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My first three months as ISNR President have been interesting, to say the least, and incredibly busy. ISNR has been steadily making progress on all of the organizational changes it has undertaken. There are several things that ISNR can proudly announce are in the works or accomplished. As of the time of this writing, the most exciting news is that ISNR has hired a new Executive Director, Cynthia Yablonski, MBA will be joining ISNR around the first of May. Cynthia has been the Executive Director for The Protein Society for the last 8 years of her 20+-year career of managing various organizations. Her duties at the Protein Society included responsibility for the day-to-day operational activities, membership, managing the monthly professional journal (which has over 800 articles a year), and being responsible for a yearly conference of 1,000 attendees in the United States, and an additional European Conference every two years. In addition to her MBA, Cynthia has a Bachelor’s degree in Biology with an emphasis in neuroscience which led her to manage a Brain Bank Database at the National Institute of Mental Health. She presently lives in the Virginia/Washington DC area and has done lobbying work on Capitol Hill for The Protein Society and other organizations for which she worked. We are very excited to welcome Cindy to ISNR and look forward to her becoming a part of our organization. I will make an announcement after she is fully on board and I encourage all of you to contact Cindy, introduce yourself, and welcome her to ISNR.

I would like to thank the ISNR Search Committee for their dedication and perseverance in finding and recommending Ms. Yablonski. The committee consisted of past president, Leslie Sherlin, president elect, Randy Lyle, Judy Crawford from BCIA, and me. A special thanks to Judy Crawford for going above and beyond the call to help ISNR in its time of need. As well, a thank you to both BCIA and The Resource Center for supporting Judy’s availability to us. Karen Forbes, INSR interim Executive Director, wore two hats as staff support and consultant to the committee. Personal thanks to you all for your extended service during this process. Your diligence produced a great result.

ISNR has been steadily making progress on all of the organizational changes it has undertaken.

The next great announcement at this time of writing is that the new website is up and running after being delayed for several months during reconstruction. The new website look was showcased at last fall’s ISNR conference, and it was intended that the site would be functional shortly after that. However, delays by the company hired to work on the site led to a much later completion date. It has certainly been worth the wait. The whole look and functionality of the site is wonderful and we have received many compliments. Thanks to past president Leslie Sherlin for making this his pet project and seeing it through to the end. If you haven’t checked it out, do so and enjoy!

The beginning of the year was the deadline for comments on the ISNR practice guidelines white paper. We received many comments from both members and non-members. As most of you witnessed, this kept many list-serves very active. We had a full range of comments from positive to negative and the Standards committee has been very hard at work utilizing those comments to revise the paper. I have been monitoring the progress and am pleased with the changes I see and feel that the membership will be pleased with the changes. The process for construction of this white paper is that the committee will finish revising the paper and submit it to the ISNR Board for its acceptance of the revisions. Then, the paper will be submitted to the Journal of Neurotherapy for publication.

The call for papers is now open for the ISNR Annual Conference that will be held September 19-23, 2012 at the Hyatt Regency Grand Cypress Hotel in Orlando, Florida. Start making your plans to attend this conference.

In closing, I would like to again extend the invitation to you to become a part of ISNR and join one of its committees or offer to volunteer in some capacity. As well, consider donating to the ISNR Research Fund. Donating time and/or money to ISNR is an investment in our field and yourself.

Richard E. Davis, MS
Welcome to the spring 2012 issue of NeuroConnections. Our theme for this special issue focuses on posttraumatic stress disorder (PTSD). Exposure to trauma is certainly not a new phenomenon to humanity. Writings dating back to the period before Christ reflect an understanding that trauma can affect an individual for years after the traumatic event. As early as 490 BC, Greek historian Herodotus described an Athenian soldier who became permanently blind after witnessing the death of a fellow soldier during the Battle of Marathon, although he himself had experienced no apparent physical trauma. Nor do reports of posttraumatic stress reactions appear to be unique to the human species; elephants have been reported to suffer from posttraumatic stress upon seeing members of their herd shot by hunters, while military service dogs deployed in war zones have also been observed to develop trauma symptoms after witnessing combat.

Although it had been well known that the stresses of combat could produce long-term responses, PTSD to the third edition of its Diagnostic and Statistical Manual of Mental Disorders (DSM-III). Prior to 1980, the disorder now known as PTSD had been recognized by such varied names as nostalgia (reflecting a Civil War era notion that symptoms were caused by homesickness), shell shock (reflecting a World War I-era misattribution of symptoms to blast exposure), or battle neurosis (reflecting a World War II-era notion that development of such symptoms were a sign of personal weakness or flaw).

Reflecting on his own experience as the survivor of four Nazi concentration camps during WWII, psychiatrist Victor Frankel, in his book, Man’s Search for Meaning, offered, “an abnormal response to an abnormal situation is normal behavior.” This goes to the heart of current views of PTSD, which, informed by a growing body of neurophysiological and neuroimaging data, have increasingly come to understand PTSD as a normal and predictable physiological endpoint to the body’s efforts to adapt to levels of stress for which it was never designed.

A growing body of evidence highlights that PTSD is, at its core, a condition defined by profound dysregulation of both neural and peripheral stress circuitry (Kolassa et al., 2007; Krystal & Neumeister, 2009). This modern redefinition of PTSD as a neurophysiological, rather than a primarily psychological disorder, places PTSD squarely in our field of expertise.

As they peruse the current issue, alert readers will discern a common theme. Despite differences in treatment protocols, practitioners are reporting that, for a substantial percentage of sufferers, interventions targeting neurophysiological dysregulation are proving to be highly effective in addressing symptoms of PTSD, improving patient readiness to take advantage of more psychologically-based therapies, and in some cases, obviating the need for traditional psychological treatment. Moreover, leaders in the field are reporting a trend toward more efficient protocols, yielding symptom improvement in significantly fewer sessions than were required even five years ago. These findings stand in contrast to primarily pharmacological and psychotherapeutic treatment, where outcome data appears to suggest that the disorder is increasingly resistant to treatment once symptoms have become chronic.

Roger Riss, PsyD

References
Dear ISNR Members,

As you have read in ISNR President Richard Davis’ article, ISNR’s Board of Directors has hired their permanent Executive Director, Cindy Yablonski! While I will miss working with the Board and members of ISNR, I am thrilled that Cindy will be taking over the operational leadership of the organization. Cindy has the experience, professionalism, and vision to lead the organization for growth and expanded member services, based on strategic thinking, inclusivity of the various interests and priorities of the stakeholders and a keen understanding of nonprofit best practices. I congratulate the Search Committee, chaired by ISNR President Richard Davis and members Judy Crawford, Randy Lyle, and Leslie Sherlin on the thoughtful, transparent, and thorough process that brings Cindy to the ISNR helm, and I was honored to serve as their staff advisor.

The search process was time consuming; it began in October 2011 with determining the process, a review and update of the job description, and finally, crafting of the job posting. This review exercise allowed the committee to determine that they were looking for someone with nonprofit/association management experience, a track record in membership development, and a very high level of professionalism, especially in the area of relationship building. Once approved, the posting was listed on the ISNR website, in The Nonprofit Times, Idealist.org, Opportunity Knocks, and at ASEA (The Center for Association Leadership). This variety of sites insured that the posting was picked up by various Executive Director search engines and resulted in more than fifty packets of interest.

Each packet was reviewed with an eye to the traits and experience the committee had identified as critical for the future Executive Director, and separated into those who fit the criteria, those who had some of the criteria and those who did not fit the criteria. Those candidates with the closest match were called by the members of the Search Committee for a preliminary introductory conversation. As a result of this conversation, Cindy quickly became the leading candidate; however, the committee followed through with the predetermined process of conducting first and second interviews. Cindy remained the first choice, and after several follow-up phone calls was asked to make a presentation to the Board of Directors in January. Her presentation was professional, creative, and wonderfully overreaching!

The Board of Directors unanimously approved a face-to-face interview with Richard or his designee and the opening of the discussion on the terms of employment. It was an exciting, celebratory decision for the Board. Richard flew into Baltimore within days, and he and Cindy met and sealed the deal! Thanks to the efforts of the members of the Search Committee, ISNR will have an experienced, professional and forward thinking Executive Director early in May. The process that began with the exhausting Interim Executive Director Project through The Nonprofit Center at LaSalle University School back in May of 2011 will end successfully in May of 2012. I congratulate everyone for their commitment to a process that points ISNR to a bright future!

I also have the pleasure of announcing that NeuroConnections has a new interim Managing Editor, Barbara Trumbo. Barbara joined the ISNR team in January after we learned that the long-time Managing Editor, Cindy Kerson would no longer be serving in that capacity. Cindy, in partnership with editors Merlyn Hurd and Roger Riss, has done an incredible job over the years developing the newsletter into the high quality, respected member service that it is today. We wish Cindy Kerson well with her future endeavors and thank her for her efforts.

Barbara, using her skills, experience, and desire to move NeuroConnections to an even higher level, has picked up the reins (much to my relief and with Merlyn and Roger’s appreciation). Once Executive Director, Cindy Yablonski comes on board, permanent arrangements for the Managing Editor position will be made; until then, NeuroConnections will continue to be a valuable member service and source of research, information, products, and creative thinking in the neurofeedback field.

Finally, I congratulate the Board of Directors for their time and efforts during the Interim Executive Director Project. Opening the closets to a stranger, listening to recommendations for best practices and improvements in organizational structure, and implementing those recommendations, is never easy and is a process that requires incremental change and a long-term commitment. I wish ISNR the very best as they continue their journey of organizational development and look forward to hearing great things in the future.

With best regards,

Karen Forbes

Do have a wonderful winter and see you in the spring.

Merlyn Hurd, PhD, BCN Fellow

A higher level of suicides and sexual assaults in veterans than ever before. This is looking at just the Armed Forces; when one expands out to the larger society, the rate of PTSD is truly shocking. Now for the really odd part. In the DSM IV TM for the diagnosis code that psychologists can use, PTSD is not listed. One has to use a number of other codes to portray the disorder one is working with. Certainly, we can treat anxiety, headaches, dissociation, depression, anger, sexual issues, and a score of other disorders, so we are not hampered by the oversight of the committee who formulated the DSM IV TM or the forthcoming DSM V. The articles by the clinicians and researchers in this edition are providing you with the tools and wisdom necessary to work with these clients or refer them appropriately to the clinician that has the expertise.

Karen Forbes

In the DSM IV TM for the diagnosis code that psychologists can use, PTSD is not listed.

Merlyn Hurd, PhD, BCN Fellow

Karen Forbes

Dear ISNR Members,
Letter from AAPB ED

Volunteerism: the Life Blood of AAPB

Without the dedication and support of volunteers, organizations like AAPB and ISNR cannot exist. Volunteers provide the professional expertise for our organizations to thrive, and guide the ongoing advances that are critical in our ever-changing world. AAPB is always on the lookout for good volunteers who are interested in advancing the mission of the organization and the practice of biofeedback and neurofeedback.

It’s easy to understand when members suggest that they simply don’t have time to serve on a committee or task force. Giving of volunteer time requires a commitment that may not be appropriate for everyone. But, there are numerous volunteer options that do not necessarily require huge outlays of time, and the rewards can be tremendous. AAPB offers a number of opportunities for members to volunteer for service in ways that are time limited. When the task is complete, your responsibility is complete. For example, members may serve as abstract reviewers, new member, or first time conference attendee mentors, or on special task forces with short-term goals and objectives.

Naturally, we need volunteers to serve on committees with more long-term objectives as well, such as our education, membership, and conference planning committees, to name a few. If you find the shorter term volunteer options more appealing, have we got a deal for you! A few of the task forces that are in need of volunteers and their missions:

- Marketing: develop strategies to market AAPB publications, the annual conference, and other educational offerings; create and promote a speaker’s bureau; nurture organizational relationships; and promote membership growth in AAPB.
- University Outreach: develop a white paper on the need for a consistent biofeedback curriculum and the recommended elements.
- Website: work to increase the Foundation’s presence on the website; keep the Clinician’s Resource Library updated; and monitor web content for accuracy and timeliness.
- Student: evaluate ways to better engage the student community within AAPB.
- Social Networking: create and execute strategies to leverage social networks that will reach potential members and educate the public on the benefits of biofeedback.

As AAPB enters its new Board year, the Board will be setting its priorities and will need volunteers to address the objectives of these and possibly other groups.

So, you ask, what’s in it for me? Please consider these benefits of volunteerism:

- Hone your leadership skills.
- Make a difference for your professional organization and your fellow members.
- Share your knowledge and experience in ways that can offer improvements in education, best practices, business development, and other professional skills.
- Build your network to enhance your professional practice and business skills.
- Create and develop changes to positively shape your profession.
- And much, much more.

Participating at the level of the short-term can also lead to opportunities to serve on the Board of Directors, and anyone who has served in that capacity will tell you that the benefits far outweigh the costs of time and effort.

Interested? Call me directly at 303-422-8436 or by email at dstumph@resourcenter.com. We look forward to hearing from you. Together we can make a difference for AAPB and for the field of biofeedback. Call or email today!

David L. Stumph, IOM, CAE

In the fight against pain, psychophysiology and biofeedback are valuable, yet little known resources.

In this new edition, author Richard Sherman, PhD, takes a comprehensive look at:

- the mechanisms behind pain disorders
- provides specific, easy-to-understand analyses of various assessment and intervention techniques
- an overview of mechanisms of pain involving the central and peripheral nervous systems
- the endocrine system
- genetic influences
- cognitive and emotional factors that can influence pain perception.

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

Order your copy today at www.aapb.org or telephone 800-477-8892
Meet ISNR’s New Executive Director

Cindy A. Yablonski, MBA, is the new Executive Director of the International Society for Neurofeedback and Research (ISNR).

Ms. Yablonski served as the Executive Officer of The Protein Society from 2003–2012. The Protein Society is a non-profit scientific organization dedicated to furthering research and development of protein science through scientific conferences that facilitate communication and collaboration among scientists across the globe. During her eight years with the Society, Yablonski brought leadership, vision and strategic direction; focusing on increasing membership; providing quality programs; and improving the overall performance of the organization. She also had oversight for the Society’s annual U.S. and international conferences as well as the peer-reviewed scientific journal, Protein Science.

Ms. Yablonski has held a variety of positions in non-profit organizations, the government, and academia. She has served as the Vice President of Research, Science & Technical Affairs at the International Bottled Water Association; as the Research Director of the Drinking Water Research Foundation; and as a Manager of Scientific Programs at the American Industrial Health Council; and as an Associate at ICF International.

Early in her career as a biologist, Ms. Yablonski helped manage a brain bank database for use in research programs at the National Institute of Mental Health. She also worked at The Johns Hopkins University School of Medicine as a researcher studying Alzheimer’s disease and designing and implementing neuropathological research projects.

Cindy holds an MBA from George Washington University, DC. She has a BS in Biology from Dickinson College, PA.

Ms. Yablonski is a member of the American Association for the Advancement of Science (AAAS), the Women’s Council on Energy and the Environment, and the Council of Engineering and Scientific Society Executives (CESSE), a network of leading association executives.

A native of New Jersey, Cindy resides in Falls Church, Virginia with her husband, their daughter, and a Jack Russell terrier.

ISNR looks forward to Cindy’s joining us around the first of May.

Remediation of PTSD using Infra-Low Frequency Neurofeedback and Its Implications for the Reunification of Biofeedback and Neurofeedback

Siegfried Othmer

Remediation of PTSD using Infra-Low Frequency Neurofeedback and Its Implications for the Reunification of Biofeedback and Neurofeedback

The careers of the scientist/practitioners in the field have undoubtedly had in common the experience of gradually rising expectations about what is possible to achieve in terms of improved self-regulatory capacity and mental functioning with the aid of neurofeedback. One might have expected some plateauing after a while, a firming-up of one’s expectations, but the surprises keep coming and they are consistently on the upside. In our own experience, one of the biggest surprises has been the growing effectiveness of neurofeedback with PTSD, along with the related conditions of developmental trauma and the autism spectrum. All of these conditions seemed so utterly intractable in the past.

The theme of this issue of NeuroConnections is PTSD, so I have been asked to focus on this topic as well. But this is a newsletter, not a scientific paper, so PTSD will merely serve as a focal point for larger themes. What sets PTSD apart from our clinical work in general is the concentrated effort that has gone into this area by virtue of the great need among our returning veterans. We have attempted to meet this need through a non-profit entity, Homecoming for Veterans (hc4v.org), which has attracted even international participation among clinicians. As a result of these collective efforts, a large database of clinical results has been gathered that is now available for “data-mining.”

This collection of data has allowed us to firm up some expectations even for those PTSD symptoms that are not commonly encountered in an individual practice. The data have the significant advantage of being drawn from a wide variety of sources, and therefore include contributions to outcome variance that would not be an issue in a formal research design. This gives any findings an intrinsic robustness that could not be matched in any single funded study. The accumulated data are relevant to clinical effectiveness in realistic settings, and thus answer the probative question that confronts policy-makers.

Infra-Low Frequency Training

The training technique used almost exclusively in this work is the Infra-Low Frequency (ILF) training that we have developed over the past six years. The training can still be considered in the same frame as traditional frequency-based neurofeedback, in the sense that the training effects are highly frequency-specific. But in this low frequency range, where a full cycle can take over an hour, the standard signal-processing techniques are not applicable. One has no choice but to abandon that approach and move simply to signal-following, by analogy to what is commonly done in peripheral biofeedback. The neurofeedback trainee just observes the evolving time course of the differentially amplified slow cortical potential. There is no threshold, and there are no discrete rewards. The process is no longer one of operant conditioning as generally understood. In the case of infra-low frequency training, the differentially-derived signal undulates between positive and negative phases, and one is not more virtuous than the other in the process of the brain engaging with the signal. No positive or negative valence can be attached to the signal. It simply is. The brain interprets the signal in terms of its own internal activities through a continuous process of correlation. None of the elements of this process are perfect, and therein lies the basis for a perpetual challenge in which the brain attempts to reach closure between its internal state and its interpretation of the slowly migrating signal. This is a radical departure from the way neurofeedback has been traditionally...
Multimodal Management of Military Combat Posttraumatic Stress Disorder (PTSD)

David Hagedorn, PhD

A flurry of articles and Senate Panel post-traumatic stress disorder (PTSD) hearings in November 2011 highlighted what mental health clinicians serving the military already know—combat-related PTSD is stretching the military and Veterans Administration mental health resources beyond projections. Thousands of military personnel flood the VA system each month amid estimates that only half of those with PTSD seek treatment. With the currently planned large scale combat troop reduction, there should be no surprise that we are now treating just the tip of the iceberg.

Before these men and women with varying degrees of combat stress response severity make their way from active duty to a medical out-processing procedure they are evaluated and treated by active duty and civilian clinicians for several months. They are commonly provided interventions which include antidepressants, beta- and alpha-blockers, atypical antipsychotics, anticonvulsants, prolonged exposure therapy, eye movement desensitization and reprocessing, and group therapy (Benedek, Friedman, Zatzick, & Ursano, 2009). With varying degrees of success, these treatments offer symptom reduction and condition stability. However, all too often, the combat-related PTSD patient laments that he or she is tired of trying a different pill or dose each month. Sensing a diminishing sense of personal control over the symptoms of PTSD, grief and depression often complicates the condition. After months, sometimes years, of medication and different counselors, the initial desire for a few months of intense treatment in order to make a rapid and deliberate recovery for the purpose of returning to the unit fit-to-fight begins to wane. Some are able to travel to inpatient programs that, in some cases, offer only more of the same interventions or less than what they received in the outpatient setting. This then further instills a growing sense of helplessness and hopelessness that they will ever return to their military unit, a source of self-satisfaction and identity few understand. Given these dynamics, there is a need for an added clinical approach that serves two important purposes: (1) to demystify the diagnostic label of “PTSD,” and (2) to provide a means of self-regulation on a continuum of peak performance rather than on the basis of a diagnostic pathology model—a language that military personnel respect and request. The military patient with PTSD is generally motivated by self and group reliance, and when offered a method of purposeful physiological self-management there is almost a 100% immediate buy-in and eagerness to begin. Not to mention the frequent statements of thanks that include, “I knew I was not crazy or making this stuff up!”

Through identifying abnormal electrophysiology biomarkers with preliminary assessments and an hour of time to explain the brain-body connection, to include some basic endocrinology, the process can begin to break down the negative mental health stigma that permeates military culture. This approach is well grounded in science and replicated around the world yet most clinicians have had no formal training and seem not to have reviewed the germane literature. The applied science is peripheral biofeedback and brain computer interface training.

The purpose of this article is to briefly highlight the added value of applied neuroscience assessment and treatment interventions that have a unique way of bridging the gap between stigmatized mental health approaches and the military acceptance of a self-motivated drive for total body fitness and resilience.

Assessment

There are several traditional assessment instruments available with ample literature support (e.g., Clinician-Administered PTSD Scale (CAPS), PTSD Checklist—Military Version (PCL-M), Posttraumatic Diagnostic Scale (PDS) that are helpful to quantify the symptom combination labeled PTSD. Beyond classification, these seem to have greater utility when paired with electrophysiology and biochemical measures, which makes perfect sense when the body is exposed to prolonged stress and horrific traumatic events. When the person with an elevated startle response, emotional numbing, mastoid and frontalis tension-related headaches, irritable bowel syndrome, neck and back pain, panic attacks, nightmares, inattention, and insomnia can see on a computer screen their own measure of central and autonomic nervous system biomarkers of these symptoms, there tends to be an “aha” moment that instills a sense of relief and control.

By way of an example, the computer-based PDS (Foa, 1997; Pearson Assessments) offers a simple graphic output (Figure 1). One benefit of this and other like tests is that it points out a degree of severity that military personnel grow to minimize, perhaps because they know someone that is worse and come to use their trauma-exposed peers as the measure of what is normal. For example, a distinguished Navy Corpsman of 16 years was about to be medically separated from active service due to symptoms that included angry outbursts at work, choking his wife during a nightmare, and low crawling and screaming on the bedroom floor in the middle of the night. Distraught over the prospect that his career would be cut short just 4 years from retirement, he still questioned the need for treatment. He commented that some of his peers were worse. It was the threat of divorce and loss of access to his children which motivated him to be seen for an evaluation.

Figure 1. Pre- and post-treatment PDS measure of a patient with combat related PTSD (Foa, et al. (1997; Pearson Clinical Assessment).
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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Military Combat PTSD
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When he was provided with a set of computer-based questions, followed by a printout of the relative severity of his condition, it became clearer to him that his symptoms warranted treatment (Figure 1). During the same visit, he received a quick set of electrophysiology measures (3-lead ECG, 19-channel qEEG, and ERPs). His condition was then solidified into a clear treatment plan. The combined results allowed for a physiological system-based explanation for his signs and symptoms. A review of the central and autonomic nervous system established a foundation from which he was able to grasp his condition in terms of human performance rather than what is often perceived as a nebulous “mental disorder” linked to personal failure. When basic biochemistry and endocrinology were then reviewed, his low testosterone, elevated cortisol, and other lab tests also made more sense. The collection of resting ECG statistics and EEG amplitude graphics most often have a pronounced positive impact on the person and stirs a desire to pursue nervous system health—a peak performance model rather than a mental disability model resonates with military patients.

ECG for HRV

Electrocardiograph (ECG) with 3-lead bipolar placement at wrist and forearm locations permits an easily measured R-wave from which heart rate variability statistics can be quickly obtained. Post artifact removal calculations such as the standard deviation of normal R-R intervals (SDNN) can provide a window into the degree of autonomic nervous system (ANS) balance (Figure 2). As a general guide, keeping in mind short interval recording and respiration confounds, a healthy SDNN range estimate of 65-150 is used along with resting heart rate and frequency domain calculations. It is often the case, with few exceptions (e.g., elevated high frequency power), that those with PTSD have a low SDNN and high relative power in the very low frequency range. Although 3-lead placement may be less sensitive than multi-lead monitoring the author suggests special attention be paid to the QT-interval, the indirect measure of ventricular repolarization, and if abnormal, there should be a following referral to cardiology (Drew, 2004). There is an inherent ethical responsibility when collecting human electrophysiology data to have familiarity with abnormalities and to make appropriate referrals to medical specialists. While this caution holds true in general, the clinician assessing those with PTSD should be aware that this population of patients is at significantly higher risk for heart diseases and sudden cardiac death (Boscarno, 2011; Ahmadi, 2011). Because coronary artery calcification is one of the leading risk factors, use of cholecalciferol supplementation for the often low D3 levels should be provided slowly, with lab values as a guide (25-hydroxyvitamin D3, calcium) and perhaps with supplemental K2 or alpha-lipoic acid (Rees, 2010; Kim, 2011).

EEG

The added measure of EEG with 19-channels or more offers additional insight into the person’s reported symptoms and can further direct individualized self-regulation interventions. In a medication-free military PTSD study with qEEG analysis, the authors found that diminished alpha1 (7.5-9.5 Hz) power and increased frontal and central beta1 (13.5-18Hz) were significant markers for combat exposed patients with PTSD compared to veterans without PTSD (Jokić-Begić & Dražen Begić, 2003). Beta2 was also reported to have been elevated (Begić’ D, 2001). The finding of alpha suppression in the left dorsolateral prefrontal cortex (DLPFC) thought to be a PTSD marker might also be due to hyperven-

A review of the central and autonomic nervous system established a foundation from which he was able to grasp his condition in terms of human performance rather than what is often perceived as a nebulous “mental disorder” linked to personal failure.

ERP

Event related potentials (ERPs) provide a rapid and relatively simple measure of information processing with very good temporal resolution. Varied test paradigms have been found useful to assess sensory abnormalities beginning with early components like the vertex P50 measure of auditory sensory gating (Erwin & Buchwald, 1986), positive and negative components around 100ms and 200ms (Shucard, McCabe, & Szymanski, 2008), and later components such that a cursory review of ERPs can be somewhat confusing. The P3 components associated with attention and complex memory processing are perhaps the most widely studied. When interpreting the P3 literature it is im-

Figure 2. Heart rate variability frequency and time domain (SDNN) measures of a 61 (left) and 22 (right) year-old with combat related PTSD. Frequency reference range: VLF (0.003-0.04 Hz), LF (0.04-0.15 Hz), HF (0.15-0.4 Hz).
Importantly, consider controls for age differences and to evaluate the test paradigm as a discrepancy between P3a and P3b amplitudes may, in part, have to do with the nature of the task (Katayama & Polich, 1998). Some use a standard oddball task (repeatedly presented NoGo stimulus with an unpredictable target or Go stimulus). Others use a continuous performance task, which offers a preparatory or warning stimulus followed by a second stimulus; depending upon the stimulus type, a decision to respond or withhold a response takes place. Varied tasks reported in the literature yield different measures of early sensory processing, later cognitive processing, or both. When thinking logically about what ERPs one might expect to see among those with combat PTSD, consider that the person adapts to the very physically and emotionally stressful environment sustained over many months. During this time, vigilant attention to subtle and seemingly irrelevant sound or visual stimulus is a mark of primary sensory cortex activation. Perhaps this early component deviation among those with PTSD is a result of sensory gating dysfunction resulting from cortical over activation and reduced inhibitory processes. Additionally, we often see an elevated P2 over activation and reduced inhibitory processes. Some may have a combination of symptoms and EEG results. There are enough differences that each case merits individualized sensor placement and frequency band selection. Some may have a combination of excess beta in the right DLPFC and elevated parietal and occipital alpha (11-13Hz) combined with deficient slower frequency alpha yet others may have a different pattern with elevated frontal theta. With respect to treatment frequency, we suggest a minimum of two sessions per week, three if possible (Smith, 2008). When these methods are applied the results are often quite positive.

A complex case example is a young Marine with PTSD and IED blast exposure, who was found to have midline vertex and occipital elevation of fast alpha peak frequency with lower slower alpha power. A pressure plate to ignite a bomb becomes a chronic life dependent skill. In fact, this is what researchers have found, frontal P3a amplitude is larger among those with PTSD when presented with novel distracter stimuli (Kimble, Kaloupek, Kaufman, & Deldin, 2000; Shucard, et al., 2008). It has been our observation with a Go/NoGo task that there are, at times, with combat PTSD cases, a larger N1 (Figure 6), a mark of primary sensory cortex activation. Perhaps this early component deviation among those with PTSD is a result of sensory gating mechanism dysfunction resulting from cortical over activation and reduced inhibitory processes. Additionally, we often see an elevated P2 that may suggest a cortical excitation for enhanced stimuli recognition (Paige, Reid, Allen, & Newton, 1990). Clinically, we observe and hear from patients that they attend to the most seemingly irrelevant sound or visual stimulus and that when even a subtle change occurs they notice it. This level of vigilance is exhausting.

**Figure 3.** Right frontal beta excess in PTSD with patient with emotional dysregulation

**Figure 4.** Occipital elevation of fast alpha peak frequency with lower slower alpha power

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**TREATMENT**

**ECG**

The assessment using ECG is useful to direct frequency and time domain training with paced diaphragmatic breathing. The intervention is offered in a graduated manner using abdominal and chest respiration sensors and either wrist ECG sensors or a blood volume pulse (BVP) sensor. Guided by skill acquisition, the respiration sensors are removed leaving just the BVP/ECG sensor feedback. Near the end of the training we remove the feedback screen and permit occasional viewing or access to just auditory feedback. This method is used in conjunction with instructions to practice the skill under real world rather than in-office conditions to further help achieve skill generalization. The clinician is reminded that paradoxical anxiety is not uncommon and should be reviewed prior to and during the procedure. Similarly, the mention of peripheral warming should be explained so it is not a surprise. These and other factors associated with ethical practice are of vital importance (Striefel, 2009). Another observation is that once per week training is inadequate. Rather, twice per week seems to be a minimum number of sessions that are enhanced by home practice with temperature sensors and diaphragmatic breathing or home-use devices. When learning a new skill such as playing an instrument it is understood that the 30-minute lesson once per week is followed by daily practice—we find that this same model applies with heart rate variability biofeedback training.

**EEG**

Similarly, the quantification of the EEG amplitude with spectral images and source localization software features can help explain certain symptoms and guide the application of brain computer interface training (Walker, 2009). Although there are some general consistencies among those with PTSD, such as elevated beta power and decreased low alpha band power (Jokić-begić & Dražen Begić, 2003), it is suggested that the location and frequency selections be guided by the combination of symptoms and EEG results. There are enough differences that each case merits individualized sensor placement and frequency band selection. Some may have a combination of excess beta in the right DLPFC and elevated parietal and occipital alpha (11-13Hz) combined with deficient slower frequency alpha yet others may have a different pattern with elevated frontal theta. With respect to treatment frequency, we suggest a minimum of two sessions per week, three if possible (Smith, 2008). When these methods are applied the results are often quite positive.

A complex case example is a young Marine with PTSD and IED blast exposure, who was found to have midline vertex and...
QEEG / TOPOGRAPHIC BRAIN MAPS:
Generalized Anxiety Disorder Subtypes

![Delta Theta Alpha Beta](image1)
High Beta Subtype: Anxiety, Insomnia, Alcohol / Drug Abuse

![Delta Theta Alpha Beta](image2)
High Alpha Subtype: Anxiety, Depression, ADD

![Delta Theta Alpha Beta](image3)
Low Alpha Subtype: Anxiety, Insomnia, Alcohol / Drug Abuse

![Delta Theta Alpha Beta](image4)
Cingulate Dysfunction: Anxiety, Rumination, Obsessive Compulsive Disorder

High Mean Frequency Beta: Anxiety, Alcoholism, Insomnia

High Mean Frequency Alpha: Anxiety, Insomnia

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B) Eyes Open Linked Ears Z-scores // Eyes Open Laplacian Z-scores

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Millicent Combat PTSD
Continued from page 14

Frontal slowing and right DLPFC beta excess. His symptoms included significant inattention, poor short-term memory, emotional regulation difficulty (sudden rage), headaches, dizziness, violent nightmares, and obsessive-compulsive symptoms of thinking about and needing to take objects that did not belong to him. After a medication-free electrophysiology assessment requested by his psychiatrist he was prescribed Effexor, Trazodone, Midrin, fish oil, vitamin D3, and Prazosin which provided some symptom relief. During the 5-month course of combined heart rate variability and qEEG and ERP guided brain computer interface, his treating physician was able to gradually remove the prescription medication and maintain essential fatty acid supplementation. After 30 sessions a repeat set of electrophysiology tests were provided (Figure 5). He reported a complete remission of symptoms. In the previously mentioned case of the Navy Corpsman there was also symptom remission after 8 months of combined heart rate variability biofeedback, brain computer interface training, slow reduction of psychotropic medications, and several integrated supplements (e.g., essential fatty acids, CoQ10, magnesium, GABA, acetyl-L-carnitine, phosphatidylserine, glycerophosphocholine, multi-vitamins with essential minerals, D3). His EEG amplitude and ERPs likewise improved to a comparison database (Tereshchenko, Ponomarev, Müller, & Kropotov, 2009) measure of normal (Figure 6).

Figure 5. Pre-treatment (left) and post-treatment (right) qEEG amplitude maps of military related PTSD with IED blast exposure injury.

Figure 6. EEG amplitude spectra (left) and event-related potential components to novel stimuli N1P2 (right): pre- and post-treatment comparisons (Note: calculated with Mitsar, Ltd. and HBImed software)

Conclusion

The complexity of the human stress response (e.g., glucocorticoid impact, hypocortisolism) and the variations of type of trauma, duration of trauma, pre-existing early childhood trauma neurobiological processes (Bremner & Randall, 1997; van der Kolk, 1994), and individual brain function or dysfunction differences make it difficult to assume a basic set of biomarkers and a one-size-fits-all treatment approach. The irony worth bearing in mind is that the very training and environmental conditioning that can keep a young Soldier, Airman, Sailor, or Marine alive in a war zone does not permit functional adaptation when he or she returns home. What appears to be somewhat lacking in the clinical approach to aid a post-deployment transition is that of deliberate and measurable self-regulation training. It may be the case that those exposed to combat who meet criteria for PTSD differ from those that do not based on the capacity to allocate attentional resources or the flexibility to differentiate between trauma-relevant stimuli and non-relevant stimuli. This cortical discriminant capacity, measureable with millisecond resolution of ERPs, might allow a measure and training target for improved combat resilience.

With the rapid international growth of neuroscience evidence, medical and non-medical clinicians are advised to expand their skills so that they might facilitate the addition of self-regulation skills for the person with combat PTSD. Though not discussed in this article, pituitary and adrenal gland functions and hormonal adaptations to chronic stress (i.e., daily exposure to 120 degree temperature, weeks without a shower, being shot at, and 15 hours of daily work) are also very important facets to an understanding and treatment of PTSD (van der Kolk, 1994). The military men and women that leave their families home and stand beside uniformed peers to engage a distant enemy intent on killing them are exposed to a level of stress few understand. So when they swallow their pride long enough to make a mental health appointment and show up early for help, the least we as a body of clinicians and researchers can do is bring our A-game, which should include an understanding of all current and potentially effective assessment and treatment methods with demonstrated success. With the growing numbers of those in need of treatment for PTSD there is ample room for multimodal management of combat PTSD.

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References


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Multivariate Proportional Neurofeedback and the Future of Live Z-Score Training

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

We have reported in the past on Live Z-Score Training (LZT) methods that provide Multivariate Proportional (MVP) variables for use in training. (Collura, Guan, Tarrant, Bailey, & Starr, 2010; Collura, Thatcher, Smith, Lamброс, and Stark, 2009; Collura, 2008a, 2008b, 2009; Smith, 2008; Stark, 2008). This report describes further progress in software technology that focuses on LZT dynamics, and real-time feedback. MVP variables are continuous, proportional values that are used in training in the same ways that conventional values such as absolute power, relative power, or raw coherence values, have been used in the past. The key to this approach is that the new MVP variables provide complex yet intuitively simple measurements that are field-proven in producing client results that are rapid, concise, and lasting. With the introduction of the Z-Plus series of software, we define a new area of LZT technology for training of 1, 2, or up to 19 channels.

Other approaches to using live z-scores typically produce only an “on/off” response, depending on whether one or more z-scores are within a range. Thus, the brain is provided with information that tells it whether or not it meets a condition, but does not provide any proportional or “how much” information to the trainee. This limits the brain’s ability to learn and respond to the important EEG parameters. Also, such methods do not lend themselves to tuning the training, beyond setting the target sizes. MVP methods produce new quantitative variables that are not simply “yes/no,” but provide real-time, proportional feedback that can be used for sounds, videos, games, or other feedback methods that respond to either “on/off,” “how much,” or a combination of such control variables. This provides a level of guidance that reduces the number and length of sessions necessary to see results. Currently, with the MVP variable “Percent Z OK” (PZOK), it is common to see visible EEG changes in 1 or 2 sessions, and to see results significantly faster and more specific than seen with conventional amplitude-based training.

Starting with the PZOK training method, we have developed a family of training variables that intuitively incorporate any or all of the z-scores and turn them into a single proportional variable. With these variables, any combination of channels, parameters (absolute power, relative power, power ratios, coherence, phase, asymmetry), or frequency components (e.g. delta, theta, etc.) can be trained. Regardless of the number of channels or parameters chosen, this variable always has the same meaning. It is the “percent of z-scores that are within the target limits.” It has a maximum value of 100 (100% normal) that continuously varies in time, and is useful both for training and for assessing the overall condition of the client. This method has been proven in over three years of field experience, and has been published in a variety of peer-reviewed journals, books, and industry publications.

Another key element of our approach is the ability to dynamically change the difficulty of the training on multiple levels, in real time, without interrupting training. This is analogous to being able to adjust the throttle, choke, etc. of a vehicle while it is in motion, which is an essential element of clinical application. With the PZOK method, clinicians commonly adjust the size of the training window and also the percentage of z-scores which are required to be met, in order to obtain a reward. This was a non-obvious, yet critical step in the evolution of practical LZT technology.

PZOK provides a uniquely flexible and powerful approach to adjusting training conditions, particularly in real-time. By alternating changes in either the target sizes or the percentage of z-score required, the clinician can adjust the difficulty level of the training, as well the distribution of the z-scores which are being trained. For example, requiring a large percentage of the z-scores to fit within a wide range emphasizes the “outliers,” while ignoring smaller z-scores. On the other hand, requiring a small percentage of the z-scores to fit within a narrow target can provide a “challenge” form of training that emphasizes mid-range values, while ignoring outliers. This latter method can, for example, leave the brain free to exhibit abnormalities that are compensating or coping mechanisms that persist, and allow the brain to formulate its own self-regulation strategy. The ability to ignore outliers is, at times, an important benefit. At other times, it is desirable to focus on outliers. The new metrics in the Z-Plus package address the outliers in new ways that increase the power and flexibility of PZOK training.

PZOK has been shown to have significant clinical value, and it can also be combined with other methods. A number of our protocols combine LZT training with “biased” training such as alpha up, theta down, or other types of protocols. The combined protocols provide the same simple feedback to the client, but also guide their brain in a particular direction desired by the clinician. All new Z-Plus-based designs can also be combined with traditional training, as the clinician sees fit.

Building on experience with the PZOK family of protocols, we re-examined the function and purpose of the LZT approach, and now introduce the Z-Plus extensions, designed to extend and reinforce the PZOK approach. Rather than changing or replacing the PZOK methods, the new software and displays provide additional information, flexibility, and direction for LZT training.

We first review PZOK in detail, and then introduce the new metrics, PZMO and PZME.

**PZOK: Percentage of all trained z-scores that fall within a given target range**

PZOK provides an overall assessment of “how normal” by counting how many of the z-scores fit within the desired target range. The exact position of the z-scores is not important, only whether or not they are within the target limits. PZOK is useful as a real-time training variable. The clinician sets the size of the targets, and also the percent of z-scores required to achieve reward, and the client learns when the PZOK value exceeds the percentage target. It was found important to allow the percentage target to go below 100%, in order to avoid simply training on “outliers” all the time.

**PZOK has the following behavior:**

- Minimum value: 0 (no z-scores are within range).
- Maximum value: 100 (all z-scores are within range).
- Intermediate values: 0–100 (what percentage of z-scores is within range).

**Limiting behavior:**

- PZOK with very small target limits (<0.5 SD) is less useful: PZOK becomes very small, even zero (no z-scores within range).
• PZOK with very large target limits (>3.0 SD) is less useful: PZOK will always be 100 with very wide limits (all z-scores within range).

**Strengths of PZOK:**

• With any percentage less than 100%, PZOK allows you to ignore outliers (allows for coping or compensating mechanisms).

• Adjustable target sizes to set difficulty of targets.

• Adjusts percent of targets that establishes total reward rate.

• Alternates between “challenge” and “easy” conditions for dynamic control of feedback, training of flexibility.

**Weaknesses of PZOK:**

• When targets are small, outliers are ignored, might deviate further.

• When targets are wide, inner values are ignored, even if they move toward abnormal.

• Only counts whether values are in range, does not analyze their size.

• Treats all z-scores the same, no weighting at this time.

• Requires attention to target limits, which should generally be adjusted.

**Z-Plus: The next generation of LZT training software**

When introduced, PZOK was met with a measure of disbelief, even disregard, by some in the industry, while it was adopted and studied by others. Nonetheless, over time, it has become clear that PZOK is uniquely capable of delivering meaningful and useful feedback in a wide range of client situations. The existing PZOK technology is entirely consistent with principles of operant conditioning, learning, and physiological adaptation. All that is special is that the information fed back (the operand) is a complex yet useful reflection of brain state. This new series of functions extends the intuition and usefulness of PZOK into new dimensions, the dimensions of Z-Plus.

Based on our experience and analysis, we now introduce two new families of metrics, plus additional displays, combined into the Z-Plus software option. Z-Plus is entirely consistent with, and extends, the existing LZT software, designs, and methods that have been proven over the last five years. Like PZOK, the new functions are also accessible as UL versions that use different upper and lower limits. The new functions are incorporated using the Event Wizard, and no new control panels or settings are required. This provides complete flexibility in how they are used, and does not require the clinician to stop using PZOK, or to choose between methods. All metrics are always available, and protocols can be designed as desired combining old with new, as desired.

As will be seen below, one interesting aspect of the new metrics is that, while they are useful with various target sizes, they are particularly useful with very small, even zero, target sizes. When target size is zero, the new metrics incorporate all z-scores into the calculation, providing true indicators of total system state and state change, and no z-scores will be ignored. This provides the ability to account for both outliers and intermediate z-scores, without ignoring any z-scores.

**PZMO: Percentage of Z-Score Movement (PZ Motive)**

PZMO is an outgrowth of the PZOK approach, and is an aggregate statistic reflecting change in the outlying z-scores. PZOK tells how many z-scores are within the target range, as a percentage. We usually use a percentage of between 50% and 80%, which means that a substantial portion of the z-scores are outliers.

As a dynamical systems approach, this gives the brain flexibility to choose which z-scores to normalize, and which to leave as outliers. PZMO is the aggregate momentum of these outliers. It is a measure of their net motion, and is a dynamic systems concept. Think of the z-scores as having a life of their own, having mass and velocity. PZMO measures the group momentum, and tells you what percentage of the net motion is toward the target range. PZMO is generally below zero, as nothing is moving particularly toward the targets in general. However, when PZMO goes positive, it tells you the net positive movement.

A value of 5% for PZMO is significant. It means that in the last instant, there was 5% net motion toward the targets. That is a very big deal. This is therefore a derivative measure that tells your client that at that moment, the outliers moved inward. We typically see only a few PZMO reward beeps every few seconds, so it is an added reward. It is like giving the brain a “gold star” when it has particularly good improvement that moment in time. In our view, it has a similar effect on the brain as the effect the derivatives market had on Wall Street. Small changes can have huge effects, and major learning processes become possible.

PZMO provides an overall assessment of the instantaneous movement (change) of all z-scores that are outside the specified range. Z-scores that are within the target range are ignored. PZMO uses concepts from physics to introduce the idea of “momentum” of the z-scores, which reflects their “velocity, direction,” and also a weighting factor suggesting their “mass.” It is not necessary to weight all z-scores the same. With PZMO, it is possible to weight different z-scores differently, providing an additional dimension of flexibility and control. PZMO is a z-score motivator and reflects the net z-score motion. PZMO takes into account not just the direction (towards or away from normal) but also the amount of movement (a little or a lot) and the weight of each z-score (lightweight versus heavy). PZMO can be positive or negative, and reflects the total change in momentum of all z-scores. When it is positive, then the net movement of all z-scores outside the target range is inward, toward normal. When it is negative, then the net movement of the outlying z-scores is outward, away from normal. Thus, PZMO provides an instantaneous indicator of the change in the z-scores, indicating the brain’s immediate tendency toward normalization or toward dysregulation. Technically, PZMO is the instantaneous change in the total momentum of the system, as defined in physics.

PZMO is intended to be used in addition to PZOK. Existing protocols do not have to be changed, only extended (with a single Event Wizard event) to incorporate the PZMO data. Typically, when PZMO is above some positive threshold, the client will receive a bell, tone, or other reward. This provides an additional, highly dynamic reward (think of it as a gold star) when the client moves in the right direction.

PZMO incorporates useful and intuitive concepts from astronomy, celestial mechanics, in particular. The client is learning about their gravitational potential which is the tendency toward normalization. The training limit region is like a star, and the outlying z-scores are like planets. Ideally, z-scores tend to move inward, to be captured by the sun. If all planets are in the sun, then all z-scores are within range, and the client’s EEG is deemed normal. If a client can increase their potential, then z-scores will normalize more directly and consistently. The training limits...
define a capture area similar to the event horizon of a black hole. Once z-scores go inside the boundaries, they disappear (are ignored). Only the z-scores moving outside the boundaries (the orbiting z-scores, if you will) are incorporated into PZMO. Thus, PZMO captures the tendency for z-scores (planets) to move toward, not away from, their sun. This puts the training into a highly visual and dynamic context. This informs the clinician as well as the client, as to what is happening and to what extent, in the complex dynamic “z-solar system” of the brain.

PZMO does not provide an overall assessment of “how normal” in the way that PZOK does. If all z-scores are within the target range and none are outside, then there is no net movement to reflect, and PZMO will be zero. At that point, PZOK would be 100. Thus, PZMO gives a rapid, intuitive indication of the direction of change, and has higher resolution and responsiveness than PZOK. As an analogy, it is somewhat like adding a tachometer, or actually an accelerometer, to a car dashboard, so that you can see how rapidly, and in what direction, your velocity is changing. It is also like a dieter monitoring the change in their weight every day, as an indication of how the diet is working. PZMO introduces the idea that z-scores closer to normal have lower “potential energy,” and that the client’s brain has a natural tendency to normalize. The normal brain is a “rest state” toward which the brain should naturally move. Abnormalities require the brain to expend energy, and can be normalized as the brain relaxes, and brain dynamics settle into an optimal state.

PZMO can be thought of as conveying motion, movement, momentum, or related concepts to LZT training. It introduces concepts that derive from physics, including gravity, velocity, acceleration, and dynamic behavior. Using PZMO, the practitioner can begin to think of z-scores as objects that have mass, direction, even intention. The intuitive view of PZMO is that if it is 100, then that is the maximum inward movement and thus, all the outlying z-scores have just moved inside the target limits. If PZMO is zero, then there is no net movement, that is, there is just as much inward movement as outward movement. If PZMO is negative, then the z-scores are, in general, moving outward. For example, if a client clenches their teeth, PZMO will immediately become a very large negative number. When they relax, it will become a very large positive number. In the long run, if there is net improvement, PZMO will be positive more often than it is negative. The client should get a reward when PZMO is sufficiently positive; for example, say above 10, which would mean that the net motion of the outliers is to move 10 percent of the distance towards normal. PZMO will not generally be positive all the time, as the z-scores in their typical patterns of movement simply cannot always be moving towards normal all the time.

PZMO emphasizes variability and dynamic change. It is analogous to a financial derivative that focuses on the change of a system, not simply its current state. As such, it has the potential to leverage LZR training by providing highly accurate information relating to dynamic change, and delivering it to the client. Again, PZMO is not intended to replace PZOK; it is intended to be used as a supplemental training or assessment variable. If the client receives an extra reward every time there is a significant inward movement, then they will learn that skill as well, and tend to reinforce the process of normalization, not just the state of being “more normal.”

As an example of the use of PZMO, one might use the following Event:

If “x=PZMO();” IS GREATER THAN 10 THEN (play wav file)
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This event would allow the user to hear a beep every time they achieved a 10% movement toward normal during the session. They would hear the reward whenever the z-scores had significant improvement, even if PZOK was not yet above the target percentage. This, consequently, rewards improvement in the right direction, regardless of the current state. This motivating feedback is a significant addition to watching the PZOK variable rise and fall; it allows the client to know when they are moving in the right direction.

PZMO has the following behavior:

- Minimum value: negative value, unlimited (z-scores are moving outward)
- Maximum value: 100 (all z-scores have just moved within the target range)
- Intermediate values: typically -100 to +100: (what is the overall motion toward or away from normal)

Limiting behavior:

- PZMO with very small or zero target limits: useful, it simply incorporates all z-scores into the metric.
- PZMO with very large target limits: not useful: PZMO would also be zero, as all z-scores would be ignored.
- PZMOL: provides PZMO for all upper z-scores, i.e. those above upper target limit
- PZMOL: provides PZMO for all lower z-scores, i.e. those below lower target limit

Strengths of PZMO:

- Capable of reflecting all z-scores (with target size of zero)
- Reflects dynamic change in the training process
- Consistent with existing PZOK approaches
- Provision for giving different weights to different types of z-scores

Weaknesses of PZMO:

PZMO can become large in the presence of artifact, producing feedback when it is not desired. This is because, as the z-scores normalize when the artifact reduces, PZMO “sees” a lot of improvement! But it is improvement from an abnormally noisy situation, hence is not really to be rewarded. To manage this, designs should include both PZOK and PZMO in the reward mechanism. When artifact is present, PZOK will fall rapidly, thus inhibiting feedback.

PZME: PZ MEAN OR PZ MEASURE

PZME is a measure of the mean distance of the outliers from the zero point. It is a measure of the global size of the scattering of outliers in the collection of z-scores. As it moves lower, the outliers are moving closer to the targets. We mostly use this as a long-term statistic throughout the session, watching for a small change, say from 2.5 to 2.2, over the session. PZME provides a measure of the mean size of all z-scores that are outside the target range. For every z-score considered, its distance from zero (normal) is computed, and these are combined into population mean (average). This provides a simple assessment of how abnormal all z-scores are as a group. Different types of z-scores can be given different weights, if desired. PZME is intended to be used primarily as an indicator of overall improvement, but can also be used for training. Training PZME (downward) would conform to the naive principle of simply “training everything toward normal,” and is conceptually a step backwards, yet is still an important new capability.

The interpretation of PZME is simple. If it has a value of 1.7, for example, then the average size of all the z-scores is simply 1.7. Direction is taken into account, so that z-scores above the range are treated the same as z-scores below the target range. There is also a separate function to get the average z-scores in the positive direction and in the negative direction. Technically, PZME is the mean error as defined by statistics. In the solar system analogy, PZME is the average distance of all the planets, hence reflects the overall size of the client’s z-score solar system. Generally, a smaller solar system is preferable to a larger one.

PZME is intended to be used as an indicator, to see progress within and across sessions. It provides a single number that has a very clear and simple interpretation. It may, for example, be useful in assessing the overall progress, and whether to terminate training. For example, when clients tire, z-scores sometimes are seen to lose their tendency to be improving. If PZME shows an increase for more than 3 or 5 minutes, for example, then the client is moving in the wrong direction, and training should be re-evaluated.

PZME also has the potential for use in creating target limits for LZT training. By providing an instantaneous measure of the average length of all z-scores across the board, PZME provides a basis for adjusting target limits for training. While the use of autothresholding is controversial and may or may not be desired in a particular case, PZME provides an objective, sound approach to creating target thresholds that are based on the instantaneous state of the desired z-scores.

For example, the following Event Wizard expression: x=PZOKUL(PZMEU(0), PZMEL(0)); would automatically train PZOK using the average of all positive z-scores as the upper target limit, and the average of all negative z-scores as the lower target limit.

Ironically, PZME is what some thought we were doing with PZOK, when they believed that we were simply “training them all together.” The simplest approach to combining live z-scores would be to add them together (using absolute value) to get a single number. With PZME, we have decided to provide just that, a simple, total assessment of how all the z-scores add up. We leave it to clinical and research progress to determine the utility of PZME for training, control, or for assessment. Intuitively, and from our experience, if trained z-scores are seen to visibly move toward normal, then the PZME variable would also have to go down in a uniform fashion. PZME simply provides a number that can be used to estimate the total instantaneous condition of all z-scores, treated as a whole.

PZME has the following behavior:

- Minimum value: zero (all z-scores are exactly normal)
- Maximum value: unlimited, but typically will not reach as high as 3.0 (if z-scores are very abnormal)
- Intermediate values: typically 0 to 2.0: (the average size of all z-scores)

Limiting behavior:

- PZME with very small or zero target limits: useful, it simply incorporates all z-scores into the metric.
- PZME with very large target limits: not useful: PZME would also be zero, as all z-scores would be ignored.
- PZMEU: provides PZME for all upper z-scores, i.e. those above upper target limit
- PZMEL: provides PZME for all lower z-scores, i.e. those below lower target limit

**Strengths of PZME:**
- Extremely simple and intuitive
- Capable of reflecting all z-scores (with target size of zero)
- Reflects total state of the brain
- Consistent with existing PZOK approaches
- Provision for giving different weights to different types of z-scores
- Can be used to develop targets, i.e. autothresholding for LZT

**Weaknesses of PZME:**
- Moves very slowly, useful for monitoring, less useful for training

In brief, PZOK only knows the percentage inside the target range, it does not know about the outliers, except that they must be out there somewhere. PZMO tells you the net motion of the outliers at any instant. PZME tells you how far out they are in general. While PZMO is a very fast, derivative measure, PZME is a very slow, aggregate measure. It all feeds into a view of the brain and the z-scores as comprising a dynamic system that can determine its own rules for self-regulation if you give it the right information.

Thus, this approach, which we call “Z-Plus,” gives you more than one type of information. There are various ways to use PZMO, but usually people give a reward when it jumps something above zero, indicating net motion toward the targets.

**Z-Bars**
A new display called Z-Bars shows all z-scores as bars with dynamic lines that show short-term changes. It is interesting to note that, when the instantaneous variations are visible, one live z-score that has essentially no variation is also the most deviant (negative) one. This indicates that this value is stuck and cannot change. This underlies the importance of LZT training as flexibility training, not just to make large things small or vice versa. Stark (2008) described momentary changes visible in LZT text displays, and this approach now makes these changes visible as instantaneous graphic displays. The MVP approach allows the brain to let the z-scores vary, and to become aware of their movement. When the deviant z-score breaks loose and can become variable, that is the key to training, not just a change in value.

**Z-Maps**
In order to provide further access to visualizing brain dynamics, we have now introduced Z-Maps. These are live maps of the Z-Scores that can be used for training or for following training progress.

We provide two types of maps. Instantaneous maps show the moment-to-moment changes, and can change rapidly. We also offer damped maps, which show the damped z-score, which is what is also used in the text display. This provides a more stable map for viewing the effect of changing the target size. The training parameters change in the expected way as the targets are widened.

Live data from actual training, showing PZMO and PZME reflecting the training effects.

All z-scores as bars with dynamic lines that show short-term changes.

As many z-scores as are being trained can be seen. This panel shows 192 coherence z-scores from 19 channels.
Simultaneous Z-Bars and Z-Maps

With this new set of tools, it is possible to visualize and train new metrics and displays that are responsive to brain dynamics. The field of QEEG has moved from being focused on assessment and static variables, to the realm of dynamics and real-time, providing new avenues for operant training and brain conditioning.

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

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Doc, it Really Rang My Bell...

David Trudeau, MD

Editor’s note: Over the past decade, clinicians treating returning combat veterans of Iraq and Afghanistan have been confronted with a large cohort of servicemen and women who have been exposed to both psychological and biomechanical trauma to the brain. In a pioneering 1998 paper, Dr. David Trudeau was among the first to report that veterans with history of mild brain injury due to blast exposure were at increased risk for development of PTSD, an observation now supported by neuroimaging studies which have consistently identified an overlap in the neurocortical networks implicated in the two disorders (Stein and McAllister 2009). Dr. Trudeau discusses the background to this seminal work with our readers....

In 1995, while working with PTSD vets at the Minneapolis Veteran’s Affairs Medical Center, I chanced upon a discovery that was a bit before its time—a relationship between combat blast trauma exposure and enduring problems with cognition and PTSD symptoms. I was attempting to replicate Gene Peniston’s well-known work on alpha-theta feedback in combat PTSD. Although my background and expertise was in addiction medicine, I made my way to the PTSD program because that’s where the neurofeedback equipment resided at the time, and worked out a deal with Steve Barton, MD who headed the PTSD program at the time, to use his Lexicor 24 and Capscan, providing some of my work was with veterans in the PTSD program.

While in process of doing alpha-theta sessions on a number of vets with chronic PTSD, I kept hearing a similar repeated story from vets who had exposure to blasts that resulted in brief unconsciousness, ringing ears, and a feeling of being dazed—often for hours. “Doc, when that grenade went off that killed the man next to me it really rang my bell—I haven’t been right since.” Many seemed to date the onset of their troubles to an event where they were exposed to blast and concussed mildly, and because there were no other injuries, resumed duty. Many times those closer to the blast were killed or severely injured. These fellows were the “lucky ones,” walking away without a scratch but dazed and amnestic and confused.

I looked at quantitative EEGs on these men, and although I couldn’t really come up with anything common by inspection or analysis, I did Thatcher’s discriminant function for mTBI and saw that these blast survivors scored in the mTBI range. Of course, this did not prove anything as the mTBI discriminant can be influenced by many things and is not in itself diagnostic. PTSD is fraught with comorbidities and any of them might be influencing these results. I went to the literature and could find little to help me. There were anecdotal accounts of “shell shock” in World War I trench warfare. There were storied accounts of John Paul Jones-era sailors becoming dazed with repeated cannon blasts on the gunnery decks in old time naval battles where tall-masted men o’war traded broadside salvos.

The literature I could find on brain damage from blast exposure, however, was confined exclusively to underwater explosions, where the percussive shock waves in an inelastic medium (water) went straight to the brain without the cushioning and dampening effect of air propagation. Interestingly, the studies on animals to produce the contra-coup type of injuries incurred in sudden deceleration injuries (read: car crashes) were problematic because only primates had the necessary cranial vault configuration and head bobble to produce anything similar to human “whiplash” micro brain trauma. The animals

Figure 1.
Overlap of symptoms associated with mild traumatic brain injury (mTBI) and post-traumatic stress disorder (PTSD)
Symptoms that are common to mTBI and PTSD include impaired concentration, tension headaches, mental slowness, and indecision. Distinct features of PTSD include intrusive flashbacks, recurrent nightmares, hyperarousal, and social avoidance. Distinct features of TBI include neurological deficits, seizures, and headaches with migraine features. 
Figures reprinted with permission.

Figure 2.
Traumatic brain injury (TBI) may influence the development of post-traumatic stress disorder (PTSD) following a traumatic event. Following a traumatic event, several factors influence whether an individual develops PTSD. TBI may influence the likelihood of an individual developing PTSD following a traumatic event by compromising the ability of an individual to psychologically adjust to a traumatic event. While combat mild TBI appears to increase the likelihood of PTSD developing, severe TBI may reduce the likelihood of PTSD developing.

Continued on page 27
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were strapped into a high-speed sled apparatus and exposed to deceleration. However, they lacked the anatomic frontal lobe hypertrophy of humans and the force necessary to produce corresponding brain lesions of micro axonal disruption. The successful model that emerged for studying deceleration micro trauma in animals was a simple apparatus that passed a shock wave from a piston pulse to a saline filled syringe via a tube inserted through a tiny hole in the calvarium communicating with the cerebral spinal fluid space. This could be much more easily applied to rodents, and was way less expensive.

So, I thought, there seems to be some connection between the possibility of air distributed blast effects on the brain and the type of injury thought to occur to the brain in deceleration accidents, and it might be fair to compare combat blast survivors with automobile accident victims. However, if Thatcher’s mTBI discriminant—which of course was derived from automobile accident survivors who had mTBI. I was also impressed that many of these blast survivor veterans complained of attention problems, so began to test them with T.O.V.A and an assessment of symptoms associated with ADHD, and even though they had no childhood ADHD, they often could meet criteria for acquired ADHD.

I began a systematic study of two populations of chronic combat PTSD veterans who were similar in all respects save for one—a history of combat-related mild blast concussion that was not recognized or treated as an injury at the time. The veterans in the study came from WWII, Korea, and Vietnam. There were no veterans from Desert Storm, probably because there were very few at our VA at that time—and I wonder if it takes many years before veterans come forth with chronic PTSD. We eliminated people with other significant head trauma, including mTBI, as well as those with active substance use disorder and additional psychiatric diagnoses other than concurrent depressive disorder. We found that by comparing chronic PTSD vets with mild blast concussion to those without, there were statistically significant differences in both subjective symptoms and objective performance on tests of attention and concentration as well as in qEEG mTBI discriminant function scores. And, this effect seemed to persist after many years. What resulted was the first study on combat blast mediated mild concussion—presented at ISNR in 1996—and published in 1998. It got little attention at first, but in the last seven years or so I have had many requests for reprints.

With the experiences of the last eleven years in blast exposure TBI from IEDs in Iraq and Afghanistan, many more investigations have been published. It is the bane of returning warriors, whether or not they have other serious injuries. Little is yet known about how to prevent and treat this type of debilitating brain injury and to parse it from PTSD and depression and other disorders. Little is known about its chronicity and progression. From my experiences with combat veterans, I believe it is highly likely that a brain flooded with excitatory neurotransmitters from chronic war stress, that is then subject to mechanical disruption of microarchitecture and ensuing inflammatory cascades, will sustain long term damage, and that this damage is an important component of what is called post combat traumatic stress disorder. The bottom line is this: developing strategies to diagnose and treat the organic insult of combat blast injury is one of the most important contributions neurofeedback can make to help returning warriors be whole again.

References:

Five Case Studies Using Live Z-Score Training Percent-Z OK on Individuals Diagnosed with PTSD

Penijeuan Rutter, LMHC, BCN

Introduction
Recent advances in technology are changing the field of neurotherapy by offering clinicians information-gathering and feedback-producing paradigms that are more sophisticated than anything previously available. Using neurofeedback as an adjunctive therapy to promote relaxation and rebalance poorly regulated EEG mechanisms is growing in popularity, with the increasing ability of practitioners to scientifically demonstrate changes in EEG activational patterns that appear to correlate with clinical improvements. Emerging tools such as live channel EEG recordings now equip clinicians with the ability to create not only measurements of brain activity before and after neurofeedback interventions (Thatcher 1999), but can also be the basis for complex and intelligent feedback that takes into account how individual regions function within the global context of the entire brain.

The therapeutic relationship between posttraumatic stress disorder (PTSD) and neurofeedback can be traced back over forty years due to the efforts of researchers such as Kamiya (Kamiya, 1970), Peniston and Kulkosky (Peniston, 1991), to name a few, and has often been comprised of giving the brain feedback designed to increase deficient alpha and theta rhythms, and decrease excess beta activity at a few selected 10-20 sites. These interventions were devised to be sensitive to 2-5 qEEG variables such as amplitude asymmetry, coherence and phase. This allowed practitioners to provide feedback to clients based on not only what was happening at each individual site being observed, but also the dynamic relationships between the recorded sites (Collura, 2010).

In 2007, Collura and colleagues released an innovative software program that introduced an entirely new paradigm into the field of neurofeedback (Collura, 2009), and named it live Z-score training (LZT). Not only did LZT track the power and amplitude measures of the EEG at each site being recorded, but it also tracked connectivity parameters, such as amplitude asymmetry, coherence and phase. This allowed practitioners to provide feedback to clients based on not only what was happening at each individual site being observed, but also the dynamic relationships between the recorded sites (Collura, 2010).

Initially, this was only possible between four sites at a time, but as the capabilities of the software have continued to evolve, it is currently possible to provide feedback to a brain system based on 19 channels of live EEG. This means that a practitioner can now create training protocols based on global brain activity, and even if they choose to focus on specific brain regions, the feedback given to the client can still be delivered in the context of how other areas in the brain are functioning and responding to the changes in one particular location.

Implications for PTSD Treatment
The revolutionary neural integration capabilities of LZT are particularly relevant to a population suffering from PTSD symptoms, not the least of which are difficulties with cognitive processing, memory integration, and regulating arousal responses to situational stressors. One of the current paradigms of treatment for PTSD is Eye Movement Desensitization and Reprocessing (EMDR). While research on EMDR has resulted in mixed reviews (Davidson, 2001), one of the theories held by some EMDR providers is that the physiological tasks often involved in EMDR sessions, such as lateral eye movements, alternate hand tapping, and bi-lateral auditory tones, create an interhemispheric activation in which the brain assists the individual to process and integrate experiences (Levin, 1999).

Hippocampi, areas in the brain associated with storing and retrieving memories, and regulating fear responses, are often implicated as key regions affected by PTSD (Gilbertson 2002), while the frontal cortex, the basal ganglia and the parietal lobes are all involved in cognitive processing, attention, and an individual’s sense of time (Kolassa, 2007). Functional neuroimaging studies support the hypothesis that poor communication between these brain regions play a role in exacerbating symptoms of PTSD, such as flashbacks, intrusive thoughts and sensations, recurring dreams and panic attacks (Stein & McAllister, 2009).

Case Histories
The following five cases are individuals who were diagnosed by medical professionals as meeting the DSM-IV criteria for PTSD. A qEEG brain map was performed on each person before neurofeedback was introduced, and additional qEEG brain maps were acquired after a number of sessions of neurofeedback were completed.

In Figure 1, the subject is a 44 year-old female with a history of early childhood abuse and trauma. The map on the left was taken before any neurofeedback was done. The map on the right was taken after 40 sessions of LZT using a Percent-Z OK protocol unique to BrainMaster software. All sessions were performed using four channels of feedback, placed at F3, F4, P3, and P4. The placements were chosen to optimize integration and increased communication between brain areas commonly found to be affected in individuals exhibiting symptoms of PTSD, as previously discussed in this article.

Before neurofeedback, she reported difficulty functioning socially and professionally, and described an inability to form healthy romantic relationships. After completing 40 sessions, she said that she felt “100% better” and “like a different person”, reporting a significant decrease in her symptoms, particularly the avoidance and numbing behaviors that drove her interpersonal frustrations.

Figure 2 contains the brain maps of a 19-year-old male from a broken family who had severe PTSD symptoms, a history of chronic substance abuse and traumatic brain injury. He had dropped out of high school, could not maintain consistent employment, and attempted suicide by slitting open his own throat with a kitchen knife. He spent six months in an inpatient facility before he started neurofeedback. The map on the left is pre-intervention, and the map on the right is after 30 sessions of four-channel LZT neurofeedback, site placements at F3, F4, P3, and P4, using the Percent-Z OK protocol. In the five years since these sessions, he has held a steady job for the first time in his life, enrolled in school and married a lovely girl. He credits the neurofeedback with having “saved his life”.

These first two cases were individuals who received LZT neurofeedback in 2008 when only four channels of training, using up to 248 variables, were available. The observable changes in the EEG and the reports of clinical improvements were welcome, however the idea of being able to achieve positive clinical results with fewer sessions was appealing. When BrainMaster Technologies released an additional software option that allowed practitioners to record and provide feedback using up to 19 channels of EEG, with 5700 training variables to choose from,
the next step was to incorporate additional training channels to see if there were discernible clinical improvements in fewer sessions using more training sites.

While early forays into training individuals using the full 19 sites yielded promising data, the idea of connecting 19 leads to a client every session was proving daunting to the everyday practitioner. In an effort to create an interim option, the idea of attaching nine training leads was explored, and the following two cases (Figures 3 and 4) are images of pre- and post-EEG recordings that use the LORETA algorithm to project activity in deeper brain structures and assess how feedback from scalp EEG recordings can affect limbic regulation. The images were generated from 19 channel EEG recordings, and analyzed using norms from the NeuroGuide 2.6.9 database.

In Figure 3, the subject is a 24 year-old male diagnosed with PTSD who presented with specific triggers related to social interactions. He reported hyper reactivity and irritability, outbursts of anger and volatility, and general difficulty behaving appropriately within his environmental context. The image on the left is prior to neurotherapy, and shows activity 2.98 standard deviations above the norm at 19 Hz in the cingulate gyrus, most notably Brodmann area 24, a region in the anterior part of the cingulate gyrus which is involved with emotional and cognitive processing (Critchley, 2005).

The image on the right of Figure 3 is after five sessions of LZT Percent-Z OK feedback using nine training sites: F3, Fz, F4, C3, Cz, C4, P3, Pz, and P4. The design was intended to address integration of frontal, central and parietal areas. The subject verbalized that he noticed after the first couple sessions he felt “calmer and clearer”, and after five sessions he found it easier to interact socially, saying that he “didn’t get as angry and impatient, and thought less about violent acts while talking to people”. The 19 Hz activity in the cingulate gyrus reduced from 2.98 standard deviations above the norm to 0.6, as can be observed in the panel on the right hand side of Figure 3.

The fourth case underwent an identical regimen as case number three, which consisted of LZT Percent-Z OK feedback at the same nine central 10-20 sites for five sessions. Case number four is a 29 year-old female who presented with a history of chronically abusive relationships that resulted in her experiencing hypersomnia, generalized anxiety and avoidance, motoric apathy and indifference to daily activities of living, including showering, eating, or even getting out of bed. In her pre-intervention LORETA image, she exhibits activity 2.96 standard deviations above norm at 26 Hz in the cingulate gyrus in the region of Brodmann area 23, a location which is correlated with limbic associational integration (Lane, 1998).
She reported feeling more relaxed and energetic after her first session, and over the course of the five sessions said that she was in a “happier state, and was finding it easier to wake up in the morning and get moving. She also verbalized that she was thinking more clearly and was less overwhelmed with day-to-day activities. In her post-intervention LORETA image, seen in the right panel of Figure 4, the observed cingulate activation that was previously at 2.96 standard deviations above the norm was reduced to 1.6.

The final case subject, a 27 year-old female, came from a severely abusive family of origin, exhibiting the most extreme symptoms of PTSD out of all five cases discussed in this article. She had a history of insomnia, chronic nightmares and flashbacks, emotional rigidity, physiological reactivity and startle response, multiple panic attacks a week, hyperventilation and self-mutilating behaviors. She was also given LZT Percent-Z OK feedback for five sessions at the same sites as subjects three and four: F3, Fz, F4, C3, Cz, C4, P3, Pz, and P4.

She reported she felt relaxed halfway through the first training session, after which she slept through the night for first time in 16 months, described reduced symptoms of anxiety, a cessation in self-mutilating behaviors after third session, a noticeable reduction in number of panic attacks and flash backs, and more emotional and social flexibility by the fifth session. Her before and after qEEG maps can be seen in Figure 5.

**Discussion**

A review of the data presented in this article indicates a positive outlook regarding the use of live Z-score training as an adjunctive method to assist individuals recovering from traumatic experiences. Reports of symptomatic reduction after LZT neurofeedback sessions point toward the potential benefits of further investigation into the clinical utility of a training approach that includes both connectivity measures and external referential parameters in the informational paradigm presented to the brain during feedback.

The first two cases used four channels of EEG to inform the training paradigm and the brain, and required 40 and 30 sessions respectively, to make the observed changes in brain activity seen in the before and after qEEG maps in Figures 1 and 2. The last three cases used nine channels of EEG and clients were beginning to notice results within the first two sessions, in contrast to the first few cases, in which clients reported beginning to notice results between 12 and 20 sessions. Preliminary case studies using the full 19 channels simultaneously to inform the feedback paradigm have also yielded rapid changes in symptomatic presentation within a few sessions.

A limitation of this study is potential reproducibility using Z-score training approaches created by other software developers. The Percent-Z OK protocol used in these cases utilizes an algorithm that regulates feedback by allowing the brain to meet a percentage of the required criterion by interacting organically with the variables being tracked and designing an individualized protocol that is updated in real time using continuous proportional feedback. This combination of features is currently not available in other software packages offering Z-score training options, and additional studies will be needed to make a valid comparison of results between the differing approaches.

As with any powerful intervention, clinicians using LZT neurofeedback need to exercise caution when working with a population that suffers from post-traumatic stress disorder. Individuals under the care of a neurofeedback provider will also potentially need additional emotional and cognitive support to help process experiences and memories as neural integration takes place. Clients described in the present paper received appropriate amounts of cognitive behavioral therapy embedded in the neurofeedback sessions provided by this clinician. If not trained in such strategies, it is the responsibility of the neurofeedback provider to assist the individuals receiving neurofeedback in finding resources that can offer the necessary support.

It is an incredible time to be working in neurofeedback, with many brilliant and talented individuals making contributions to the field that are changing the face of healthcare forever. Using a solid scientific approach, supported by data and quality standards of care, neurofeedback providers will continue to assist many individuals suffering from not only PTSD, but also a variety of other conditions that can be positively affected with improved neural connectivity, systemic relaxation and self-regulation.

**References**


Remediation of PTSD using Infra-Low Frequency Neurofeedback continued from page 10
done, as well as from the conventional understanding of the process.

**A PTSD Case Report**

With this brief description of the process, we turn to an actual case report. The training history of a veteran with PTSD is illustrated in Figure 1, where symptom-tracking data are shown for the dominant complaints. This case was chosen over others for two reasons. First, this individual had a larger than usual list of significant complaints, and secondly the training was done in our own office, so we have familiarity with this person beyond the data. In this case as in all others, a wide-ranging initial clinical interview established the critical issues for this person. These are quantified here in terms of severity on a Likert scale of 0-10. (The initial symptom list included several hundred items, of which some 60 are commonly reported in PTSD. Twenty-five of the more significant symptoms in this case are shown in the illustration.)

The impression given by the results of brain training over time is one of joint resolution across the seemingly disparate symptom categories. The similarity in learning curves is more clearly reflected in Figure 2, which just re-plots the same data. It is noteworthy that this list includes such varied symptoms as depression, bruxism, migraine, irritable bowel, addictive behaviors, and chronic constipation. All of the symptoms move toward clinical insignificance at a similar rate over the period of forty training sessions. This pattern is a common observation within the entire data set of over 400 cases.

**PTSD in the Dysregulation Model**

This striking commonality in learning curves across the board allows us to reframe the issue of PTSD as a disorder of dysregulation. Resolve the dysregulation and the symptoms collectively diminish to clinical insignificance. If this kind of reframing is supported by the collective data set, then it is appropriate to shift attention to the dysregulation status per se, and attempt to resolve that by whatever means are at our disposal, using primarily the methods of applied psychophysiology. With the adoption of this perspective, all symptoms of dysregulation become criteria by which we judge our success in restoring self-regulatory integrity, quite irrespective of whether or not these are regarded as classic or defining symptoms of PTSD.

Regarding PTSD as primarily a psychological condition has been an understandable but unfortunate misdirection. This is not to say that traditional psychological approaches are entirely irrelevant to the resolution of PTSD. Rather, it is to argue that these play only a supportive role, particularly in the early phases of recovery. Much of the success of exposure therapies is also traceable to physiological normalization, an objective which can now be achieved much more readily and with less drama with psychophysiological techniques. Once the physiology has been stabilized, there is much still to be done with psychotherapeutic methods. But by that time PTSD will likely no longer be diagnosable.

**Overview of Symptom Tracking Data**

So what do the collective data indicate with respect to the recovery of regulatory integrity using ILF feedback? In broad brush, trainees can be grouped into three principal groups: 1) A rapidly responding group consisted of nominally 25% of the population. Here functional recovery could be achieved within twenty sessions, with a median value of less than 10 sessions. 2) A conventionally responding group in which recovery was achieved within 20-40 sessions, in line with common expectations within the field for a variety of indications. And 3) a treatment-resistant group in which recovery could not be achieved by the time training was terminated. This group consisted of nominally 25% of the population.

These divisions are reflected in the trends seen with the most common symptoms. Thus flashbacks of trauma were found to resolve in 75% of the trainees. Panic attacks largely subsided in 80%. Agitation responded in 70%; likewise irritability; anger resolved in three out of four cases. Anxiety and depression responded very similarly, becoming clinically insignificant in about 80% of cases. Fears resolved in two-thirds of cases. Sleep dysregulations responded in a manner representative of the entire data set, with nominally 25% rapid responders and some 50% typical responders. One exception was night sweats, where there was 80% favorable response and nearly half of all cases responded rapidly. The most favorable outcome was seen for migraines, where less than 10% were either non-responders or very slow responders, and some 40% of cases were in the rapid-response
are the starting points for all of our clients, irrespective of diagnosis, and this is all being done within the frame of the dysregulation model. Either there is a rather simple core deficit underlying all of the disparate symptoms being addressed in PTSD, or the simple challenge which the brain undergoes in ILF training manages to reorganize the whole system despite its complexity—or both.

**Historical Development of ILF Training**

Before we take this further, one must also ask whether the quality and comprehensiveness of the above results collectively are attributable specifically to the ILF training. It is unquestionably our clinical impression that the ILF training has allowed us to do our best work. But we can also bring some supportive evidence to bear. The story goes as follows: We have been individualizing our training for well over a decade now, and have observed a gradual trend to lower and lower reward frequencies over the years, with the further development of clinical expertise. The modal value in the distribution of rewards tended always to be the lowest frequency we had available in software. For years this was limited to 1.5 Hz because the software utilized 3-Hz bandwidth.

The extension of the reward frequency range down to 0.05 Hz occurred in June of 2006, and over the first six months thereafter some 52% of clients trained at the lowest frequency. Over the subsequent six months, this dominance increased further to 66% as more familiarity was gained with the low-frequency training. More than 250 clients were involved during this phase. With the extension of the reward frequency range to 0.01 Hz in 2008, 65% trained optimally at the lowest available frequency. With the further extension to 0.001 Hz soon thereafter, the percentage training at the lowest frequency rose to 77% over a period of time as clinical skills were honed. The range was further extended to 0.1 mHz (0.0001 Hz) in March of 2010, and over time some 90% of patients ended up at the lowest target frequency within their first few sessions. It appeared that the lowest frequency was the best tolerated as well as the most effective. Notably, with each extension of the range to a lower limit, the clinical reach extended to more complex clinical presentations that had not responded well earlier, and outcomes systematically improved.

The trend in reward frequency distribution is certainly paradoxical for a number of reasons. The more we extend the reward frequency to lower limits, the more the client population congregates at the lowest value. The opposite was to be expected. One would think that at some point the population would distribute itself more broadly across the newly available spectrum. A second paradox is that the lower the value of the target frequency, the less information is being presented back to the brain per unit time, and yet clinical outcomes are not only better, but are achieved more rapidly.

**The Training of Resting State Functional Connectivity**

All of this makes sense if we postulate that the ILF training is efficiently re-normalizing

Figure 2
the functional connectivity of our resting state networks, and that the relevant information to achieve this most efficiently lies at these very low frequencies. EEG feedback has largely been about the training of resting states of the system. SMR training was first modeled as a calming of motoric excitability. Alpha training is certainly about training the brain toward calmer states as well. Biofeedback techniques are oriented toward calming autonomic nervous system over-activation. It is in the nature of our resting states that they persist over time, and to the extent that they are functional, they will resist “outside” interference. By operating at these very low frequencies, we are challenging resting state functional organization where it is most persistent, and intrinsically most resistant to interference. Hence an even subtle challenge can evoke a response by the brain, which over time mobilizes its functional re-organization.

Let us take a more “wide-angle” perspective on all of the above. The facts before us are that all of the symptoms of dysregulation that make their appearance within the penumbra of PTSD resolve to a greater or lesser degree with a very simple protocol, or small set of protocols, acting upon the functional connectivity of our persistent states, which we now know to be organized around a modest number of resting state networks. There is no bias in this intervention; no directed challenge; no explicit targeting; no overt conditioning. The brain is simply responding to information about itself, about its own instantaneous state. The immediate impact is on activation and tonic arousal, with a longer-term impact on the regulatory ambient of arousal and of specific network activations. This in turn implies that the myriad symptoms we tracked can be seen as grounded in the dysregulation of core regulatory functions—of central arousal, autonomic balance, affect regulation, vigilance, executive function, and of motoric excitability. The results indicate that improving state regulation is sufficient to render most of these symptoms clinically insignificant. This feature of neurofeedback, namely that we obtain broader benefits from the training than we feel entitled to for our modest efforts, has been noted even from the early days of SMR and alpha band training. It is also characteristic of peripheral biofeedback.

In fact, the method of ILF training bears more resemblance to traditional biofeedback than to conventional frequency-based neurofeedback. It bears the greatest resemblance to heart rate variability training, which also happens to be the most promising approach to PTSD among the options in “peripheral” biofeedback. It seems appropriate at this juncture to try to find common ground in the mechanistic underpinnings of our respective tools.

It’s Relaxation Training after all.

We may finally be in a position to give a more scientific description to what we have been calling relaxation training. Our brains’ functionality rests ultimately on the functional integrity of our resting state networks. But how does one access these? Unburdening the system by quieting body and mind takes us there by a variety of means: increasing the amplitude of our cortical resting frequencies can take us there; and perhaps the witnessing of our own resting state network activity takes us there most efficiently. We can finally tell the FDA forthrightly that what we are doing is relaxation training after all. That claim was starting to look a bit threadbare.

Feldenkrais was perhaps the first to appreciate the advantage of working near the least-challenged state, the still-point of the motor system, in order to improve its regulatory capacity. ILF feedback can likewise be seen as still-point training across all of the relevant regulatory domains concurrently. Its therapeutic complement is Alpha-Theta training, the other key contributor to the resolution of PTSD. This can also be understood as a kind of still-point training, a quieting in the psychodynamic domain. At the time of Eugene Peniston’s work, the emphasis was largely on the alpha training component of the Menninger protocol. With the emergence of ILF feedback, Alpha-Theta training has been eased into more of a supporting role, and sometimes a minor one at that. This is testimony to the fact that as our effectiveness in regulating our physiology grows, the burden on heavy-duty psychological interventions is diminished.

PTSD in this model can be described most simply as a disruption of resting state network functional connectivity. The cerebral turmoil of a severe emotional trauma, or of a concatenation of minor traumas, bars a return to quiescent baseline states in many people. The same holds true in minor traumatic brain injury, with physical insult added to the emotional impact that surely must also accompany TBI. A similar mechanism is operative in schizophrenia, in the autistic spectrum, in developmental trauma, in addiction, in the demenias, and in chronic pain syndromes.

In all of these cases, the first resort should be to retrain the brain toward integrity in resting state functional connectivity. Over time, we benefit from a virtuous cycle in which control systems regulate each other as opposed to dysregulating each other. At some point, of course, the incremental benefits diminish, and if necessary, one adds on other methods—many already well established within the field—to complement the ILF training. So, we are not claiming that ILF training is the “universal solvent” of the neurofeedback world, but it does appear to get at the core of the most intractable psychopathologies, and is therefore to be recommended as the central focus, and in particular as a starting point, of neurofeedback strategies in the general case.

Finally, a note of caution must be sounded. What is probably a general rule in feedback applies to ILF training as well: The stronger techniques place the greater burden on the clinician for proper direction and management of complex clinical presentations. Readers may be enticed by the above to rush into practice with ILF training simply because their instrument allows it. That temptation should be resisted until proper training has been undertaken.

Conclusion

The evolution of Infra-Low Frequency training over the last six years has given us a technique that bridges the disciplines of traditional biofeedback and of conventional neurofeedback. It is frequency-based data construction, along with the rest of neurofeedback, and yet one is training on a time-domain waveform that is more characteristic of biofeedback. ILF training is an alternative to biofeedback for restoring autonomic regulation, and it exceeds prior neurofeedback approaches to affect regulation. These are the principal keys to the resolution of PTSD, which ILF training accomplishes efficiently.

All this is best understood in a systems perspective in which core regulatory functions are targeted as a first priority, yielding comprehensive coverage of the state regulation issues that dominate in psychopathology. Of course ILF training is not exhaustive, either in the perspective of neurofeedback and of biofeedback. In application to complex disorders such as PTSD, and with the objective of optimal functioning in mind, multiple brain challenges are called for. That means bringing our “multiple intelligences” to bear from a variety of scientific and clinical perspectives. It is time to break down the internal barriers within our discipline to bring that about.
BCIA Certification Exams—A New Look at This Standard

“‘I didn’t fail the test; I just found 100 ways to do it wrong.’
~Benjamin Franklin

Written exams are a necessary part of our professional lives. While most of us don’t enjoy taking them, we do like to challenge ourselves and expand our knowledge. We don’t fear learning new material; we fear the unknown. What will the exam be like? Did I study the right material?

According to the Institute for Credentialing Excellence (ICE), formerly the National Organization for Competency Assurance (NOCA), “certification of specialized skill-sets affirms a knowledge and experience base for practitioners in a particular field, their employers, and the public at large. Certification represents a declaration of a particular individual’s professional competence.” Most certifying agencies set standards of education and training to demonstrate entry-level competency. Most share the challenge of evaluating whether applicants have mastered a body of knowledge required for entry-level competence. BCIA, like many other certifying agencies, has found it challenging to build and maintain meaningful, current, and psychometrically sound assessment instruments. BCIA’s exam task forces have taken this work very seriously.

BCIA believes that tests should be fair, reflect essential knowledge, and allow test-takers to demonstrate their mastery. We believe our tests should have passing scores that clearly differentiate candidates with advanced knowledge from those who cannot demonstrate mastery of the fundamentals. We would like to demystify how we build our exams.

Exam 101

The Blueprint of Knowledge Statements relevant to each certification program identify the fundamental science, history, and concepts underlying each modality, independent of specific theories, vendors, or brands of equipment. This information is the basis of the didactic training required for certification. We divide each blueprint into content areas called rubrics and then assign a specific number of hours to each rubric to help training programs structure their workshops. An 8-hour rubric contributes more items to our exams than a 2-hour rubric. BCIA exams are built using 100 multiple-choice questions with only one right answer. We source each exam item to references in our Core Reading Lists.

BCIA now offers our exams in both paper/pencil and online formats. A candidate may choose any of our regularly scheduled exam sites or may locate a nearby university or public library proctor to monitor the security of their testing. We have successfully offered our exams in English in several other countries and it appears that 2012 will see many more new certificants outside of the US because we have made testing locally available.

Sounds so simple when presented like that doesn’t it? So, what are you waiting for? File your application and set a study schedule!

The Challenge of Building Quality Certification Programs

Many certifications are specific to a profession, such as truck drivers, nurses, or accountants. BCIA, in contrast, certifies professionals with backgrounds as different as neurology, counseling, and physical therapy. Our applicants’ experience levels also vary. While many individuals sit for their exam after years of experience, others have just entered the field and started to use the methodology. While some professionals treat a wide range of clinical disorders, others focus on problems like pain or urinary incontinence. We value our applicants’ diverse backgrounds, experience, and clinical specialization, and have developed exams that assess the common knowledge base that competent professionals should share.

Preparing for the Exam

The BCIA website contains valuable resources to help you prepare for our exams. You will find links to our Blueprints of Knowledge, Recommended Reading Lists, didactic educators, and mentors. Our Blueprint is a roadmap of the content you will need to master. Our Core Reading List contains a compact list of affordable entry-level references that cover the Blueprint and serve as the basis of our exam questions. We don’t publish our own textbook because excellent resources are already available and we want to expose our certificants to multiple scientific viewpoints.

BCIA does not teach didactic content ourselves for the same reason we don’t publish a textbook. We want to encourage a “marketplace of ideas” in which didactic instructors teach our Blueprint in university courses and vendor workshops from their unique perspectives. We believe that our field is stronger when applicants can learn from instructors with diverse teaching styles, materials, and emphases.

Never underestimate the power of a good mentor! Mentors help guide applicants’ personal self-regulation training and development of clinical skills. They help their mentees transform theory into practice. Good mentors are invaluable because they can reinforce didactic training and share their wealth of clinical knowledge. Since BCIA-certified mentors have passed our exam, they are ideal exam coaches.

Passing the Exam

More than 70% of our candidates pass our exam on their first attempt, compared with rates as low as 50% at many larger professional certification organizations. We find low pass rates to be unacceptable. Certification should guide and motivate professionals to acquire entry-level knowledge and develop competent clinical skills; not demoralize them.

What is the value to the field of a certification program that only passes five professionals a year? Our applicants are well-educated practitioners. In most cases, they have invested their own money in equipment, study materials, didactic training, mentoring, and certification fees. Since state law rarely requires them to gain our credential, they are motivated by their ethics and professionalism.

BCIA has found that we can maintain rigorous standards and achieve high pass rates. Our applicants pass our exams for two reasons. First, they are highly-motivated elite professionals. Second, we have designed our program for success. We provide a detailed Blueprint of Knowledge that is directly linked to our Recommended Reading List. All exam items are based on these references and deal with scientific findings instead of opinions. We supply applicants with a directory of excellent university and vendor-based didactic programs, and a list of gifted mentors when they are ready for consultation. Finally, we continuously monitor the psychometric performance of exam items to ensure their validity.

Does a high pass rate mean that our applicants know the answers to all of our exam questions? Of course not! The exam identifies the topics they mastered as well as the areas that may require more reading and study. Certificants can use this feedback to guide their future continuing education.

The Future

BCIA’s exams performed very well during 2011, both in North America and internationally. This fall, 100% of our new certificants in the Netherlands passed our Neurofeedback exam with scores that were higher than average! Keep in mind that English is their second language.

Despite the success of our exams, we have already started to reevaluate our Biofeedback, Neurofeedback, and Pelvic Muscle Dysfunction Biofeedback Blueprints, Reading Lists, and exam questions for the second time in five years. We invite you to submit multiple-choice exam items using our Recommended Reading List’s sources. For each exam item you submit, you will earn one-half

Continued on page 38
Audio-Visual Entrainment as a Treatment Modality for Post-Traumatic Stress Disorder

Part I of II

Dave Siever

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

INTRODUCTION

The American Psychiatric Association defines psychological trauma as “the development of characteristic symptoms of intense fear, helplessness, or horror, following exposure to an extreme traumatic stress or involving direct personal experience of an event that involves actual or threatened death or serious injury, or other threat to one’s physical integrity; or witnessing an event that involves death, injury, or a threat to the physical integrity of another person; or learning about unexpected or violent death, serious harm, or threat of death or injury experienced by a family member or other close associate,” (American Psychiatric Association, 1994).

The ensuing severe and chronic stress further brings about a host of trauma spectrum disorders, which may include impairments in learning and reasoning, impaired alertness, and increased destructive behavior including smoking, alcoholism, drug abuse, family violence and reckless risk taking (Bremner, 2002).

Somewhere between 25% and 50% of all Americans are exposed to a psychological trauma at some time in their lives, related to a wide variety of incidents including child abuse, assault, rape, car accidents, house fires, and natural disasters, (Acierno, et al, 1999). Of these, about 15% will develop PTSD, roughly comprising 5% to 8% of the
American population, making it one of the most common illnesses in the USA. PTSD is 10 times more common than cancer, yet society dedicates only one tenth the funding to PTSD research as it does for cancer research (Bremner, 2002).

The Aftermath of War
About one million young men experienced the stress of the Vietnam War between 1963 and 1971 and several hundred thousand were deployed in the Gulf War from 1990 to 1991 (Bremner, 2002). Service members assigned to combat support units that are not on the front lines are just as likely to be exposed to the effects of PTSD, since rear echelon units no longer have the traditional distinction of being non-combative. The individual service member’s physical condition, training and experience for combat will certainly prepare him or her for these various traumatic experiences often encountered during military missions, but no matter how trained an individual is to deal with the tragedy of war, trauma is inevitable. For all groups responding after deployment, there was a strong reported relation between combat experiences, such as being shot at, handling dead bodies, knowing someone who was killed, or killing enemy combatants, and the prevalence of PTSD.

For all groups responding after deployment, there was a strong reported relation between combat experiences, such as being shot at, handling dead bodies, knowing someone who was killed, or killing enemy combatants, and the prevalence of PTSD.

PTSD increased in a linear manner with the number of firefights during deployment: 4.5% for no firefights, 9.3% for one to two firefights, 12.7% for three to five firefights, and 19.3% for more than five firefights. Rates for those who had been deployed to Afghanistan were 4.5%, 8.2%, 8.3%, and 18.9%, respectively (Hoge, et al., 2004).

The effects of PTSD are not limited to the soldier; as much or more stress is experienced by the spouse and children. If not diagnosed and treated promptly, PTSD quickly manifests itself into a socio-economic burden on society. To exemplify the far-reaching aspects of PTSD, it has been reported that more veterans have died in motorcycle accidents at home in the USA from thrill seeking (350 deaths) as compared with 259 deaths in Afghanistan (Edmonton Journal, 2006). With the exceeding numbers of civilian and military Americans that suffer from PTSD, research to develop a non-drug treatment/method of therapy for treating PTSD, and without adverse side effects, would be an asset to both the afflicted as well as society.

About the Zone
Socialized mammals, and particularly humans, have two performance zones (Figure 1), one zone requiring higher arousal for simple tasks and the other requiring lower arousal for complex tasks. Running fast, climbing a tree, spearing some food, punching an attacking enemy in the nose, are examples of peak performance under high arousal, in other words, fight-or-flight activity. A highly aroused state of body/mind involves the suppression of serotonin and increased production of norepinephrine, the brain’s adrenalin (Bremner, 2002). This is why athletes who perform highly physical activities do better under the influence of caffeine, as caffeine has been shown to increase norepinephrine.

Complex tasks, on the other hand, such as calculating a math formula, learning new concepts, driving a car in busy traffic, or socialization, are most efficiently performed in a state of calm alertness. Because of their chronically high physiological arousal, those with PTSD may find it harder to shift into the calm-alert state which is optimal to support learning, reasoning, and social interaction (Bremner, 2002).

Physiology of the Fear Response
The survival response rapidly activates via the hypothalamic-pituitary-adrenal (HPA) axis, comprising a triangle in which the hypothalamus and pituitary glands communicate with the adrenals. In response to stress, the hypothalamus releases corticotrophin-releasing factor (CRF), which causes the anterior pituitary gland to produce adrenocorticotropic hormone (ACTH). This, in turn, causes the adrenals to produce glucocorticoids such as cortisol and adrenalin (Bremner, 2002).

The Role of Serotonin in Behavior
Serotonin acts as the brain’s brakes, keeping basic emotions (such as sex, mood, appetite, sleep, arousal, pain, aggression, and suicidal ideation) in check. Serotonin also influences social competence and has been shown to be high in salesmen with outstanding sales performance (Walton, et al., 1992). When subordinate monkeys were given a serotonin reuptake inhibitor like Prozac, they became dominant through friendship and alliances with the females, whereas dominant monkeys deficient in serotonin ruled with aggression (Kotulak, 1997). Like the “Prozac monkeys” and salesmen, college students with the most friends have serotonin levels 20% to 40% above the norm, and connect better socially, with improved ability to read facial, verbal, and body expressions (Harmer, et al., 2003). By contrast, low levels of serotonin are tied to loss of control and helplessness, which may manifest as temper and rage (Sapolsky, 2003), and other impulse-control disorders (Kotulak, 1997). A study by Linnoila (1983) of prisoners who were in jail for manslaughter, used serotonin levels as a basis in predicting with 84% accuracy those who would recommit manslaughter following their release from prison.

Figure 1 Arousal Curves for Different Types of Function.
The Role of Norepinephrine in Behavior

Noradrenalin or norepinephrine (NE), a close relative of adrenaline, is also a player in vigilance (Bremner, 2002). Low levels of noradrenalin are associated with under-arousal, including lethargy and mental fuzziness (Amen, 1998); while above average levels are related to peak performance, abnormally high levels correlate to impulsive “hot-headed” violence (Kotulak, 1997).

Norepinephrine activates rapidly with the perception of a threat, increasing heart rate, blood pressure, and behaviors of aggression (Aston-Jones, Chiang & Alexinsky, 1991). However, a threshold exists where, past a certain point, animals exposed to repeated stress (including family members), could constitute a perceived threat and launch the PTSD survivor into aggression, thus maintaining a trauma-generating mindset to typical daily events. The PTSD survivor could blow up in a constantly revving state. Those with PTSD have diminished capacity to perceive the typical range of facial, verbal and body expressions—only the extreme ones. They struggle to read social and emotional expression; they generally overreact in a condition known as hypoadrenia or adrenergic fatigue (Wilson, 2001). As the adrenals fatigue, so does the locus coeruleus resulting in reduced NE levels in the brain and increased suicide ideation.

**Human Socialization**

Those with PTSD have diminished capacity to form memories later in life (Esch, et al., 2002). Anxiety and fear increases cortisol in the brain, counteracting a brain-nourishing hormone called brain-derived neurotrophic factor or BDNF (Bremner, 2002). Loss of BDNF leads to neuronal cell death in various regions of the brain. The most common structural changes from PTSD are reduced hippocampal volume, amygdala (emotional) activation, decreased Broca’s area activity, and decreased pre-frontal lobe activity. Hippocampal damage impairs both declarative or explicit memory and the ability to recall details of events (Sapolsky, 2003). Hippocampal loss also plays a major role in the early onset of dementia, where the ability to form memories later in life is impaired (Levy, 1996).

Evolutionary concerns may have prompted PTSD to reduce blood flow to the hippocampus and medial prefrontal cortex (Bremner, et al., 1999). The medial aspects of the pre-frontal cortex are instrumental in extinguishing fear responses to conditioned stimuli (Leducx, 1996). People with PTSD do not have normal activation of the prefrontal medial cortex and are not able to extinguish their own fear responses while watching a movie involving violence (Bremner, et al., 1997), whereas people without PTSD are able to rationalize that they are only watching a movie and do not show a trauma response to the movie. This means that people with PTSD have crossed the threshold of being able to return to a relaxed homeostasis, and therefore live in an irrational and constant state of fear. This continued state of fear appears to inflict damage to the frontal and temporal regions, increasing risk for later development of frontotemporal dementia (Bremner, 2002).

**Electroencephalographic (EEG) Changes from Chronic Fear and Trauma**

The increased norepinephrine and cortisol levels in those with PTSD have an effect on EEG activity. The bulk of Quantitative EEG (QEEG) studies involving PTSD, suggest that most often there is reduced alpha activity and increased beta activity, coincident with high arousal (Jokic’-Begic’ & Begic’, 2003), and alpha asymmetry with heightened right-frontal activity in those who have developed depression (Gordon, et al, 2010; Rabe, et al., 2008), as well as elevated beta and theta activity (Begic, et al., 2001).

**Affective Disorders Stemming from Trauma**

Many people, in the aftermath of trauma, also succumb to affective disorders, including depression, anxiety, and mania. Of these, an alarming 15% will commit suicide (Rosenfeld, 1997). For these unfortunate sufferers, the helplessness of depression is not a quiet, passive state; rather it can become active, all consuming, and deadly! The reality of this

Serotonin depletion has been well implicated as a driving mechanism for suicide, where both genetic factors and a string of upsetting life events combine to trigger suicide (Ezzel, 2003). Arango and Mann (Oquendo, et al., 2003) observed with positron emission tomography (PET) scans, a direct correlation between ventral pre-frontal hypofunction levels of serotonin, also in the pre-frontal cortex, and the severity of the chosen suicide method. Slightly lower levels may produce death by an overdose of sleeping pills while extreme deficits will lead to the person jumping off of a cliff or blowing his/her brains out. Violence and suicide are related. Aggression is aimed at others when there is a combination of low serotonin and low norepinephrine, whereas aggression is aimed inward (increased suicidal ideation) when there is a combination of low serotonin and low norepinephrine (Kotulak, 1997).

Conclusions

The fear response involves the reduction of serotonin and activation of cerebral norepinephrine and the adrenals, as the threatened prepare for battle. However, severe traumas can cause a dysfunctional and never-ending activation of the fear response, which fatigues key neurotransmitters and the adrenals as it manifests into PTSD. The implications of PTSD include brain damage, a combination of family and societal violence, alcohol and drug abuse, loss of wages and increased suicidal ideation. No adequate drug or medical treatment of PTSD exists today. Often, pharmaceutical agents and electroconvulsive therapy (ECT) may alleviate the depression, but usually drive the brain further into dysregulation, leaving the patient feeling emotionally numb and struggling with increased cognitive and social impairments. A new non-drug and non-ECT approach needs to be considered.

About the Author

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

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