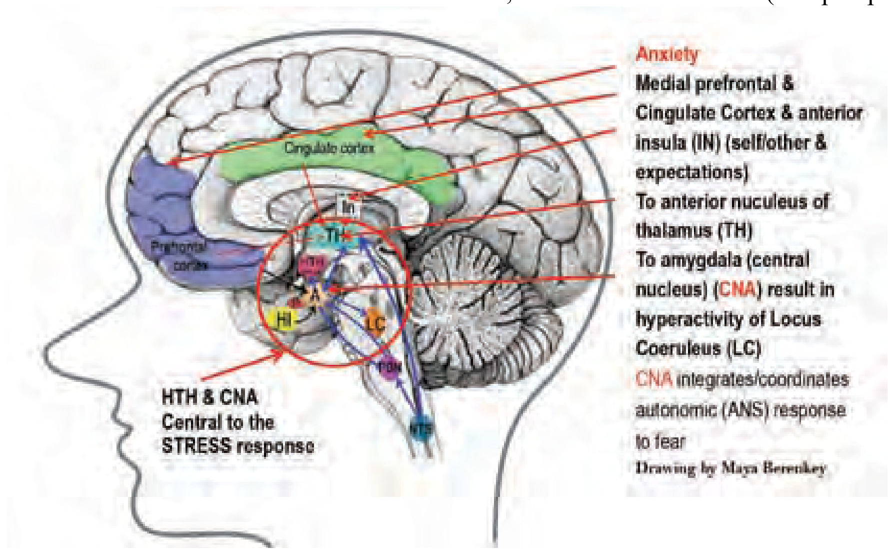
Figure 3: *Heart rate variability training screen.*

nects, via the central tegmental tract, to the ventral posterior medial nucleus of the thalamus (VPMN) which produces the 40 Hz rhythm. This 40 Hz increase, as well as an increase in SMR, produced from the ventral posterior lateral nucleus of the thalamus(VPLN) is observed in the EEG recorded from an alert, immobile, focused cat (Steriade, 2005). Since the VPLN is related to SMR production, and SMR is produced on the exhale during HRV training, we assume there are some connections between these thalamic nuclei. When HRV training is done, it is this oscillatory system of reciprocal increases and decreases in blood pressure and heart rate that is being influenced. Training an oscillatory system appears to have lasting effects on the pacemakers that set the rhythms; when training EEG it is the thalamus that sets the pace for various rhythms and when training HRV it is this loop involving the vagal connections between the heart and the nucleus solitarius in the brain stem. Heart rate variability training is the only biofeedback modality that influences the vagal response and makes it possible to recruit parasympathetic calming influences.

Improving a client's ability to handle stress

Recruiting the calming influence of the parasympathetic nervous system is obviously helpful when helping to train a client to improve their ability to recover from stress. As seen in Figure 4, the connections within the affect network that are involved in fear and anxiety are those that are central in a person's response to a stressor. There are connections from the medial prefrontal cortex (under FCz) to the anterior cingulate gyrus and the insulae. These cortical areas connect to the amygdala and to other basal ganglia, which then control anterior thalamic outputs. The anterior thalamus connects to the lateral nucleus of the amygdala (LNA) and then to the central nucleus of the amygdala (CNA), which integrates outputs and coordinates autonomic and behavioral responses to fear and to stress (Thatcher, 2012). The CNA connects to the brain stem. Important areas for our work include the nucleus ambiguous, and the dorsal motor nucleus of the vagus that affects parasympathetic slowing of heart rate, and the locus coeruleus (norepinephrine production) and other nuclei that activate the sympathetic system and which thus can cause an increase in heart rate. Most important, however, are the CNA connections to the hypothalamus. The CNA and the hypothalamus both have connections to the parabrachial nucleus that controls breathing rate, depth, and regularity. The CNA connections to the hypothalamus (HTH) are to the lateral nucleus (LH), which exerts control over the sympathetic branch of the autonomic nervous system, and to the paraventricular nucleus (**PVN**) to control the parasympathetic system. The **PVN** efferents to the dorsal motor nucleus of the vagus and to sympathetic relay brainstem nuclei will influence heart rate and HRV. The PVN also has direct connections both to and from the nucleus of the solitary tract (NTS). The PVN of the Hypothalamus is the only brain site in a closed efferent-afferent reflex loop that connects with both the sympathetic and parasympathetic nervous systems (Dougherty, 2013).

The PVN of the hypothalamus can also activate the hypothalamic-pituitary-adrenal axis (HPA). This HPA activation will result in an increase in adrenocorticoids (cortisol). Thus the CNA (amygdala) can stimulate the hypothalamus to produce corticotrophin-releasing hormone (CRH–also called releasing factor, CRF) which will cause the pituitary to produce adrenocorticotropic hormone (ACTH). This will result in the adrenal production of glucocorticoids (GC). With chronic anxiety and stress, hippocampal regulation of CRH production fails, CRH and NE levels are held high and the CRH stimulation from the central nucleus of the amygdala will override the modulating effects of GC feedback to down-regulate the production of CRH from the amygdala and the hypothalamus and the production of neorepinephrine from the locus coeruleus. The result is a continued



TH = Thalamus	CNA = Central Nucleus Amygdala	HI = Hippocampus
IN = Insula	A = Amygdala	PBN = Parabrachial Nucleus
HTH = Hypothalamus	ANS = Autonomic Nervous System	NTS = Nucleus Tractus Solitarius
P = Pituitary	PVN = Paraventricular Nuc.	

Figure 4: *Central Pathways for Response to Fear, Anxiety, & Stress*

Diagram by Maya Berenkey from The Companion to the Neurofeedback Book (in press)

Jonathan E. Walker, M.D.

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

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activation of CRH production (Smith-Pelletier, 2002). The symptoms of overactivity in the HPA axis are familiar to anyone in the mental health field. There is a narrowing of the person's perspective, producing a focus on their own preoccupations with ruminative thinking and poor cognitive performance. Sleep is impaired, which further compounds their difficulties (Thompson, 2003).

In Figure 5 we have a summary of the connections discussed above. Note that the amygdala innervates the parabrachial nucleus, which controls respiratory rate. The amygdala also innervates the locus coeruleus, which produces norepinephrine, and thus relates to an increase in blood pressure and to fear-related behaviors.

Since the cortical affect network is connected to important structures in the midbrain and brainstem, heart rate variability training may have such a profound effect on this affect network and, in particular, enhance the control of anxiety.

In summary, the locus coeruleus production of norepinephrine (NE) can stimulate, and the hippocampus can inhibit, the production of corticotrophin releasing factor (CRF) from the hypothalamic paraventricular nucleus. NE and CRH are both involved in the affective responses to stress including fear, constricted affect, and even stereotypical thinking. In the normal stress response there are acute improvements in arousal level, memory, focus, and in the immune system. The normal stress response is meant to be short-lived. Problems occur when stress is chronic Training clients to use HRV can help them to recover more rapidly from stress and is highly effective, whether one is dealing with a patient or an athlete who wants to optimize performance.

NFB Training done over central midline structures (CMSs) at FCz (or training done with specific targets within the CMSs using LORETA NFB) will influence the key structures within the cortex that, in turn, influence the affect networks. These structures connect with midbrain and hindbrain nuclei, as discussed above, that are key structures involved in control of heart and respiration rates. We call NFB "top-down" training. The same structures can be affected by BFB, in particular, by HRV training, and we call this "bottom-up" training. In both cases there are direct connections to parts of the cingulate cortex, the insulae, the amygdalae and the paraventricular nucleus of the hypothalamus, with further connections to midbrain and brainstem nuclei, particularly the nucleus solitarius. Network properties in the brain greatly facilitate the possibility of influencing one part of the system to get change throughout the system.

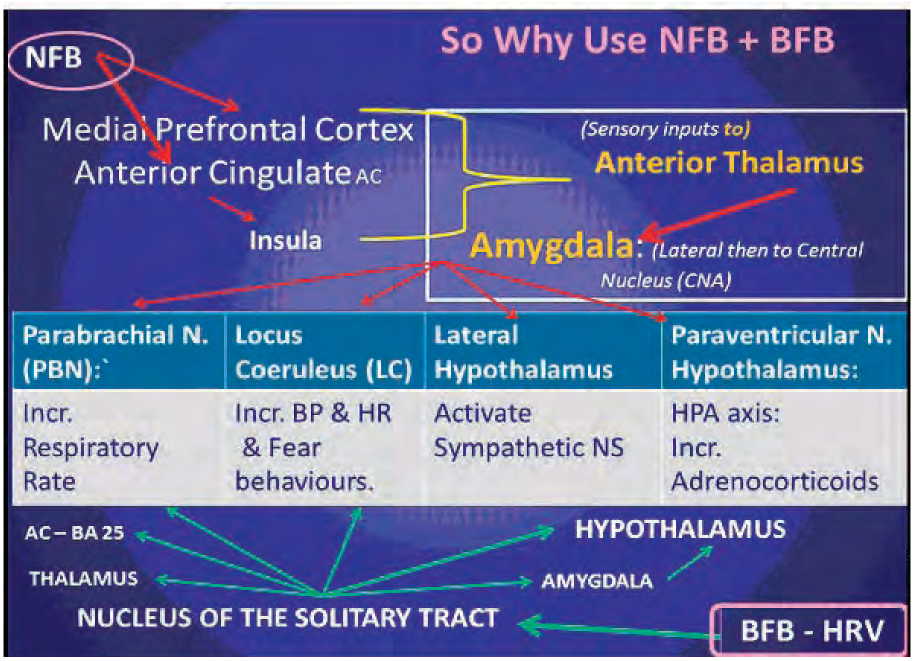

Figure 5

Clinical Tips for Effective NFB plus HRV Training:

- When doing single channel NFB, have clients do HRV training simultaneously with NFB by placing a respiration and heart rate line graph onto brainwave feedback screens. (If the client is too young or too fidgety to have the pulse monitor on their finger, just focus on their tummy breathing as a method for relaxing and releasing muscle tension.)
- If you are doing LORETA Z-score NFB, have the patient do HRV training while you are putting on the cap.
- Try having HRV feedback available using a second computer while the LORETA NFB is proceeding. Then the trainer can direct the client's attention to his/her HRV if the synchrony between breathing and heart rate changes starts to slip.
- Have your patient practice their relaxed diaphragmatic breathing with broad external attention (no ruminations) in the waiting room before training and when doing activities of daily living such as upon awakening, when falling asleep, while watching television or driving. They can have 2-minute breathing breaks (about 12 effortless diaphragmatic breaths) while sitting at their desk in the office or at school, too.

Discussion and Conclusion

Though neurofeedback has established efficacy for only two conditions, Attention-Deficit/Hyperactivity Disorder and Epilepsy, it can clearly be helpful in a

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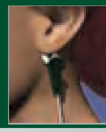

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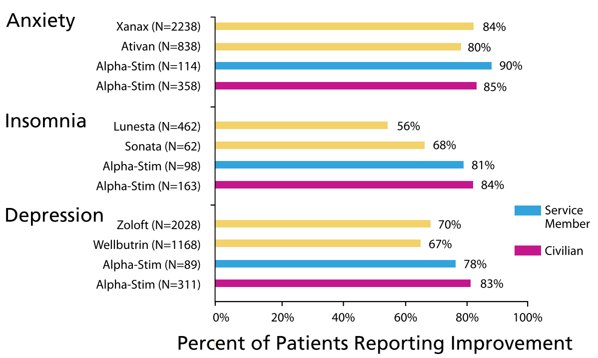

Patients who reported a positive response according to WebMD Drug Surveys, and Alpha-Stim Service Member and civilian surveys. Alpha-Stim Data from 2011 Military Service Member Survey (N=152) and Alpha-Stim Patient Survey (N=1,745). Conducted by Larry Price, PhD, Associate Dean of Research and Professor of Psychometrics and Statistics, Texas State University. Pharmaceutical Survey Data from www.WebMD.com/drugs. Accessed on October 28, 2011.

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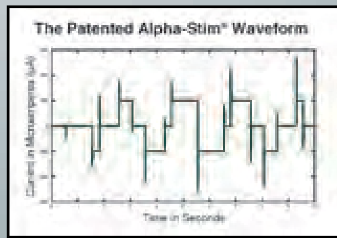

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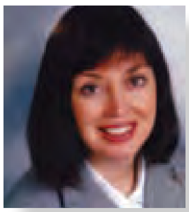
wide range of other disorders, including anxiety and depression, particularly if it is combined with heart rate variability training. There is, in addition to resolution of many symptoms and reduction in the use of medications, enhanced stress management, which builds resiliency and guards against future problems. Emotional dysregulation correlates with changes in multiple central midline structures in the cortex, as well as nuclei in the midbrain, brain stem, and the hypothalamic-pituitary-adrenal axis. All of these areas can be influenced by both neurofeedback, through efferent connections (top-down) plus biofeedback, through afferent connections (bottom-up), due to network connections. Both approaches connect to basic structures, in particular the nucleus solitarius in the medulla and the paraventicular nucleus in the hypothalamus, just from different directions.

Success with clients like JB is encouraging. A one-year telephone follow-up was conducted on April 8, 2008 and the report was that he had been working all year and planned to stay at it. At times he had felt he could use more stress management training but he would use his breathing and get through whatever crisis had arisen. He had further improved his diet, going gluten free and adding a magnesium supplement, as recommended by a naturopath, which further improved his energy level. He was not yet totally weaned off Imovane ("The time just never seems right." said his wife) but his dose was down to half. He had not returned to using an antidepressant or Ativan, nor did he report feeling either depressed or anxious. This story about a bright chartered accountant who returned to productive, full-time work in his fifties, after a decade of being sidelined by depression and anxiety, is more than a heart-warming anecdote. Combined with the comments on functional neuroanatomy that provide a rationale for the neurofeedback plus biofeedback intervention, it provides pilot data that can be cited when planning a randomized, controlled trial that could help establish efficacy for combined neurofeedback plus biofeedback in the treatment of these conditions.

Authors

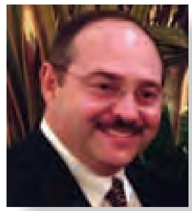
Michael Thompson, MD, *devotes his time to the administration of the Biofeedback Institute and to teaching. Formerly, when practicing medicine, he was Associate Professor and head of post-graduate education in Psychiatry, University of Western Ontario, examiner for the Royal College of Physicians (Canada), and chairman of their examinations committee in psychiatry. Numerous professional publications include A Resident's Guide to Psychiatric Education. While Associate Professor, University of Toronto, he was psychiatric consultant to the Hospital for Sick Children's neurology department.*



Lynda Thompson, PhD, *is a psychologist who has taught, practiced clinical and school psychology, and owned learning centers. She became executive director of The ADD Centre in Toronto in 1993 after discovering the world of neurofeedback and deciding to specialize in that intervention. Her doctoral dissertation (1979) dealt with hyperactive children treated with methylphenidate. She is co-author with William Sears of The A.D.D. Book: New Understandings, New Approaches to Parenting Your Child.*

Lynda and Michael co-authored The Neurofeedback Book: An Introduction to Basic Concepts in Applied Psychophysiology, AAPB, Wheat Ridge, Colorado, 2003.

Andrea Reid-Chung, MA, *CPsych Assoc (Supervised Practice), BCN, is a certified Canadian counselor and is the Clinical Coordinator of the Centre. She has a Science degree from U of T specializing in psychology and cognitive science and a Master's Degree in mental health counseling, from Medaille College, New York.*



David Hagedorn, PhD, *is an Assistant Professor, Department of Military and Emergency Medicine and Assistant Professor of Family Medicine, Military Emergency Medicine at the Uniformed Services University of the Health Sciences, Bethesda, Maryland. In addition, he is the CEO at Evoke Neuroscience, Jacksonville, North Carolina.*

James Thompson, PhD, BCN, *obtained his PhD from the Faculty of Kinesiology (Psychophysiology) at Pennsylvania State University. His doctoral dissertation involved developing a new metric that included EEG data for assessing concussion in athletes. James has done numerous invited professional presentations entitled, The Role of Electroencephalography in the Assessment and Rehabilitation of Concussions, and Applications of Biofeedback and Neurofeedback in Sports Psychology. In addition, he holds the highest certifications for coaching downhill skiing and sailing. He works in New York City and is Chief Science Officer & founder with Dr. Hagedorn of Evoke Neuroscience.*

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

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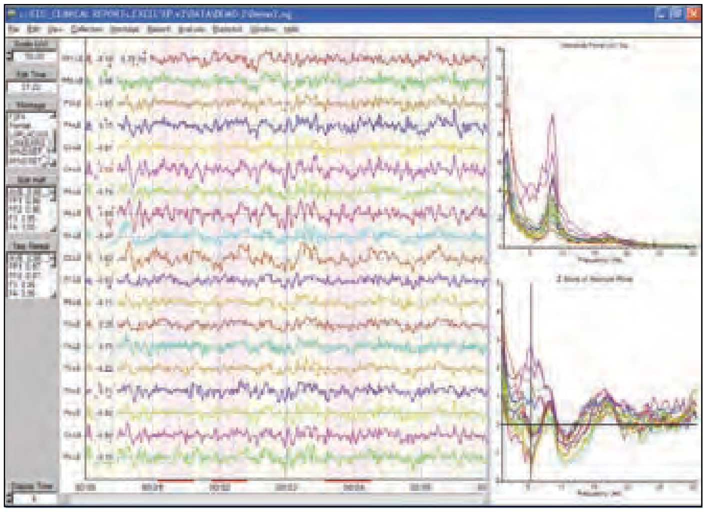

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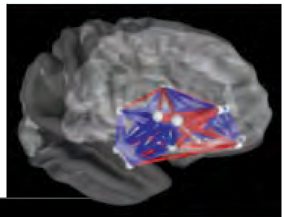

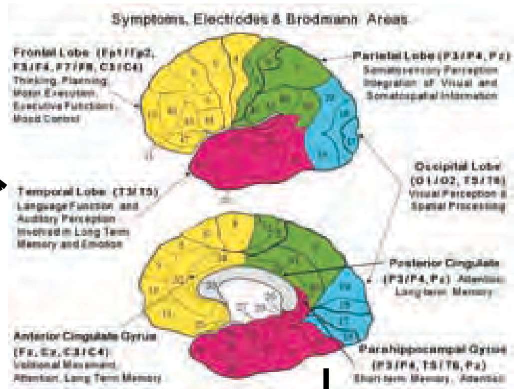

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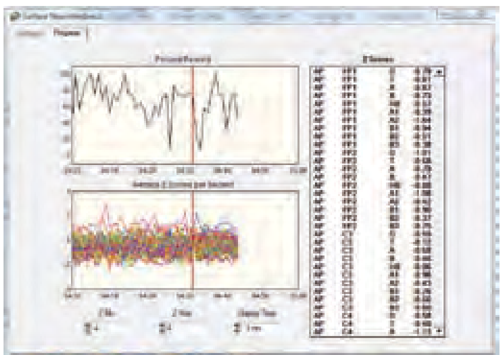

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The Value of a qEEG Assessment, Especially Looking at Theta Unity

David Kaiser, PhD and Orli Peter, PhD

After six unsuccessful treatments with five doctors, a 44-year-old male patient sat in the office desperate for a solution to his insomnia. Two and a half years before, he had moved into a new home and his sleep difficulties began. He found it difficult to fall to sleep, and at times even to feel tired, and when he finally would fall sleep he would wake up an hour later only to repeat the same cycle through the night. At most, his broken sleep would aggregate to a mere four hours.

He first sought treatment at a sleep recovery center where he received neurofeedback that rewarded the stability of his EEG. His sleep difficulties showed slight improvement, but his progress stagnated early on and the sleep center suggested he obtain a qEEG to better understand why his sleep was resistant to improvement. The qEEG results indicated his delta activity was insufficient, and his neurotherapist suggested they increase this frequency. The results were disastrous; the patient reported having suicidal ideation for the first time in his life and he was unable to sleep for four days. Becoming increasingly worried, the patient sought out another neurotherapist who used milder Z-score training. Although the patient reported his sleep returned to four hours per night, it began to feel like he was "conscious during sleep" and he could hear his "brain running." He then sought pharmaceutical help and was prescribed Ambien, Celexa, and Gabapentin. Unfortunately, the medications had a barely noticeable impact on his sleep, and the side effects were not worth continuing their use.

It was after this first bout of unsuccessful treatments that the patient came to our office for help. We took the data from his original EEG recording and analyzed it using SKIL software to evaluate the limbic and cortical contribution for 55 brain areas (Brodmann areas).

In the past, our analysis has been useful for evaluating the integrity of each brain area and the cortical networks involved: default-mode, dorsal attention, executive control, salience, and sensory networks. We have developed a handful of promising neuromarkers strongly associated with symptoms, behaviors, or life histories. For instance, an impairment in socioemotional awareness, a deficit found in 11 of 12 death row inmates, is associated with a disconnection of Brodmann area 44R (Kaiser et al., 2012). Childhood abuse commonly results in left or right auditory cortices being disconnected (Kaiser & Meckley, 2012). But in our case, we found a disconnected posterior cingulate, which we observed in all of our trauma cases to date. More than 30 studies have identified posterior cingulate problems in PTSD patients, and our data is consistent with this research. As a result, we feel comfortable calling a theta disunity of the posterior cingulate a neuromarker for trauma.

Our analysis of the patient's EEG revealed this trauma marker—his posterior cingulate cortex (PCC) exhibited what we call a corticolimbic dysrhythmia. Theta activity in the PCC was out of synch with theta activity in the adjacent cortex, suggesting another origin for its activity in the PCC. Given that the PCC anchors the default network (see text box, following), this deviation in shared activity indicates diminished functional integrity of the default mode network. This conclusion explains why the many sleep treatments he tried were unsuccessful—they were not treating the source of his sleep disruption, a dysfunction in his default mode network which made it difficult for him to transition from an awake to sleep state. Activity in the default-mode network must increase for sleep onset to occur (Picchioni et al., 2008).

In his intake, the patient denied he had experienced any trauma in his life. However, when we presented the findings to him, he revealed he had indeed suffered through three significant traumas in his life: emotional abuse by his mother throughout his childhood, the sudden death of his older brother in a car accident when the patient was 17, and the suicide of his fiancé when he was 21. Nevertheless, he insisted that he was "over these traumas" and left our office refusing to believe they had any relevance to his sleep problems.

Five months and two more unsuccessful medical treatments later, the patient called our office and was ready to consider that our analysis was relevant. He agreed to try an integrated trauma treatment that consisted of alpha-theta training combined with psychological trauma techniques, such as somatic experiencing, and a specialized mindfulness training that targets the dissociation often seen in trauma.

After 20 sessions of alpha-theta training at site Pz, directly over the PCC, the patient's sleep normalized. He became drowsy at night, was able to fall asleep within 20 minutes, and he was able to sleep for 6–7 hours without interruption. The patient was happy with his improvement and we agreed to terminate treatment. It is now three months after termination, and the patient still reports that his sleep difficulties did not recur. Without the EEG assessment, we did not know the patient's problems very well, nor did the previous practitioners who treated the symptom (sleep disruption) and not the mechanism (trauma).

ONLINE SUPPLEMENT

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

QEEG Certification Board Update

The QEEG Certification Board

Congratulations are in order for Dale Foster and Nancy Wigton, who have moved from QEEG Technologist to Diplomate and to Mark Jones who, having completed all other requirements, is a new QEEG Diplomate after passing the exam at ISNR, a wonderful accomplishment that will soon be shared with others who are making their way through the process. There are currently over 25 active candidates seeking certification.

The QEEG Certification Board (QEEGCB) has been in existence since 1995, with recent application for non-profit status. The Board was established to promote excellence in providing EEG acquisition and qEEG assessment by establishing a practitioner certification process.

When one looks at our field and the array of different types of licenses that are held by our members, one sees that we are primarily a mixture of medical and psychological entities. We are a field of psychophysiologists with extensive crossover into one anothers' fields. Often those in the medical field are conducting traditionally psychological assessments and treatments and psychologists are conducting medical activities. No wonder the questions of, "What is a qEEG report?" "What is neurofeedback?" and "Who can provide a qEEG report or neurofeedback therapy?" are so often asked.

QEEG certificants mostly hold doctoral level education (41 of the current 55). The remaining hold master's level (7), MD (5), MS (2), QEEGD (1) and Reverend (1) status. Technologist and Diplomate status is further broken down in Table 1. While most are in the United States (53), we have one each in The Netherlands, Chile, Singapore, and Canada. Of the US certificants, the leading states are California (10), Texas (7), and then Florida and Arizona, (5 each),w North Carolina (4), Pennsylvania, New York, Washington, and Minnesota (2 each), and Alaska, Arkansas, Colorado, Hawaii, Kansas, Missouri, Nebraska, New Jersey, Ohio, South Carolina, Tennessee, and Utah each have 1.

Educational Background	Master's	MD	Doctoral	Other	Total
Technologist	0	2	14	1	**17**
Diplomate	7	3	27	1	**40**
Total	**7**	**5**	**41**	**2**	**55**

Table 1 *Educational Background of Certificants Holding Technologist and Diplomate Status*

The Certification Process

The original certification procedure required only a letter of recommendation from a Diplomate and the passing of the exam. Over the last two years, the exam has undergone extensive revision with input from top clinicians and researchers in the field who sit on our Board and advisory committee. The restructuring has been via a thorough analysis of the history of test takers' responses to the specific questions as well as comments and recommendations from our advisory committee members, who are Robert Thatcher, Jay Gunkelman, Tom Collura, Martijn Arns, Joel Lubar, and Barry Sterman.

The terms licensure, certification, and accreditation are often misunderstood. State licensure, regulated through governmental agencies, is required to perform specific treatments with validated competency. Licensure ensures that the practitioner is qualified to practice psychology, medicine, massage, acupuncture, etc. Certification is voluntary and is a formal process that is designed to endorse practitioners who have met a certain standard, usually involving testing, mentoring and coursework. Private entities and universities can provide training for specific certification. Thus, various manufacturers and software companies can certify people who have achieved proficiency in the use of their product or style of practice. Or, it can be offered on a broader scope by such organizations as BCIA and QEEGCB.

Accreditation is a process by which an institution or other entity is deemed qualified and given recognition to provide a learning environment for the individual seeking certification or licensure. In our field, the BCIA and QEEGCB approve didactic course instruction for the entity that hosts the training, once the entity provides learning materials that meet the standards of the certifying board.

Now**, i**n order to demonstrate competence in the collection and interpretation of qEEG data, we are additionally requiring mentorship from a current Diplomate on five qEEG records. The criteria for these five evaluations differ according to what level an individual is applying for, Technologist or Diplomate. We are also requiring attendance at a didactic course, which provides learning in the content areas in which we require expertise, and must follow the blueprint criteria.

An extensive reading list of required and recommended articles, chapters, and full books can be found at the QEEGCB website[1] as well. The exam questions, by and large, are pulled from the required reading list, which will undergo revision

1 *www.qeegcertificationboard.org*

and updating as more high-quality research articles are published and the test evolves. (While we strive to be able to provide the PDF articles directly to the applicant, this may not always be possible. We hope applicants will understand that in some cases copyright issues present an obstacle to this goal.)

Finally, as criteria for maintenance of QEEG Board Certification status, the attainment of 40 CEUs over a four-year period has been implemented. There are various ways these criteria can be met. You can visit *www.qeegcertificationboard.org* for more information and specifics regarding these changes and more details on the application process. Briefly, attending or providing any approved workshop or scientific talk and writing scientific articles related to qEEG are currently accepted for continuing education. We hope to add reading and publishing activities over time.

> *Accreditation is a process by which an institution or other entity is deemed qualified and given recognition to provide a learning environment for the individual seeking certification or licensure.*

This Board was developed, and remains, separate from BCIA, ABRET, and ECNS, and, as a certifying body, cannot be controlled by any membership organization. However, we have asked for guidance from these other organizations and strive for their endorsement.

We hope that these changes will positively reflect on our field and our professional status in the health services community by providing guidelines that result in qualified qEEG practitioners. Our long-term goals are to 1) continue to certify qualified individuals as competent to provide qEEG services; 2) provide an organization that third party reimbursers can rely upon for professional qualifications; 3) provide an organization and web presence that the general public can rely upon to assist in locating professional and competent certificants.

The QEEGCB Board of Directors: Jonathon Walker, President; Nancy White, Vice-President; Robert Crago, Treasurer; Cynthia Kerson, Secretary; Kirtley Thornton, Member at large; Dan Chartier, Member at large; Merlyn Hurd, Member at Large. Contact us at qeegcb@gmail.com.

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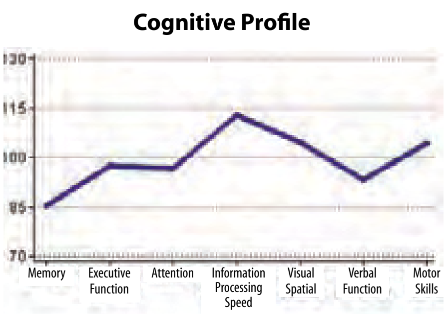
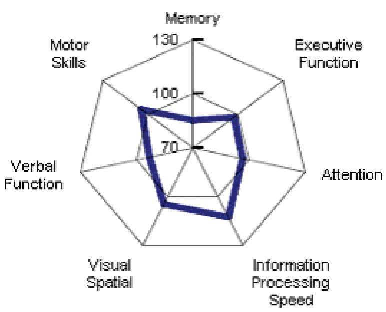
Alzheimer's Dementia as a Potential Target of Z-Score LORETA19-Electrode Neurofeedback

J. Lucas Koberda, MD, PhD, Andrew Moses, Paula Koberda, Andrew A. Bienkiewicz, Laura Koberda.

Alzheimer's disease (AD) is the most common form of dementia. It is a neurodegenerative disease characterized by gradual loss of mental ability, severe enough to interfere with normal activities of daily living. AD typically occurs in elderly age, and is marked by a decline in cognitive abilities such as remembering, reasoning, and planning. A person with AD exhibits a gradual decline in mental functions, often beginning with slight memory loss, followed by losses in the ability to maintain employment, to plan and execute familiar tasks of daily living, and to reason and exercise judgment. AD usually becomes symptomatic after age 60. However, the histopathological abnormalities involving amyloid formation are likely to start many years before the disease manifestation. After age 80, close to 50% of individuals display clinical symptoms of this disease. In Florida, it is estimated that the number of individuals with AD is close to 500,000. There is currently no effective treatment of AD. Medications available on the market have only mild symptomatic benefits, frequently with significant side effects. A typical MRI of the brain of AD patients shows brain atrophy that is most pronounced in the temporal and frontal areas. A recently FDA-approved PET scan (amyloid imaging) generally shows increase in amyloid deposits in individuals affected by this disease. QEEG frequently reveals increase in delta and theta frontal and temporal power (Koberda, 2013). Neurofeedback has previously been shown to be helpful in selected cases in memory and cognitive enhancement (Koberda 2012, Becerra 2012, Rodrigues 2012). However, our experience with patients suffering from AD is still very limited. Therefore, I would like to share my experience describing testing and results of NFB therapy in an AD patient from my practice.





Global Cognitive Score:	99.2
Memory:	85.4
Executive Function:	97.4
Attention:	96.7
Information Processing Speed:	112.8
Visual Spatial:	104.6
Verbal Function:	93.2
Motor Skills:	104.3

Figure 1. *Computerized neurocognitive testing of 68-year-old female. Abnormal is short-term memory score of 85.4 (expected 100) Cognitive Profile*

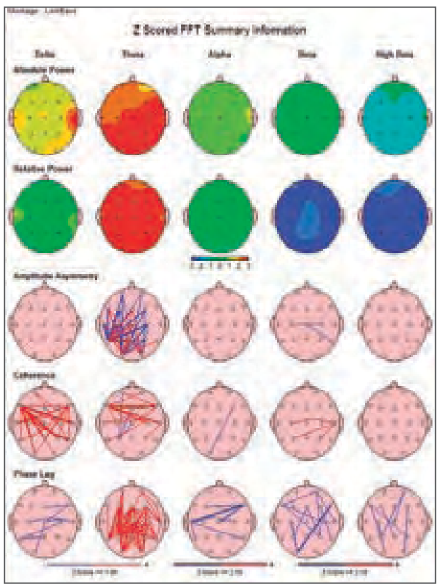


Figure 2A *shows the summary page of qEEG of 68-year-old patient with memory problems. There is evidence of increased bilateral temporal delta and global theta power (NeuroGuide, Applied Neuroscience, Inc.). Green, yellow, and red colors indicate areas within 0-1, 1-2, and 2-3 Standard Deviations (SD), respectively.*

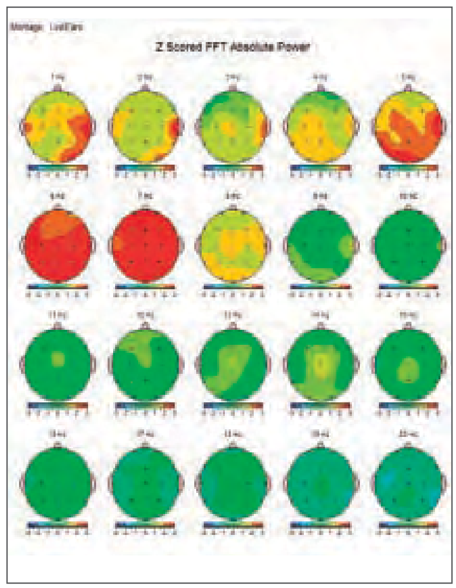


Figure 2B. *QEEG maps of the 68-year-old patient with memory problems. Map shows frequencies between 1-20 Hz. Most extensive increase in theta power was recorded in 5-7 Hz bins. Cognitive Profile*

Case report

A 68-year-old female (retired accountant) complained of 1-2 years' history of progressive forgetfulness. The patient came to my office for a second opinion with her husband. She was previously seen by another neurologist/neuropsychologist, and was diagnosed with early stage dementia (likely mild AD). Treatment with Aricept was recommended; however, the patient was reluctant to initiation of pharmacotherapy. Initial computerized neurocognitive evaluation with Neurotrax (Bellaire, TX) showed mild impairment of short-term memory-score 85.4 (expected 100), with no major involvement of other cognitive functions. (Fig. 1).

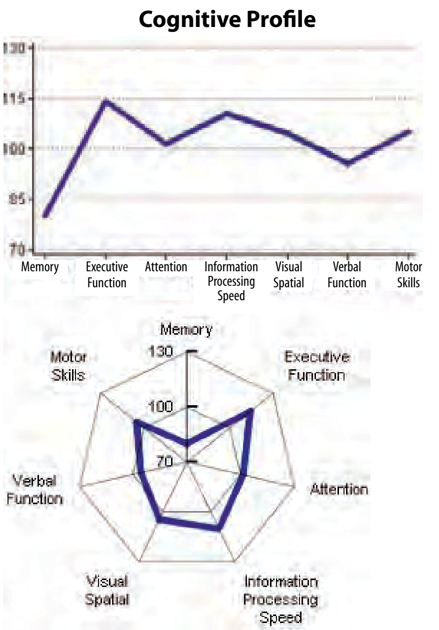
QEEG evaluation showed increase in bilateral temporal delta and global theta power (Fig 2. A, B).

Her blood work was unremarkable, including B12 level. The patient was interested in starting alternative therapy, initialized with the standard 1-electrode NFB (Cygnet). Despite several months duration of NFB (with frequency of 2-3 times a week), further decline in memory function was noted on follow-up computerized neurocognitive testing, with memory score of 80.1 (Fig. 3). At that time, since her symptoms were progressive, the patient decided to start 19-electrode Z-score Low Resolution Electro-Magnetic Tomography Analysis (LORETA) NFB (Neuro Guide, Applied Neuroscience, Inc.), which became available in our clinic.

After 10 sessions of Z-score surface and LORETA NFB (alternate sessions with once a week frequency), the patient reported subjective improvement of her cognitive functions. Repeated Neurotrax testing confirmed improved short-term memory score of 93.2 (Fig. 4).

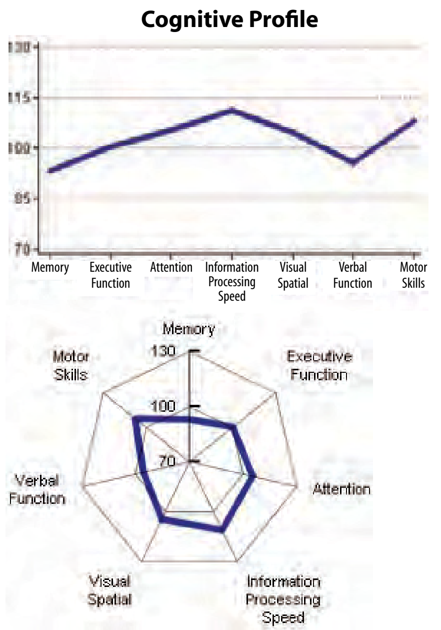
The patient decided to discontinue Z-score 19-electrode NFB, and subsequently her subjective and objective memory declined. Repeated Neurotrax testing completed three and a half months later showed evidence of short-term memory decline, with a score of 76.3 (Fig. 5). In addition, her visuospatial orientation score also declined from 104.6 to 67, indicating progression of neurodegenerative disorder and involving additional cognitive function.

This case illustrates a potentially ben-



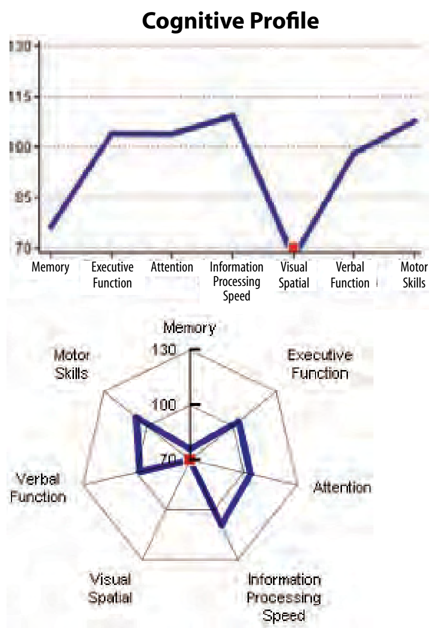
Global Cognitive Score:	101.6
Memory:	80.1
Executive Function:	114.1
Attention:	101.4
Information Processing Speed:	110 3
Visual Spatial:	104.6
Verbal Function:	95.6
Motor Skills:	105

Figure 3. *Neurotrax testing recorded after completion of 1-electrode standard NFB therapy. Notice further decline of memory-score (80.1).*

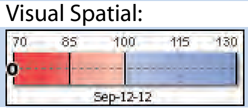


Global Cognitive Score:	102.6
Memory:	93.2
Executive Function:	100.4
Attention:	105.3
Information Processing Speed:	111.1
Visual Spatial:	104.6
Verbal Function:	95.6
Motor Skills:	107.9

Figure 4. *Follow-up Neurotrax testing after completion of 10 sessions of Z-score surface/ LORETA NFB showed improved memory score of 93.2.*



Global Cognitive Score:	95.1
Memory:	76.3
Executive Function:	103.9
Attention:	103.8
Information Processing Speed:	109.2
Visual Spatial:	67



Verbal Function:	98
Motor Skills:	107.5

Figure 5. *Follow-up neurocognitive testing three and a half months after discontinuation of Z-score 19-electrode NFB. See abnormal memory score, 76.3 and visual spatial score of 67.*

eficial use of 19-electrode Z-score neurofeedback in patients suffering from Alzheimer's disease. NFB treatment of this neurodegenerative disorder resulted in temporarily improved cognitive function. Unfortunately, since AD is a progressive disorder, further decline of cognitive functions subsequently has occurred. This was not unexpected, especially after discontinuation of NFB therapy. Further studies of Z-score surface/LORETA 19-electrode NFB with a larger number of individuals suffering from AD would provide insights into potential benefits of NFB in this group of patients. One possibility could be implementation of quantitative EEG as a complementary method in early diagnosis and staging of AD. Given results from this study, it is also likely that neurofeedback could provide therapeutic means for improvement of cognitive abilities in AD patients, especially in the early stages of this disease.

Dr. J. Lucas Koberda is a board certified neurologist and an internationally trained physician. Prior to his neurological training, he received his PhD based on his research in the area of tumor immunology. Dr. Koberda is currently 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 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.

ONLINE SUPPLEMENT

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

Recertification: The Class of 2014

Judy Crawford

The BCIA Board spent a long time reviewing and discussing the policies that impact certification and recertification. For the class of 2014, there are some significant changes to recertification policies that we believe will be good news for you.

As you know, recertification for biofeedback and neurofeedback, required every four years, is accomplished by documenting blueprint-relevant, accredited, continuing education (CE), or successfully completing our certification exam; agreeing to follow the Professional Standards and Ethical Principles (PSEP) of BCIA; and paying your recertification fee.

The board researched state CE requirements for healthcare professionals as a way to establish a benchmark. Starting with the recertification classes of 2014, we've reduced our CE requirements from 55 hours to 48 hours (which includes 3 hours of ethics) for certificants who have recertified up to the Senior Fellow level, when the requirement is reduced to 30 hours. This change means that you will have to pay for fewer CE courses, an immediate savings!

Certificants may satisfy our 3-hour ethics requirement by taking courses provided by their state or national organizations or even through regionally accredited university courses, either face-to-face or online. BCIA-sponsored ethics webinars and workshops provide inexpensive coverage of professional issues. The goal of this requirement is to aid in your understanding and sensitivity to ethical issues that can protect your clients as well as your own practice.

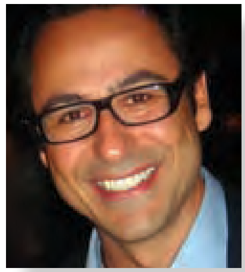
Online courses and webinars have become far more accessible, affordable, and available. We agree that they can be a great way to learn. BCIA offers webinars and records each one just in case you miss the original presentation. The price is the same, $25 for the live or recorded webinar. We charge $15 for the online CE exam and certificate.

We are also pleased to let you know about Massive Open Online Courses (MOOCs). MOOCs are online courses offered for free by many renowned professors from a wide variety of universities. Free? What's the catch? We haven't found the downside yet, but as with everything, buyers beware. We ask you to do some internet research and locate courses you believe will suit your educational goals. Email us with the link to that course for review to ensure that we believe the content will be blueprint-relevant and we'll provide a quick answer as to the suitability of your selection. The day the board voted to allow the use of MOOCs as a way to earn both CE for recertification and to fulfill the human A&P requirement for certification, several of the board members were so intrigued with the concept that they signed up for a course during their coffee break! To date, we have heard positive feedback, but we'll keep you posted as we learn more.

We also want to encourage you to seek professional guidance as your clinical skills grow. You may now use up to 20 hours of formal mentoring as partial fulfillment of your CE requirement. Log the time you spend with your mentor, just as you did for your original certification. Please use the form found online in the Recertification area in the Certificants section of our website.

In 15 years, we have only raised recertification fees once, from $195 to $225. We continue to offer a $225 rate for certificants who complete their CE requirements and file their application before the June 30th deadline. This timetable helps our staff provide the high level of customer service you expect and allows us to freeze recertification fees. The cost of a second recertification has been reduced to $175.

You have worked hard to attain this credential and we hope that these changes will be of value to you as you consider maintaining your certification.



Healing Stuttering with Z-Score Neurofeedback

Leonardo Mascaro

In this paper I will present a clinical case of extreme interest to a wide range of the population, since it involves, at the same time, neurological functional difficulties, as well as psychiatric impairment disorder.

A young undergrad student was referred to me with simultaneous complaints of stuttering, attention, and concentration deficits. He had difficulties socializing due to the stuttering and was also was being treated for depression with psychiatric medication. When he came to me, he had already faced a good deal of psychiatric treatment for depression and had taken a plethora of medications (see table 1).

During that time, he had episodes of loss of control and explosive outbursts, mainly when he felt contradicted.

There was never a neuropsychological evaluation done. I always do an intake interview, get the patient´s history, medications, if any, and confer with the doctor in charge.

In designing my intervention, I simply relied upon neurophysiological knowledge to make sense of his symptoms. His most deviant areas were both at the frontal lobes, as the literature (Ozekgi, A. et al, 2004, The role of Hemispheral Asymmetry and Regional Activity of Quantitative EEG in Children with Stuttering, Child Psychiatry and Human Development 34:269-280) indicates, being the left frontal where we´ll find Broca´s area, even though the higher deviations are on the right. The literature references match what the maps show, especially in the Delta band, but contrary to what the literature noted, in this client we have a lack and not an excess of Delta activity, which is contradictory.

In my opinion, sometimes it´s not a matter of just too much of something (in this case, Delta) but actually, much more, simply a case of dysregulation of a given brain area or structure, be it an excess or a lack of a frequency, the clinical manifestation depending then on both what area is involved in the given dysregulation, and in which frequency(ies), not to mention connectivity measures that, in this case, were an issue, too (again, in the left hemisphere).

Below is a snapshot of his raw EEG. I used both a linked ears and a bipolar montage to conduct all this patient´s electroencephalographic analysis. And, since there´s such a marked disconnection of T3, at the left temporal lobe, in what regards all the remaining of the left hemisphere, if one looks at this channel´s raw, one can readily and clearly see that the neurological activity there does not relate to EMG.

The Initial Evaluation Map (see Figure 2) confirmed his complaints, with reference to a normative database composed of samples of electrocortical activity obtained from more than 700 subjects that were considered normal, after having their neurological, psychiatric, and neuropsychological activities screened. The database in case is known as the Life

Trial Date	Medication
August 2011	Paroxetina 20mg (2 cp night) + Rivotril (max 20 gts/day)
October 2011	Paroxetina 20mg (2 cp night) + Risperidona 1mg (1cp night) + Rivotril
April 2012	Paroxetina 20mg (3 cp night) + Risperidona 2mg (night) + Rivotril
June 2012	Quetiapina 100mg (1 cp morning) + Quetiapina 200mg (1 cp night)
July 2012	Procimax 20mg (1 cp) + Depakote 500 mg (1 cp morning+ 1 cp afternoon)

Table 1: *Symptoms persisted despite multiple medication trials.*

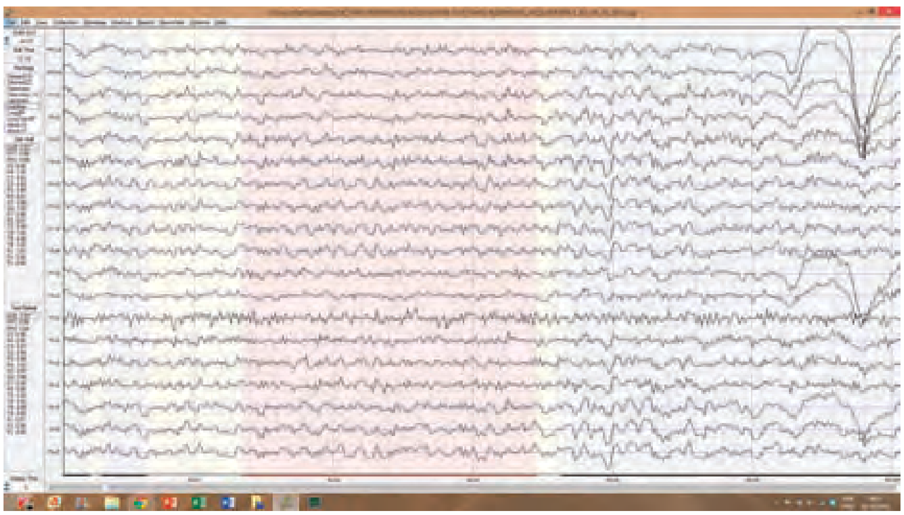


Figure 1: *Raw EEG, Patient OR—Initial Evaluation 06/18/2012*

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13. ERP Tasks | Elena Labkovsky
14. Fundamental Neuroscience for Neurofeedback | Don Cooper
15. Getting to Know LORETA | Joel Lubar
16. ERP Fundamentals | Elena Labkovsky
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BA, BCN

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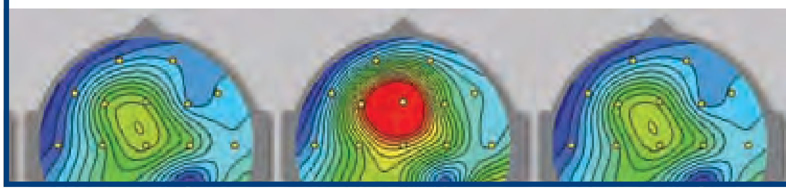
PhD

Robert Turner
M.D., MSCR



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Span database, developed over more than a decade, by Dr. Robert W. Thatcher.

First, one must understand what the maps are showing. The small colored ruler below the first row of heads in the maps illustrates the number of standard deviations (statistical measures for a given population, defined by sex and age) that any neurological activity in a series of parameters, such as power and connectivity measures, in each frequency is near or far from the statistical mean, which corresponds to a normal average.

In order to allow you to follow the analysis that I will do, consider the maps as a matrix depicting brain activity. Thus, note that each head column relates to a given frequency, from Delta (0.5-3.5Hz), all the way up to High Beta (23-30Hz). In the lines, each of the parameters mentioned previously, and others, are illustrated from power to connectivity measures, such as coherence and phase lag.

The first aspect worth mentioning in this patient´s electroencephalographic quantitative map is the low activity in his frontal lobes, practically in all frequencies. This low activity, as demonstrated by Richard Davidson, PhD, was the great villain behind the depression this young man had been experiencing before beginning his treatment.

The reason for this, according to Davidson´s findings (Davidson, R. J., (2004). Biological Psychology, 67, 219-234), is that since the left frontal lobe is responsible for processing positive emotions, and the right lobe, the negative ones, this functional deficiency clearly was preventing this young man from establishing a positive liaison with the events in his life as well as preventing him from properly dealing with his negative emotions.

Furthermore, the frontal lobes are responsible for the executive functions of attention, concentration, abstract reasoning, and even memory functions. The low activity in the frontal lobes helps explain his other complaints of difficulties in attention and concentration, as well as slowed thinking.

Also note that within the last two rows, which reflect connectivity (coherence) and information transmission delay (phase lag), there is a functional disconnection of his left hemisphere (specifically in the language processing areas) in relation to the rest of his brain activity, which caused a delay in his neurological processing in the area in question, especially for the language function. This was clinically manifested as stuttering and difficulty in reading and comprehension of written language.

This pattern, in conjunction with the low activity in the frontal lobes, corresponded perfectly not only with his speech problems but also with his study difficulties that, as explained before, prevented him from performing as expected academically.

Observe now the results, obtained after only four months of treatment with surface neurofeedback, depicted in his post treatment map (Figure 3).

First, see the normalization of the frontal lobes' activity in all frequen-

Continued on page 48

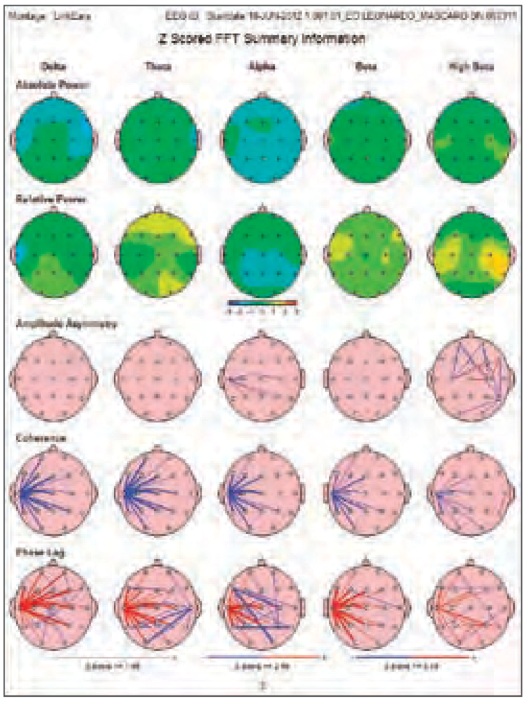

Figure 2: *Patient OR—Initial Evaluation 06/18/2012*

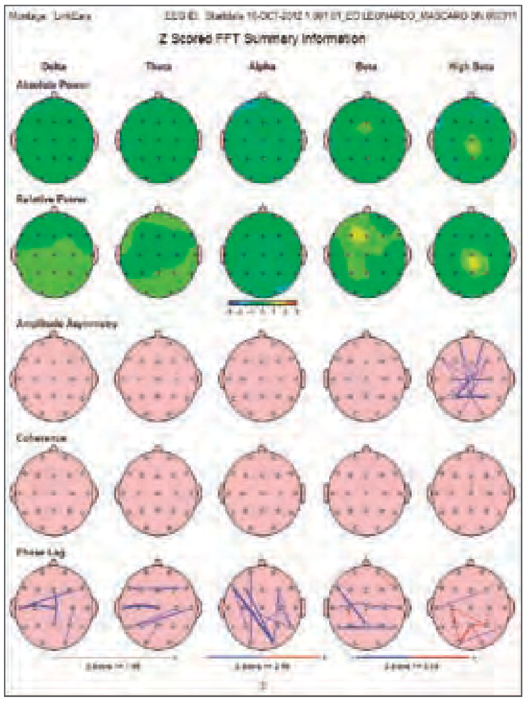

Figure 3: *Patient OR—Final Evaluation 10/16/2012*

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Professional Development and Practice Success: A Report on the Correlation

Jonathon E. Larson, EdD, LCPC, CRC

This study explored the influence of neurofeedback (NFB) continuing education (CE) hours on NFB practitioner net income. One-hundred and forty NFB practitioners completed online surveys gathering demographic information, CE hours, practice revenues, and practice costs. For data set analyses, we utilized descriptive statistics, frequencies, means, standard deviations, and an independent samples t-test. Our results indicated group one, averaging 7.24 monthly CE hours, reported significantly higher net income rates compared to group two, averaging 2.77 monthly CE hours. Group one reported monthly average net income of $4,532.33 and group two reported monthly average net income of $2,932.42. These results suggested that CE hours affect NFB practitioner net income. We offer that investing resources into CE positively shapes client outcomes as well as NFB practitioner outcomes. In this report, we review our findings, discuss explanations for these findings, and offer implications for NFB practitioners.

This brief report provides additional findings to our previous 2012 publication in the *Journal of Neurotherapy*, "The relationship between burnout, interpersonal commitment, client adherence, and quality of work life among neurofeedback practitioners," In our previous study, we found increasing monthly client training sessions significantly increases net income; however, we pondered a question that we did not include in our first study, "Does NFB CE affect net income?" To get right to the point of our question, yes, we found that CE significantly affects net income. We found practitioners in our high CE condition reported significantly higher net income compared to practitioners in our low CE condition.

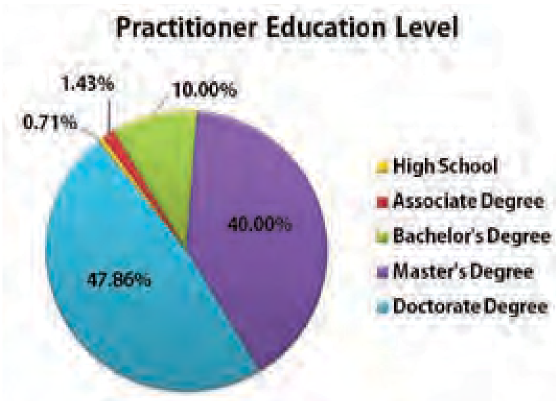
Our literature searches indicated that there has been extensive growth of NFB efficacy and effectiveness studies on psychological and physiological disorders. However, other than our previous study, our literature review did not locate research that explores factors influencing neurofeedback practitioners' practice success. Understanding factors influencing net income may assist practitioners when faced with business and resource allocation decisions, such as, "What are my investment options to maintain or increase net income?" We must point out that we are not promoting the premise of gaining profit from client health problems. We offer that ethical health care providers seek to improve their NFB competency through CE, which may lead to a more successful practice, increased client referrals, and improved client outcomes. For purposes of this study, we hypothesized that continuing education for neurofeedback practitioners improves net income.

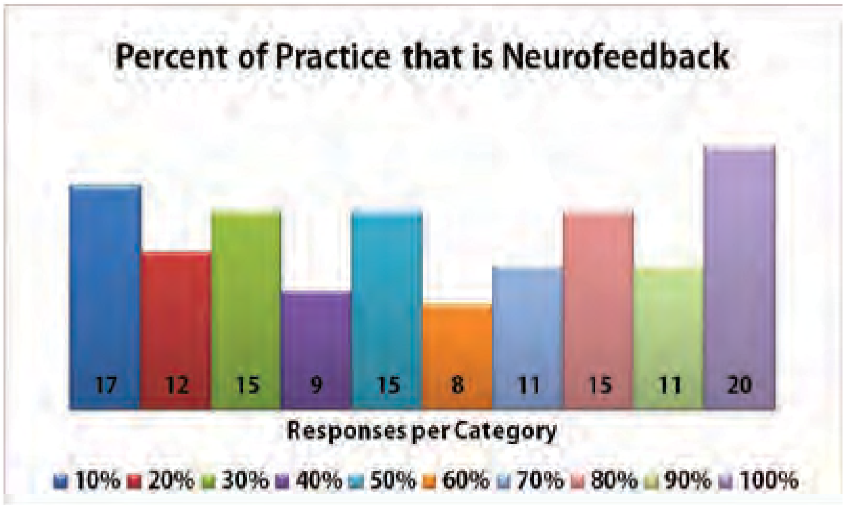
For the following analyses, we utilized the same data set from our 2012 publication in the *Journal of Neurotherapy*. In the preceding study, our sample included subjects with both monthly financial gain and financial loss for all analyses. However, our following study utilized subjects reporting monthly net income gain and we discarded subjects reporting monthly financial loss. We decided to include only practitioners reporting monthly net income gain because we were interested in factors leading to practice success rather than items leading to financial loss. In order to control for financial loss, we filtered out financial loss within our sample before starting our analyses. To obtain our findings, we analyzed 140 usable practitioner surveys collecting gender, age, education, years of NFB experience, monthly NFB sessions, percentage of NFB practice, practice revenues, practice costs, CE hours, and net income information.

Our monthly CE variable was gathered from the survey item, "For an average month, how many hours of NFB training do you receive?" We defined CE as training opportunities to learn and/or improve neurofeedback skills. Our monthly practitioner net income variable was calculated by subtracting the survey item, "For an average month, what are your total NFB financial costs?" from the survey item, "For an average month, what are your total NFB financial revenues?" We utilized SPSS Version 19.0 to complete descriptive statistics, frequencies, means, standard deviations, ranges, and an independent samples t-test.

Demographic information for 140 research participants indicated: male (58%); age (*M* = 54.39 years, *SD* = 11.65); NFB experience (*M* = 9.51 years, SD = 9.39); high school (N=1), associate degree (N=2), bachelor's degree (N=14), master's degree (N=56), and doctorate degree (N=67); percentage of practice is NFB: 10% (N=17), 20% (N=12), 30%

For purposes of this study, we hypothesized that continuing education for neurofeedback practitioners improves net income.





(N=15), 40% (N=9), 50% (N=15), 60% (N=8), 70% (N=11), 80% (N=15), 90% (N=11), and 100% (N=20).

For our net income variable, we found (M = 3339.95, SD = 2859.70, Mdn = 3000.00). For our CE variable, we found (M = 5.45, SD = 8.23, Mdn = 4.00). We utilized the CE variable Mdn = 4.00 to divide our sample of 140 subjects into 2 equal groups for our independent samples t-test. Our first group of 73 subjects indicated (M = 7.24, SD = 7.44) of monthly CE hours, we labeled this group the high CE condition. Our second group of 67 subjects indicated (M = 2.77, SD = 2.25) of monthly CE hours, we labeled this group the low CE condition. An independent samples t-test was conducted to compare net income in high CE and low CE conditions. There was a significant difference in net income scores for high CE (M = 4532.33, SD = 2932.42) and low CE (M = 2159.90, SD = 2140.41) conditions, t (138) = 5.43, p = 0.00. These results suggested that CE influences net income; specifically, results indicated that when NFB practitioners increase monthly CE hours, their net income increases.

Our hypothesis that NFB CE improves net income was supported by an independent samples t-test finding. When NFB practitioners are faced with resource allocation decisions, we offer that financial investment in CE leads to increased net incomes. Improving knowledge and skill sets appears to partially influence financial stability and growth. To partially demonstrate the connection between CE and net income, we offer four explanations:

1. CE improves skill sets, leading to increased client adherence and workload leading to increased net income.
2. Participating in CE affords opportunities to network with other practitioners, leading to referral sources that indirectly increase net income.
3. CE opportunities provide cutting-edge technologies, expanding the scope of disorders addressed by NFB, leading to a larger client pool.
4. CE sessions on evidence-based NFB lead to quicker and more effective outcomes, allowing practitioners to expand the size of their practice.

We do not offer these findings as the only method to increase earnings; however, it appears continuing education offers an effective avenue to increase net income. A potential next research step may explore various CE options and effective methods of disseminating knowledge and the effect on client and practitioner outcomes. We may have missed additional factors due to our study design, sample size, and method of data collection. Overall, we attempted to provide findings to assist practitioners when faced with business investment decisions.

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Reference

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The Bright Future of Brain Fitness Technology

Olga Kara, PhD

The modern world dictates the conditions where individual success often depends on personal adaptability. Adaptation is a fundamental physiological function that adjusts an organism's physiological and behavioral responses to external or internal stressors and increases its chances for survival. The individual adaptation capacity reflects homeostatic equilibrium in autonomic and central stress-regulation systems, where the exhaustion of resources can be a reason for development of pathological conditions.

The human brain is complex, hierarchically organized, modular (both in anatomical and functional levels), and a dynamic information processing system. Brain structures constantly undergo functional optimization that strengthens and modifies existent brain networks in accord with everyday experience and learning and therefore plays a significant role in adaptation processes. The ability of the brain tissue to reorganize its properties by increasing neuronal connections, creating new neurons, and activating additional brain regions to restore lost function (functional compensation) is known as *neuroplasticity,* which provides adaptability, robustness, and control (Shaw et al., 1994).

Yet, the effect of neuroplasticity may have both positive and negative outcomes. Aging, prolonged stress, traumatic brain injury, genetic, environmental and other negative factors can cause the development of pathological connections or loops that affect normal cognitive and behavioral performance and lead to the exhaustion of adaptation reserves. At the same time, any dynamic system tends to perform at the optimum efficiency level; therefore, maladaptive brain performance can be restored with training that allows for positive reorganization of the brain's functional circuits. The interest in brain fitness technology as an instrument of mental health and mental hygiene (Bertolote, 2008) growing along with recent influx in the incidence of mental conditions, which , according to a recent World Health Organization report, can be the second-most important reason for disability and death by the year of 2020 (WHO, 2001; Kessler and Ustun, 2008).

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Special brain training programs have been successfully applied for rehabilitation and education (Kueider et al., 2012; Lee, 2013; Preiss et al., 2013). Recently a number of internet available computer-based technologies were introduced for individual brain training (Posit Science, *http://www.positscience.com/*; Lumosity, *http://about.lumosity.com/overview*), but benefits of these interventions is a subject of discussion and additional studies are needed (Connor and Standen, 2012; Kueider et al., 2012).

The Sharp Brains Virtual Summit: Optimizing Health through Neuroplasticity, Innovation, and Data (June 7 –14, 2012, *http://www.sharpbrains.com/summit/*) offered a unique opportunity for specialists from different fields to meet and discuss the latest trends, innovations, and perspectives in brain fitness technologies. This meeting opened up new horizons for professionals and health consumers seeking understanding of brain health and increased awareness of both existent and newly emerging technologies.

Neuroplasticity induced by training

The process of neuroplasticity underlies human ability to learn and obtain new skills, to make decisions, to perform consecutive actions, and to recover after sudden brain injuries. The question here is how the carefully designed brain training programs may increase particular skills, influence individual performance, and revamp cognitive functions. The latest research defined that neurogenesis continues for a lifetime although it declines with aging (Spalding et al., 2013). Research findings presented by Prof. Keith Wesnes demonstrated that age-related decrease in neurogenesis in the dentate gyrus (DG) of the hippocampus can cause memory deficit and affect reaction time. At the same time, Dr. Wesnes demonstrated that pattern separation training is associated with enhanced activation within the DG, and thus supports the idea of selective stimulation of particular brain structures with specially designed cognitive tasks, giving direction for researchers and developers of brain fitness technology (Wesnes, 2012).

Prof. Michael Posner reported augmented axonal density and improved brain connectivity in response to the training of attention and the attention state (Posner, 2012; Tang et al., 2012). Dr. Adam Gazzaley found significant alterations in brain electrical activity (EEG) as a result of the functional reorganization of neural networks after a set of perceptual training sessions (Zanto et al, 2013). These structural

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and functional changes at the same time are linked with a variety of psychological improvements, which clearly supports the idea that practicing appropriate tasks can activate the exact neural network and enhance its performance.

The generalization of brain training: yes or no?

The transfer theory of learning claims that effects of training may have a positive outcome that boosts performance in another activity or it can have negative or zero effect on other skills. In line with this statement, Dr. Henry Mahncke, CEO of Posit Science Corporation (http://www.positscience.com), pointed out that the results of a particular cognitive training intervention may generalize to other cognitive abilities only if they share the same core neural network (Mahncke et al., 2006; Smith et al., 2009). Thus training of visual speed would not be expected to improve verbal memory or reasoning, but instead will be more likely to generalize to visual attention, self-confidence, or driving safety.

Prof. Michael Posner suggested that structures simultaneously activated during particular tasks are interconnected through specific neural networks (Posner, 2012; Hampshire et al., 2012). Therefore the transfer of training benefits depends on the type of skill trained (perceptual, motor, and cognitive) and the relation between networks implementing definite action. For example, Dr. Adam Gazzaley reported that action video games improved cognitive functions by boosting resistance to the distraction, breadth, and capacity of attention (Anguera et al., 2013). Peter Kissinger, the president of AAA Foundation for Traffic Safety, presented data suggesting that the specifically designed internet-based attention training for drivers appears to successfully increase their awareness and decrease risk of accidents (*https://www.aaafoundation.org/*). Another type of attention exercise presented by Prof. Michael Posner resulted in broad behavioral benefits including increased executive attention, IQ, and self-regulation (Tang and Posner, 2009), while Dr Tracy Packiam Alloway presented a program designed to positively influence both working memory and attention (Alloway and Warn, 2008).

Intuitively, it is clear that the best outcome will be achieved by cross training that activates different brain networks, to increase likelihood that new skills will generalize beyond the specific training task. Moreover, professional teachers and trainers know that the learning or training outcome depends on the particular mental state of the trainee, and that a state of motivation and relaxed concentration yields far better results in comparison with the state of anxiety and distraction. The brain structures that control emotions (affective system) also regulate our autonomic nervous system and are the first to react to environmental inconveniences. Prof. Michael Posner discussed the fascinating results, illustrating not only the influence of the mental state on self-regulation and performance, but its effect on brain physiology (Posner, 2012).

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Neurocognitive assessment, personalized medicine, and biomarkers

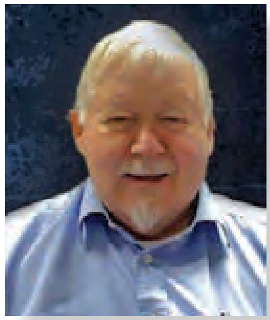
Another important aspect in the field of brain fitness is the individual neurocognitive assessment that defines specific features of brain functioning. The idea of personalization penetrates to different areas of our lives as personalized medicine, and personalized learning style. Normative databases have been used for years in many areas of medicine and psychology for diagnostic, risks assessment, treatment response and outcome prediction, while an individualized approach in education based on precise knowledge of the student's personality, individual interests, and performance is linked with significantly better learning results.

Dr. Evian Gordon opened up discussion of this topic with a conversation about integrative neuroscience that aims to combine information obtained by different measures in order to understand the personal functional organization of the brain. He suggested that individual physiological characteristics, such as heart rate variability (HRV), electroencephalography (EEG), and event related potentials (ERP) may serve as objective biomarkers for neurocognitive assessment (Spronk et al., 2011). For example, a decrease of the amplitude of ERP component (P300) can precede the development of Alzheimer's by decades (Parra et al., 2012). Dr. Antony Bayer, CEO of Brain Resources, pointed out that lower HRV is associated with panic disorder (PD), mild cognitive impairment (MCI) and Alzheimer's disease, whereas higher HRV is a mark of wellness (Birkhofer et al., 2005). The existing normative qEEG and ERP databases approved by FDA and CE (for example HBImed, *http://www.hbimed.com/*) have been used to identify specific characteristics of individual brain functioning and guide in selection of the best training protocols.

The future of brain fitness technology

Interest in brain fitness is fast growing, especially because of its potential benefits for society. With steadily prolonged life expectancy and reduced birth rate, it is crucial to perform activities aimed to reduce mental decline and improve overall well-being. Brain fitness should become a part of everyday activity as an element of general fitness programs. That requires the extensive promotion of

Continued on page 46



Spike Train EEG (STEEG): A New Understanding of the Basic Neurophysiological Mechanisms of the Brain

David C. Brose, PhD

Spike Train EEG (STEEG): A suggested quantum shift in the analyses of electromagnetic activity in the brain.

Origins of the EEG measurement are examined in light of new knowledge regarding the electrophysiological mechanisms and their recording technology. A surprising twist in our views of these mechanisms is explored here.

The STEEG

Spike Train Electroencephalography (STEEG) is a new way to look at the EEG for researchers and clinicians, working under the new proposition that the brain is non-oscillatory in its most basic functioning. This paper is a prelude to developing the technology to utilize the STEEG, in preparation for patent application. The reader is encouraged to consider this quantum shift in the field of neuroscience. Comments or criticisms are invited; your professional opinions will be welcomed at the address below.[1]

Observation

There is a disconnect between neural scientists working intracortically and those working extracortically. Intracortically indicates within the cortex and subcortical features of the brain. Intracortically means there is penetration of the skull to introduce recording media, a surgical procedure, regardless of whether an animal or human preparation. The goal here is a radical change in understanding and using the extracortical explorations that are basically on the scalp. Granted, these are not precise definitions, but do delineate the disconnect. The intracortical scientific endeavor is characterized by Izheikevich (2004), whereby he argues "[If] the goal is to understand the fine temporal structure of cortical spike trains, and to use spike-timing as an additional variable to understand how the mammalian brain neocortex processes information, a spiking model that can exhibit all or most of the 20 neuro-computational properties of biological neurons … is required." He concludes, "Having a network of computationally efficient and biologically plausible cortical spiking neurons interconnected according to the principles of known anatomy of the neocortex should be the goal of every scientist exploring information processing in the mammalian brain," including neuroscientists of the extracortical and generally linked ears for common reference, EEG, functioning of the brain.

The disconnect is in the traditional and classical view of the EEG, particularly from the extracortical or scalp-derived observations of the EEG being of a cyclical nature, when in fact, spiking occurs intracortically but is not acknowledged by the traditional, extracortically-oriented clinician/scientist. In fact, the EEG is the product of the non-oscillatory spike trains recorded from the intracortical dipoles firing as spike trains. The concept of the "Brainwave" being Gaussian in nature, with various frequency bands is described here as a misnomer or at least in somewhat arbitrarily designated frequency bands. Obviously, the extracortical EEG neuroscientist or clinician cannot record these impulses intracortically. Thus, to connect the intracortical spike train with the ability to record extracortically, there needs to be a quantum shift in concept, new recording technology, and new analytical logical algorithms devised. This indeed can be a new direction for the Quantitative EEG clinician/scientist.

Spike Train Electroencephalography (STEEG) is a new way to look at the EEG for researchers and clinicians, working under the new proposition that the brain is non-oscillatory in its most basic functioning.

Spike Trains

The concept of EEG spike trains is not new. Review of numerous neuroelectrophysiologists' explanation of cortical dipole firings is helpful. The thalamo-cortical/intracortical discharges at the cortex are in the form of an impulse or spike in the excitatory synapses. Of course, there are inhibitory influences as well. Complications are exponential in explaining other synaptic firings near and far, from subcortical structures to the network of cortical layers influencing the dipoles at the cortex.

The cortical firing process for spike train formation is elegantly described in the work of Niedermeyer and Da Silva (2005). They describe "Wave Generation." Not to denigrate their wonderful analysis, there is no mention of the means or technology in obtaining waveforms.

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The transition from EEG spike trains to waveforms does not expand on how waveforms are developed in the instrumentation. Nunez and Srinivasan (2006) describe their work as being concerned with "oscillating macroscopic potentials measured on the scalp" as the EEG.

Hysteresis

Not to be heretical, but to rather look at the influences of measurement technology in the extracortical influence of understanding the spike train EEG, it may help to begin to think of hysteresis as a functional component in measuring the EEG. Oftentimes it is instructive to review the basic process of EEG recording. Etevenin (1986) explained: "Electroencephalographic (EEG) cartography" can be traced retrospectively through the entire history of EEG research and development. Three years after Berger's discovery of the human EEG (Berger, 1929), the Fourier analysis was introduced as a means of spectrally describing the EEG (Dietsch, 1932). However, Berger's original equipment was rudimentary; there was no knowledge of EEG spike trains leading to Berger's original observation and definition of Alpha and Beta brainwaves. In fact, the original recording medium was influenced by the phenomenon of hysteresis. Hysteresis has been described as an influence of lags or delays. In Berger's device, the recording device simply could not move fast enough with the electrical spike train signal from the cortex, on the order of the single millisecond duration of spikes, through the scalp, which was therefore hysterically influenced.

Hysteresis is now known as a phenomenon involved in the memory of modern computers. The older core memories of computers would not maintain their status, after an impulse or spike had been supplied, without a refreshing circuitry; otherwise the impulse diminished rapidly and hysteretically. Hysteresis is a commonly known influence in economics, cell biology, visual processes, etc. Audio recording to a CD is based on an electrical spike train, which, via both electronics and the auditory mechanisms of hearing, transform this information into a cyclical nature with a wide variety of overtones or spectrally observed frequencies. Again, these processes are spectrally influenced á la, hysteresis. Technically, slowed down, a series of clicks would be heard, still with color of sound in the hearing process. Neural integration models are based on hysteretic units, connected by positive feedback (Nikitchenko and Koulakov, 2009). A further extension of the influence of hysteresis in neural networks relates to theoretical underpinnings of associative memory. In this proposal, hysteresis improves retrieval ability in the neural network (Wang and Ross, 1990). Hysteresis is also a component of Fourier analyses, even in the computerized Fast Fourier Transform, as a means of reducing data points, in order to develop spectral frequency and intensity graphing of the Gaussian periodicity (Mitra and Bohil, 2008, p. 73, "Fournier analysis expands data into infinitely long sinusoids…" Later, explaining this as dealing with "wave packets of defined duration."

Spike Train Control

Regarding spike detection, (Mitra and Bohil, 2008) describe the necessity to invoke "noise separability." One method is an inherent library of prototypical spikes versus noise. Also thresholding detection for spike events is composing Type I versus Type II, error analyses for discriminating noise versus actual spiking parameters (p. 262). Another prototypical technique may be the cluster analysis for goodness of fit or noise rejection as means of algorithmic spike sorting. Essentially the STEEG would be seen as periodic noise, if not in the FFT (Remond, 1973). Kaiser (2011), in describing the Brodmann's Montage, "The surface EEG … [is] a composition of … chatter." He goes on to describe the "Sinusoidal burst is unlikely to be generated by random noise."

Fourier Transform

The Fourier Transform decomposes a function into oscillatory functions. More precisely said, the Fourier Transform is a mathematical algorithmic process to impose analyses of a time domain frequency and amplitude dynamics on a signal with certain regularities, integrated over a specific time period. The output is the integrated summary of spectral characteristics. These spectra may then be displayed tabularly, graphically, or as topographics, according to comparison with a known normative sample. These data may then be expressed as standard deviation or Z scores. This process, with rapid calculations, becomes the Fast Fourier Transform (FFT), a linear stochastic process. The Fourier analysis is described by (Mitra and Bokil, 2008), p. 73, as expanding EEG data into "Infinitely long sinusoids, which are inappropriate for biological data." They further explain the Fourier Analysis is composed of "wave packets of defined duration." Furthermore (p. 84), they describe the concept of the Nyquist limitations, interpolation and band limiting complications in understanding and interpretation of the Fourier time series (p. 86). The Discrete Fourier Transforms are further complications for understanding the Fast Fourier time frequency analysis, in consideration of the STEEG components from the non-linear and non-stochastic process.

Non-Linear Data

The problem is that original data, such as non-stationary and non-linear impulses or spikes are lost. This data is often interpreted as noise, suggested to not be contributing to the FFT interpretation. The STEEG is an attempt to look at all the data emanating from the cortical dipoles firings, however also anticipating noisy data, which may be difficult to understand. It is recognized that the utility of the FFT for analyzing signals that are localized in time/frequency, excludes or at least does not recognize small transients of finite extent. Likewise, the FFT used in Magnetic Resonance Imaging (MRI) is of stationary, linear, and stochastic data. The Fourier Transform (FT) is not necessarily a representation of the real world of understanding the STEEG. The FT is a theoretical model which does not account for non-stationary and non-linear data. An interesting and applicable computing environment for the advanced needs of calculating the STEEG is proposed by (Reese and Zaranek, 2011) explaining the MatLab use of General Processing Unit (GPU) processing. Their application uses a "Massively parallel array of integer and floating-point processors, as well as dedicated, high speed memory." They also indicate that "The inverse fast Fourier transform (IFFT) can similarly be run in parallel." This is basically a non-linear approach to analyzing the STEEG signal. Otherwise, an outstanding means to understand the Fourier Series and other signal analytic technologies for stationary and linear data is the website *http://www.fourierseries.com*, which presents the theoretical concepts in understandable and commented manner.

An exciting direction in the non-linear development of analyzing the STEEG is the algorithm for compressed sensing (Donoho, 2004). Moler (stated, "When I first heard about compressed sensing, I was skeptical. There were claims that it reduced the amount of data required to represent signals and images by huge factors and then restored the originals exactly. I know from the Nyquist-Shannon sampling theorem that this is impossible. But after learning more about compressed sensing, I've come to realize that, under the right conditions, both the claims and theorem are true." (Moler, 2010)

Non-Oscillatory EEG

Further, the EEG is described as being based on time series as linear and stochastically driven oscillators, but "unspecific regarding the physiological causes of the underlying oscillations" (Olbrich and Wennekers, 2006). In these discussions of EEG origin, hysteresis may be alluded to in commentary about the stochastic process which, by its very nature, to accommodate data reduction, is influenced by the hysteretic component, a confounding element.

> *The focus of the Spike Train EEG (STEEG) is to recognize the influence of hysteresis in both recording/analytic instrumentation and the data reduction component, instead of using the Fast Fourier Transform.*

Proposed Instrumentation

The focus of the Spike Train EEG (STEEG) is to recognize the influence of hysteresis in both recording/analytic instrumentation and the data reduction component, instead of using the Fast Fourier Transform. With the advent of exponentially advanced computing, implementation of VLSI architecture, and massive computing potential, it is time to begin to look at extracortical dipole firings in a different way. By no means is the classical or traditional understanding of EEG to be denigrated, rather it is suggested here to move to another sphere of understanding of extracortical firings from the intracortical spike trains. Much has been written about cortical firings of dipoles, typically with the unwritten acceptance of hysteretic influence in the extracortical domain. The proposal here is to begin to observe the Spike Train EEG (STEEG) in a new way.

Clinical Applicability

Clinical applicability, of course, is necessary whenever new technology is to be introduced. We know that Spike Train EEGs of the brain are of about one millisecond time duration of spiking. Also, comparing EEG with MagnetoEncephaloGraphy (MEG), in terms of sensors, the MEG is much more sensitive (Niedermeyer and Lopes da Silva, 2005) than the EEG. In number and in the research domain, the "scalp sensors for EEG are 128 and MEG are 300, to account for scalp distribution of the activity of each [extra]cortical macro-column described by equivalent cortical dipoles." The typical clinical International 10-20 linked ears EEG electrode arrangement is for 19 active electrodes. However, the MEG instrumentation is many thousand dollars more expensive than the EEG, also involving cryobathed and cooled Superconducting Quantum Interference Device (SQUID) sensors in the MEG. It is here proposed that the STEEG approach may allow a finer comb, ostensibly approaching MEG functionality, without the cost. In addition, the STEEG may be linked with Low Resolution Electromagnetic Tomography (LORETA), new topographic presentations, and new 3D paradigms to observe this marvelous brain to expand our understanding. The STEEG may be approaching true real-time analyses of the brain's electrochemical activity. This would be an important divergence from the current time-limiting and temporal loss from imaging such as the FMRI and even the MEG.

Magnetoencephaogrphy (MEG) Ala Connectivity/Synchronicity

A real plus in near on-line analyses with the STEEG is the applicability of connectivity. Recent EEG research has identified a lack of synchronicity in the brains of the autism spectrum/Aspberger-diagnosed children. "In particular, the left-hemisphere language regions showed reduced connectivity in autistic children, which helps in explaining a common symptom of the disorder, lack of nonverbal and verbal communication." This was described as lack of coherence, with the noted emphasis, i.e. "the more synchronicity there is, the more closely connected the two regions must be," in the normal child's brain (Brice, 2012).

The Mirror Neuron in autism spectrum disorders may be identified as connectional in nature. (Oberman, et al., 2005) used C3, CZ, and C4 in the sensorimotor cortex, observing the frequency of mu suppression in autism. This is another application where STEEG could be superior in the research and potential clinical applicability.

Connectivity, as described in the quantitative EEG (qEEG) is the measure of coherence (COH), basically between two or more cortical sites. COH can be either excessive or reduced, but ultimately expresses the relationship, activity-wise, between disparate cortical regions. Collura (2008) comments that "a connectivity may be sensitive to the phase of the signals (or it may ignore phase) but may be sensitive to the time, or both. Furthermore, the data source such as raw waveforms, transformed quantities using FFT, or filtered waveforms produced by digital filters or complex demodulation will affect the connectivity measure." However, it is anticipated that the STEEG will be non-hystertic in measurement and hence a purer form of connectivity between disparate extracortical sites' electromagnetic activity. Collura (2008) also discusses the value of asymmetry between sites as a useful measure.

Magnetoencephalography (MEG) as a Functional Extension

The STEEG approach may approach MEG sensitivity, by incorporating non-linear and basically non-stochastic elements in, possibly circumventing hysteretic influences in both computational and instrumental observations of the electromagnetic signals from the brain. By no means is the STEEG approach a panacea, recognizing the profound complexity of the brain's EEG, but by the same token attempts to recognize the hysteretic component in this new observation as to how this marvelous brain works. To ignore hysteresis is to bury the proverbial head in the sand, ignoring its influence in understanding whether the brain is oscillatory or not.

"Because the electromagnetic fields reflect the instantaneous distribution of currents in the brain, EEG and MEG signals have resolution in the millisecond range. The spatial resolution is poor, however. It is limited both by the number of sensors that can be used and by the ill-posed nature of the inverse problem that relates the sources to the fields. Multiple source distributions can give rise to the same measured EEG and MEG signals." Mitra and Bokil (2008) p. 273.

VLSI

An exciting new approach to understanding the electrophysiological parameters of the extracortical EEG, or of the described spike trains of the brain, is the Very Large Scale Integration (VLSI) instrumentation for the Local Field Potentials (LFP), of the Thakor Labs. They are associated with Johns Hopkins University's Neuroengineering & Biomedical Instrumentation Lab. Mollazadeh describes the Tunable Frequency Response component, looking at the spiking nature of the EEG, which is promising in developing instrumentation for defining and looking at the STEEG method (Mollazadeh, et al., 2009).

The Riddle

The brain is a riddle in that there are many veiled meanings in understanding its functioning. Technically and metaphorically, ingenuity and careful thought processing are necessary for understanding the brain and how it functions. Ranging from phrenology to microcosmic analyses of brain function at the intracerebral level, we are faced with the riddle of expanding knowledge of this splendid brain. (Izhiekevich, 2010), addressing the Dynamical Systems in Neuroscience stated (p. 320), "The parameter values that match firing potentials of biological neurons … are only educated guesses …" and (p. 325), "One can only be amazed by the diversity of bursting patterns and time scales." Furthering this argument of the riddle of the brain, (Trappenberg, 2010 p. 79) commented that, "Noise is essential for the argument with spiking neurons … the noise in a population of spiking neurons might be essential for information processing in the brain." Otherwise, noise is an undesirable artifact to be eliminated, generally, in the current analyses of the electrophysiology of the brain.

The potential for a better understanding of the riddle of the brain, STEEG has the potential for advancing our knowledge of the brain, particularly from a clinical and extracortical perspective.

The brain is a riddle in that there are many veiled meanings in understanding its functioning.

Future Thinking

STEEG connectivity is the name of the Brain Game, to speak metaphorically. Disparate dipole firings from different regions, even as Brodmann functions are described, is a basis for recording inter-regional connectivity coherence between regions may be an excellent advance for STEEG recording and clinical utility, as to be developed in the new instrumentation. Another metaphoric comparison may well be how today's World Wide Web (WWW) of the internet physically works. Tubes (2012) is a recent published observation, exploring the physical attributes of the internet. There truly is a worldwide interconnecting network, even with built in redundancies and fail-safe mechanisms. For instance failure of one component requires a rerouting of data. Maybe the WWW is not so different from the brain, in a microcosmic scale. As stated, the recent book about "Tube"… explores the current physical features of the internet, how and where it works. Hopefully we can develop the algorithms of the STEEG to better understand this human brain better.

Computers and the brain have been considered as having some shared attributes. Attempts towards developing artificial intelligence have been successful on a small scale. It may be appropriate to consider the even more microcosmic comparability of these marvelous aspects of today in computer science. For instance, computers operate on the most basic element of 1 or 0. Some consider these as either an on or off digital status. For our purposes here, it might be appropriate to consider the 1 or ON as of a short duration spike. Extending this concept in the computer to the Hexadecimal level, of which two hexadecimal numbers are known as a byte. The combination of the byte comprises the entire ASCII set of 256 characters, which is largely the entirety of general communications within and between computers, on the machine language basis

Patent Application

Patent application is to be made for the Spike Train EEG (STEEG) to explore a new mode of understanding the profundity and complexity of electro-physiological signals from the brain, as recorded extracortically. A prototype needs to be developed. Careful consideration has been given to the development phase, towards developing such a patent.

Afterthoughts

The undertaking and championing of the STEEG is not done without quaking, recognizing the giants of our present understanding of the EEG from Berger to the present. Sometimes it helps to step back and look at our most basic assumptions. Many of my colleagues who have admonished, "Look at the raw and basic EEG" for a better understanding of both clinical and basic processes, is the attempt to achieve this now.

David C. Brose, PhD (clinician/scientist), involved in neurofeedback from the early days with the analog systems. This intricate functional brain is exemplified by the statement by A. Einstein, "God doesn't play with dice."

ONLINE SUPPLEMENT

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

The Bright Future of Brain Fitness Technology continued from page 41

mental hygiene that will add to the general public's awareness of brain fitness benefits and raise their willingness to enhance their own cognitive reserve and prolonged active life.

The future of brain fitness is in a combination of different approaches. It might imply the power of the gaming industry and suggest new forms of game environments directed to activate particular brain networks for the individual psychophysiological characteristics of the trainee. The online monitoring of brain functioning can be used to control the current mental state for optimal performance.

I believe that this type of application can be on the market in the near future as nearly every part of such a system is already on the market. The Emotive headset introduced by Tan Lee, the CEO of Emotive Lifescience is wireless, user-friendly, and an affordable EEG-based device that can be used for a number of purposes, from gaming to rehabilitation (Duvinage et al., 2013; Foc et al., 2011, Lang, 2011). The internet-based cognitive training program offers the possibilities to explore additional support for individuals aimed to enhance particular functions (Mahncke et al., 2006).

The neurofeedback approach is one part of brain fitness technology that can be used to monitor, visualize, and help the customer to acquire the desired brain state. Neurofeedback may reduce the time needed to understand and learn particular skills. Non-invasive functional brain mapping (qEEG. ERP, sLORETA, fMRI etc.) help to disclose individual problems and evaluate the exact training parameters, which can help an individual choose the best available training program. Wireless EEG devices can be used for home or on-the-go-based monitoring of the cognitive brain states that provide the user feedback of his ongoing condition. Finally, the promotion of multi-age social interaction among communities will have a significant implication on mental well-being as it was shown by Prof. Peter Whitehouse with his Intergenerational Schools project (George and Whitehouse, 2011). All this together promises a bright future for brain fitness technology.

Olga Kara, PhD, has strong academic background in Biology and Medicine with nearly 20 years of experience in human neurophysiology and psychophysiology. After she obtained her PhD in Human Neurophysiology in 1998 at the St-Petersburg State University, she was involved in a number of national and international projects concerning development of new diagnostic and treatment methods for different brain diseases including, ADD/ADHD, OCD, Epilepsy, Parkinson disease, Addition, Brain stroke etc. Her multidisciplinary scientific interests involve, among others, brain information processing, cognitive brain functioning, brain-computer interface and neuroplasticity as a basis of learning and adaptation in the brain.

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

Winter 2013 ISNR Research Foundation Update

Congratulations to Our 2013 Mini-Grant Awardees:

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University of Pennsylvania
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UC San Diego
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Got an idea for a book? Let us know!

Around the World with Neurofeedback

Dr. John Davis has been gathering information regarding activities around the world. Following are some of the reports.

Dr. Carlos Novo of Monterey, Mexico: The second Introductory Course from the Mexican Society for Biofeedback and Neurofeedback was presented in early July. The University of Mexico (UNAM), through the National Institute of Psychiatry, is about to graduate its second generation of Diplomates in Biofeedback. This is the biggest university in Latin America. With the backing of the Mexican Society for Biofeedback and Neurofeedback, the aim is to add new classes in this Diplomate, especially in EEG biofeedback. Work is also being done to create a two-year postgraduate program in biofeedback, involving the collaboration of several universities. In early October, an international faculty offered a series of teleseminars on neurofeedback through Javier Cano and Innovaciones Software of Cali, Colombia.

Dr. Rien Breteler of Nijmegen, Netherlands: The Dutch Psychological Association's neurofeedback section continues to thrive with new members.

Dr. McGill Scott of Johannesburg, South Africa:

South Africa continues to add to its numbers of BCIA Diplomates in neurofeedback. A constitution for neurofeedback practitioners is taking shape that may mandate 36 hours of didactic training along with a license in a health care profession. Efforts are under way to encourage collaboration and mutual support among providers from different schools of neurofeedback.

Dr. Santiago Brand Ortiz of Bogota, Colombia: The field of neurofeedback is both young and relatively new in Colombia. In spite of this, there are a growing number of professionals that are becoming interested in optimizing brain functioning through technology. This is due to parents of ADD/ADHD children becoming aware of the dangers and shortcomings of medication and also because of adult clients who want to treat anxiety, depression, and other conditions as well as optimize their performance using less invasive and more benign methods. With this in mind, neurofeedback is delivering on that promise. In my practice I use different modalities so as to provide my clients with the best service possible. For assessment I do qEEG and a psychophysiological stress profile. I have a CBT therapist on my staff that does all additional testing as well as psychotherapy. I also use HEG, peripheral biofeedback (with a special emphasis on HRV) and also of importance Audio Visual Entrainment, CES and tDCS. I invite health professionals in Latin America and the rest of the world to include this modality as part of their repertoire.

In the United States, the following organizations are alive and well: If you don't belong to one of these, take a look and join many of your colleagues in supporting biofeedback/neurofeedback. Biofeedback Society of Florida; Mid Atlantic Society for Biofeedback & Behavioral Medicine; Northeast Regional Biofeedback Society; Southeast Biofeedback & Clinical Neuroscience Association; Biofeedback Society of California; Biofeedback of Texas; Mid West Society for Behavioral Medicine & Neurofeedback

Healing Stuttering with Z-Score Neurofeedback continued from page 35

cies, indicated by the change from bluish green in the first map, to green on the final map, indicating signalizing normalization of activity as related to the colored ruler of standard deviations. Furthermore note, at the last two rows of connectivity (coherence and phase lag), the complete normalization of left hemisphere activity. One can see that these brain areas reconnected properly with the rest of the brain (absence of blue lines in coherence row), also presenting because of this, a normalization on processing speed and neurological information transmission (absence of red lines in phase lag row).

The result of this? The patient overcame his depression, concentration, and attention deficits, as well as the stuttering. Today, this young man able to participate in conversations with friends without feeling embarrassed, resulting in a considerable increase in his self-esteem. His school performance at college is now much better, with his grades higher than ever. Also, he can now say he has eradicated his depression and mood swings. Better than that, he has been off of medication for more than a year now, and doing very well! He even has more time to live life and enjoy the things that he truly loves, like playing rugby.

Leonardo Mascaro, MNeurosci, BCN, psychologist with more than 15 years of clinical practice, founder and owner of Brain Tech Inc., the first brain center in Brazil to work with Z-score Neurofeedback, is the only Brazilian both to have trained with Dr. Margaret Ayers and to be certified by BCIA in Neurofeedback. He is the author of two books on the subject, both in Portuguese: "A Arquitetura do Eu" (Campus-Elsevier, 2008) and "Para que Medicação?" (Campus-Elsevier, 2011). You may reach him at: leonardo.mascaro@braintech.com.br.

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