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*WinEEG computes spectra for magnitude/power/%power/log power, coherence, bispectra, bicoherence, sLORETA, phase, auto and cross-correlations. Calculations can be computed for any selected part of recorded EEG and displayed as graphics and frequency plots. Spectral and coherence parameters are computed for any customizable frequency bands and are displayed as histograms, maps, and tables. Make comparisons between tasks or to normative database.

*The HBI Database includes the processing of over 3000 EEG recordings collected from more than 1000 subjects (7 to 89 years old). QEEG comparisons are available in 7 tasks and ERP in 5 tasks: Eyes Open, Eyes Closed, Visual CPT, Auditory, Reading, Math and Mismatch Negativity. The WinEEG/HBI analysis includes spectra, coherence, ERP and variance computed in multiple montages.

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Letter from ISNR President

As my tenure as president of the ISNR winds toward my term as past-president, I can reflect on some important and encouraging developments over the past year while also pondering our history. At the same time, we can look forward in the direction the field is heading, and anticipate continued growth and development. The changes we are seeing will benefit both clients and practitioners, and impact the field of mental health and personal development as the field progresses toward a more mature and growing stature.

Things certainly seem to be progressing for neurofeedback and neuromodulation. We are seeing a continued growth, validation, and appearance of new and enhanced techniques. In the ISNR, our membership, participation, and quality of publications and presentations are all on the rise. We see increasing numbers of practitioners asking about neurofeedback, adopting it, and discovering the reality that it works, and that it is scientific. Our international membership has grown, and as a result of this year’s initiatives, several countries have aligned their associations with the ISNR.

And what of the various efforts to build the field, gain acceptance, and enlist practitioners? When I was with Bell Laboratories, always a hotbed of innovation, we had a story about how to build a tunnel through a mountain. You put an army on each side of the mountain, and tell them to dig. If they meet in the middle, you have the passage. The same is true of the ISNR.

Continued on page 6

Letter from AAPB NFB DIV President

Sharing is a hot topic in science – from the discovery of mirror neurons that allow primates to share experiences to the virality of internet events that cross the globe in a matter of hours or days. The other day I saw a study which demonstrated how brain function of two people synchronizes during conversation. The ways in which we impose our linguistics has clear neuroimaging correlates, as we all expected it would.

Science is an enterprise that requires a great deal of sharing of knowledge, which is difficult in today’s age of commercialization, patent rights, and stock options for scientific enterprise. But we have entered an age of science where there needs to be vast sharing of data to advance our understanding. In molecular genetics, epidemiology, virology, and dozens of other scientific disciplines there is little advance without a complete picture.

In neuroimaging sharing is expected. Every few months a new fMRI data-sharing center opens up, and the same may become true for EEG as the next round of young academics discover our world. Wikipedia lists a dozen neuroimaging databases already in use, and many more are in the works. Our databases will bring about an understanding of brain structure and function we have yet to imagine, one which will benefit neurotherapy immensely, as we discern what exactly goes awry in anxiety, depression, autism, and the panoply of mental health and personal development.

Do hope you have extensive testing to the issues of cognitive impairments. His works on increasing memory activities, especially after trauma, are some of the most thought-provoking studies in the literature. Do take the time to read his article in this edition. Again Jonathan Walker, MD provides us with his work and observations in identifying the issues in dyslexia. He provides suggestions for neurofeedback training that have been helpful to clients.

Do hope you have had a wonderful summer and that you attended ISNR’s conference.

Warmly,
Merlyn Hurd, PhD, BCIAC/EEG Fellow
ISNR Co Editor

Letter from ISNR Co-Editor

Hello fellow ISNR members,

Welcome to the edition devoted to reading. Roger Riss, PhD has two articles regarding the difficulties clients experience with reading and with the issues of dyslexia. There is helpful information in these articles that you may find useful. Kirtley Thornton PhD provides extensive information on working with learning disabled and especially reading difficulties. Kirtley has a long and successful history of bringing neurofeedback and

AAPB Neurofeedback Division

Mission Statement

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

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

ISNR Mission Statement

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

• Improve lives through neurofeedback and other brain regulation modalities
• Encourage understanding of brain physiology and its impact on behavior
• Promote scientific research and peer-reviewed publications
• Provide information resources for the public and professionals
• Develop clinical and ethical guidelines for the practice of applied neuroscience
Welcome to the fall 2010 edition of NeuroConnections. As students return to their classrooms for another year, the topic of our current issue could not be more timely. While as many as 40% of US school children read below grade level, developmental dyslexia is, by definition, an hereditary neurobiological disorder distinct from reading difficulties secondary to cultural factors, inadequate education, or low intellectual ability. The most common of the learning disorders, dyslexia affects 80% of all children identified as learning disabled and 5% to 17% of school-age children in the US.

Over the past two decades, scientists using “real time” brain imaging methods have made extraordinary progress in unveiling the neural systems which underlie efficient reading and how these systems differ in dyslexic readers. More recently, these same imaging methods have given rise to a an unexpected, and tantalizing finding: despite the fact that developmental dyslexia is a strongly hereditable disorder, relevant neural circuits do not appear to be unalterably “hard wired.” Rather, the effectiveness of a new generation of cognitive neuroscience-based reading interventions appears to be directly tied to their ability to promote and direct functional, and even structural reorganization within brain systems supporting reading.

What is breathtaking about these findings is the growing list of interventions for which we now have direct evidence of brain plasticity in response to treatment, including intensive traditional reading instruction (Aylward, Richards et al. 2003), musical training (Moreno, Marques et al. 2009), phonological skills training (Eden, Jones et al. 2004), auditory processing training (Gaab, Gabrieli et al. 2007), audiovisual training techniques (Kujala, Karma et al. 2001), brief daily PC-based drills (Penolazzi, Spironelli et al.) and even physical exercise (Ploughman 2008). This growing body of evidence seems to lead us to the general hypothesis that, for a reading intervention to be effective, the necessary and sufficient condition is that it must directly promote cortical re-organization of neural networks to more closely resemble those of normal readers. While additional research is clearly warranted, preliminary outcome studies bode well for neurotherapy’s place among the growing list of cognitive neuroscience-based reading interventions. We hope you enjoy the current issue.

REFERENCES

Looking to the future,
Letter from AAPB Ed

Got Professional Engagement?

AAPB has turned a new page in its history. It is an exciting process and a new focus on its members! AAPB members are engaged in their Association like never before. They are now directly engaged in planning and executing the programs and activities of the Association. AAPB has always had committees. But never before have the committees been as open as they are today. All members are welcome to join committees and get involved in the inner workings of AAPB. It is, after all, their Association, right?

Well over 150 volunteers currently serve on AAPB committees and other leadership positions this year. They are energized and preparing for current and future activities and programs of AAPB. Here are just a few of the activities that our committees are under taking:

- Program Committee: Under the leadership this year of Dr. Wesley Sime and Susan Internem, its 13 member committee is currently engaged in identifying the general session speakers for the conference, the conference theme, and planning for a wide variety of educational sessions to be offered during the conference. They will also be engaging the support of the AAPB membership Sections and Divisions to be sure that their interests are represented in the program content.

- Education Committee: This committee has four subgroups: the Teleseminar Workgroup, the Fall Workshop Workgroup, the Preconference Workshops Workgroup, and the Alternative CE Delivery Workgroup. As Chair for the Education Committee, Sharon Langvardt has her work cut out for her in keeping each of the workgroups on track. This committee is just getting geared up for the year and through the engagement of each of the workgroups, we will be announcing a number of exciting educational offerings shortly.

- Membership Committee: Speaking of engagement, this committee is off and running. Under the leadership of Richard Sherman and Tami Maes, they have already evaluated the extensive membership survey that was undertaken last winter and are in the process of developing new membership promotion strategies for AAPB and evaluating potential new services.

- University Outreach Task Force: This group, chaired by Dr. Elizabeth Bigham, has also hit the ground running. AAPB believes that this task force is serving a vital function for the future of biofeedback and the Association. Its primary goals are to identify which schools are currently teaching biofeedback, to establish curriculum exchange to help schools expand the services being offered, to conduct searches of databases to identify who is doing research on biofeedback, and to identify which universities counseling centers use biofeedback.

- Advocacy Task Force: Ron Rosenthal, Task Force Chair, has truly been engaged in working to achieve recognition for biofeedback and neurofeedback through the use of ICD coding. With his committee, Ron has submitted compelling arguments and appeals for the addition of codes designed to influence reimbursement.

- Website Task Force: About a year and a half ago, the AAPB website went through a major revision under the guidance of Task Force Chair, Aubrey Ewing. The group is now engaged in ongoing reviews of the site to keep the content up-to-date and continually evaluating ways to improve both the navigation of the site and its content.

Numerous member volunteers are also engaged in producing AAPB’s highly rated publications, *Applied Psychophysiology and Biofeedback*, our research journal, and our clinical journal, *Biofeedback*.

Engagement is the new buzz word in the association community. From the association perspective, it means providing members with opportunities to participate. And from the member’s perspective, it means getting involved. AAPB is living this new paradigm of engagement. Simply stated, if you would like to see your organization offer a certain set of benefits and programs that it might not be offering now, then get involved and help shape the future of your organization. Your engagement is essential to AAPB’s ability to meet the needs of all members. Our more than 150 volunteers helping us to lead the way is a wonderful testament to our new open door policy. We welcome and encourage all members to get involved … and get engaged!

David L. Stumph, IOM, CAE, AAPB Executive Director

ISNR President
Continued from page 4

a tunnel. If they don’t meet, then you have two tunnels (insert laugh). In our world, there appear to be multiple main thrusts to gain acceptance, including, for example, the clinical/medical model and the peak performance/sports/mental fitness model as two divergent alternatives. In time, we should expect both of these approaches to prosper, thus providing two “tunnels” into popular and professional acceptance.

Another area of divergence is the question of whether or not we should seek coverage from insurance companies or HMOs for our services. A number of offices we know have moved to a cash-only system, and have abandoned the use of insurance altogether. One practice informed me that new legislation may even make it financially impossible to continue, given the burden of record-keeping and other bureaucratic demands.

Other groups continue to look at how we can gain acceptance and support from “big medicine,” by playing into their models and methods. At this time, there are at least a few controlled studies on neurofeedback in print, and more in progress. While some argue that such studies are neither necessary nor helpful, others insist that they are mandatory. There is also a resurgence of interest in collaborative efforts to get neurofeedback accepted and reimbursed, particularly in the federal domain. These efforts will be costly, and require a coordinated, yet focused effort between and among the participants. ISNR is looking at how we can be a positive influence on such ambitions.

Again, if both camps prevail, then we shall have two more tunnels.

With regard to the model of con-
trolled studies, it is notable that the ISNR is currently considering new rules that will require authors as well as vendors to meet conditions of either demonstrating their mechanism of action, or demonstrated efficacy, in order to retain the privilege of submitting studies or showing their equipment. This is an ambitious plan, designed to raise the bar and ensure quality and credibility in systems, methods, and practice.

As we look back on historic efforts, we see the contributions of founders such as Kamiya, Sterman, Lubar, Tansey, Penniston, John, and Fehmi, among others.

We see increasing numbers of practitioners asking about neurofeedback, adopting it, and discovering the reality that it works, and that it is scientific.

Their, and others’, past and continuing research work have provided the basis for many current developments. Think back on the questions and challenges they faced. “Can an individual actually discern and change their EEG at will?” “Can SMR training strengthen the immune system and the resistance to toxins?” “How does the reduction of slow-wave activity help eliminate seizures and improve attention?” “How is brain dysfunction related to the relaxation/concentration cycle?” “Can alpha/theta training really help addicts?” “Are Quantitative EEG signatures meaningful and useful?” “What happens if the entire brain synchronizes in alpha?”

Today, we ask new and broader questions. “How can an EEG map reveal a client’s underlying dysfunction?” “Is epilepsy a learned behavior?” “Is ADD/ADHD a conditioned response that can be unlearned?” “Can the brain learn to self-regulate on a global scale?” “Can we supplant the DSM with a more functional point of view?” “How far up, or down, in frequency can we go?” “How does the brain respond to subtle energy?” “Should research be focused on validating established methods, or on innovating new approaches?” A new generation of inventors, healers, and researchers, is on the move, seeking to take up new tools and new approaches.

At one time, one channel training was all there was. Then two-channels came into use, then 4 channels, and connectivity training was born. Now we can operantly condition the entire brain, and allow it to determine its own strategy for self-regulation and normalization. A tool that once looked like a wooden flying machine has now taken the form of a 21st century spacecraft, integrating QEEG with advanced training protocols, nonvolitional conditioning, simultaneous cognitive assessment and training, and peripheral biofeedback integrated with brain training. Those of us who remember the early days of 1-channel EEG will look back with pride, much as the Wrights must have felt as they saw aviation take to the skies, transforming life and the world.

So our mountain now begins to look a lot like a Swiss cheese, with various tunnels begun and abandoned, and others under continued drilling. There is no single goal or direction, just the desire to contribute, and help others progress in their personal journeys. Achieving our goals does not seem to be as much a matter of “getting there” as it is one of continued progress, evolving old, established views, and supplanting them with new.

Recently, GlaxoSmithKline and Astrazeneca both announced that they were pulling back on drug work in neuroscience, including pain, depression, schizophrenia, bipolar disorder, depression, and anxiety. It is becoming clear that medication has minimal benefit in many of these situations, and has significant side-effects. The old guard may be dying, and they do not have a pill for that. The door is opening for neurofeedback and other neuromodulation techniques to fill the void, and make the contributions we are poised to create. The results of neuromodulation techniques are specific, objective, and lasting. We are now positioned to permanently change the face of mental health care.

In the long perspective, we have moved beyond the “Wright Brothers” phase of getting off the ground. With new technologies and clinical approaches, we are able to image the entire brain, understand its functions, and use this knowledge for interventions that are noninvasive, effective, and enduring. The neurofeedback field is now in orbit, and we are poised to take this field to the moon and beyond.

Tom Collura, PhD
ISNR President

Science is an enterprise that requires a great deal of sharing of knowledge...
The Coordinated Allocation of Resource (CAR) Model Intervention for Reading Problems in Two Clinics

Kirtley E. Thornton, PhD

Abstract
Attaining the ability to read is a fundamental goal of the educational system. However, the attainment of this goal has been fraught with problems. Over the past several decades the special education demands have incurred increased cost to federal government, state and county budgets, with no real end in sight. The problem is further compounded with the lack of an operational, cost effective intervention program. This article presents the data on an alternate approach, activation database guided EEG biofeedback, for 3 individual cases. Pre and post reading, memory and performance data is also presented for 7 children with a history of learning/reading problems. The initial average memory performance for the group was -1.39 standard deviations (SD) below the norm. Following the treatment, the group’s performance reflected an average SD gain of 2.40 (or 334%) and was -.27 SD below the norm. This was derived using the following formula: (a-b)/b where a = the mean post-treatment and b = the mean pre-treatment. Reading fluency also improved: reading time for the standard reading sample decreased from 100 secs pre-training to 72 seconds following completion of the intervention. Thus the results present an encouraging alternative to the intervention methodologies currently employed.

Background
Special education costs and remediation effectiveness has been a growing concern of the educational and political environment due to the rising costs and prevalence. In 2000, the Center for Special Education Finance (CSEF) reported that “In per pupil terms, the total spending used to educate the average student with a disability amounts to $12,639. This figure includes $8,080 per pupil on special education services; $4,394 per pupil on regular education services…. Thus, total spending to educate all special education students amounted to $78.3 billion.” More than half (63%) of the disabled students had a specific learning disability (attention, reasoning, processing,
memory, communication, reading, writing, spelling, calculation, coordination, social competence and emotional maturity. Altarac & Saroha (2007) estimated the life prevalence of a person having a learning disability is 9.7%.

The $14 million National Adult Literacy Survey of 1993 (National Adult Literacy) found that even though most adults in the survey had finished high school, 96 percent of them could not read, write, and figure well enough to go to college. Even more to the point, 25 percent “were plainly unable to read,” period (Baughman, 1994).

Specific education costs have been on the rise in recent years in (Chicago Special Education1, Massachusetts special education) as the number of students requiring these services have risen. In Chicago Public Schools, the percentage of ninth grade students receiving special education services nearly doubled from 1994 to 2002, rising from around 11 percent to nearly 20 percent (Chicago Special Education2).

The ability to read has long been the focus of remediation efforts in the educational arena, albeit with limited success. It has been estimated that the average effect size across 26 different intervention strategies is .42 standard deviations (SD) (Thornton, 2006).

The preferred intervention method for the Chicago and New York public school system is the Orton-Gillingham method. Chicago’s special education program is at a cost of $850 million (45,000 students), while New York City spends $1.2 billion dollars on special education services at non-contracted Department of Education schools (NYC Special Education). The popular FastForWord program has in-house research documenting its effectiveness (Thornton, 2006). However, three independent studies (Rouse & Krueger, 2004; Borman & Benson, 2005; Hook et al., 2001) showed no effect.

The popular Orton-Gillingham method has two controlled research studies examining its effect size (Guyer, & Sabinato, 1989; Oakland et al. (1998). The Oakland et al. (1989) article reported a .34 SD improvement on employed measures after 350 intervention sessions, a result matched by merely providing the student with the Orton-Gillingham video tape, which runs the students thru the exercises. Researchers reported that the degree of improvement in reading demonstrated by students who received the Dyzlexia Training Program by videotape and by those who received it live from instructors did not differ. Using confidence interval and sample size statistics two of the measures employed by the Oakland study were examined for significance. Neither of the measures examined demonstrated statistical significance.

Specific research into defined improvements in reading ability has also been unimpressive. Klingner et al. (1998) reported that 80% of students with disabilities read below grade level at the end of the intervention (45-90 minutes a day for a school year with a co-teaching model). Foorman et al. (1997) reported that 78% of the students who received a reading intervention, which consisted of three forms of reading intervention with extensive professional development and coaching of from the resource room teachers, showed an inadequate response rate. Torgesen et al. (2001) provided students with reading scores in the 6th percentile with intense, research-based interventions. Thirty percent of the students did not read in the average range (word reading criteria) at the end of intervention. However, if a fluency benchmark was utilized, the number of non-responders was well over half the sample. Finally, employing a resource room approach resulted in reduced scores on reading and IQ measures (Orlando & Rivera, 2004).

**Discussion of the activation guided QEEG biofeedback approach**

The Coordinated Allocation of Resource (CAR) model (Thornton & Carmody, 2008) states that the level of performance on any particular cognitive skill (auditory memory, reading memory, problem solving, etc.) is a function of a specific pattern electrophysiological variables (relative power, coherence measures, etc.) and locations. Differing cognitive tasks often employ identical or very similar brain function profiles. This model is somewhat implicit in IMRI and PET studies which report location activation during cognitive tasks. A necessary model of brain functioning asserts not just what the brain does to resolve a task but also elucidates the necessary variables and their directions. The CAR model is an initial attempt to provide the identification of these functions in terms of QEEG variables. This article will illustrate successful treatment of reading disability via a clinical plan of care directly guided by the model in two different clinics demonstrating that the model’s effectiveness is not confined to a specific clinical location.

In previous papers, we reported that successful reading memory is dependent on a different pattern of electrophysiological variables for children (ages 10-14) as compared to adults (age greater than 14) in the database developed by the author (Thornton, 2001). Thornton (2002) established the positive relationship between Spectral Correlation Coefficients (SCC) beta (13-64 Hz) from F7 and SCC alpha from T5 and reading memory in adults.

For children (ages 10-14) the QEEG positive correlates of reading memory involve the relative power of beta1 (13-32 Hz) at T5, P3, O1, O2 & T6, relative power of beta2 (32-64 Hz) at T5, T6 & Fz and beta1 (13-32 Hz) & beta2 (32-64 Hz) at O1 and O2 (peak amplitudes and microvolts) (unpublished). Developmentally, the effective predictors of the quality of reading memory change from higher amplitude in the right posterior to higher amplitude in the left posterior locations. However, the relative power values remain approximately the same (unpublished). For this reason interventions in cases 2 and 3 focused on increasing left posterior beta1 values and decreasing the theta and delta values.

**Case #1**

A previously reported case (Thornton, Carroll, & Cea, 2007) documented the QEEG problems in an individual who showed a failure to suppress alpha when he opened his eyes and engaged in cognitive tasks. He had received 12 years of specialized instruction, in a specialized school for

**Continued on page 10**

### Table 1

<table>
<thead>
<tr>
<th>WIAT Sub-test</th>
<th>Date of Testing</th>
<th>Standard Score</th>
<th>Percentile</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
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<td>1/2007 Pre-treatment</td>
<td>40</td>
<td>&lt;1</td>
<td>3</td>
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<tr>
<td></td>
<td>11/2007 Post-treatment</td>
<td>83</td>
<td>13</td>
<td>7</td>
</tr>
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<td></td>
<td>7/2010 Long-term Follow-up</td>
<td>80</td>
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<td><strong>Pseudoword Decoding</strong></td>
<td>1/2007 Pre-treatment</td>
<td>73</td>
<td>4</td>
<td>3</td>
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<tr>
<td></td>
<td>11/2007 Post-treatment</td>
<td>92</td>
<td>30</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>7/2010 Long-term Follow-up</td>
<td>92</td>
<td>30</td>
<td>8.2</td>
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</table>
learning disabled students, and had received extensive private remediation with well-known programs (Orton-Gillingham, Lindamood- Bell) prior to the CAR intervention. The 1/2007 scores on the Wechsler Individual Achievement Test (WIAT) can be found in Table 1.

There have been no further remediation interventions since 11/2007. After the treatment, he attended a year-long intensive film school and completed it on time successfully. He is currently working freelance on film sets, writing a screen play, and looking for full-time work in the industry.

**Case #2**

A 10 year old male was brought to our clinic with a diagnosis of Attention Deficit Disorder. The initial evaluation revealed the characteristic pattern of excessive theta relative power and decreased beta1 relative power posteriorly. Fifty sessions were employed between 3/2002 and 3/2003. The changes on the Terra Nova (NJ state administered tests) are noted in Figure 1. Of some interest to note is the continued improvement on the other areas (science, math computation, math composite) following the initial dramatic improvements in reading, vocabulary, reading composite, social studies & word analysis. During intermittent follow up sessions (about 20) during 2004 & 2005, the primary focus of the treatment was on the left posterior beta1 and theta relative power issues.

Figure 2 presents the changes in delta, theta and beta1 relative power values during the reading task from the initial evaluation to session #50, which was correspondingly +3.7 (% beta1), -2.5 (% theta), -9.1 (% delta). As the figure indicates, changes in the desired direction (decreased delta and theta & increased beta1) were obtained. The normative standard deviation values for the frequencies during the reading task are presented at the bottom of the figure. Artifact was manually deleted during the initial evaluation and software eliminated during the sessions.

**Case #3**

The following case involves an 11 year old female with a history of learning problems as reported by her mother. She underwent 30 sessions of EEG biofeedback focused on the left posterior locations (T5, P3 and O1). Table 2 presents the changes on the National California Achievement test which was
administered prior to treatment and repeated 9 months after the treatment was ended (see Table 2). Figure 3 presents the changes in left posterior beta1 relative power and microvolt values.

**Data for seven additional cases:**

Reading memory scores were obtained on seven subjects, which do not include the cases previously discussed in this article. The baseline reading memory score was obtained during the activation QEEG evaluation during which the subject had 100 seconds to read a story presented on a laminated paper. The reading period was followed by a 40 second silent recall period (eyes closed) and then the subject told the evaluator as much as they could recall of the story. Twenty minutes later the subject was to recall the story again (delayed recall). Periodically, during the treatment period, the subjects would be reevaluated on their reading memory abilities by having them read a story quietly to themselves, immediately tell the story back to the evaluator and then at the end of the session (33 minutes later) recall the story again. Scores were obtained for both immediate and delayed recall and then summed. The approximate Flesch-Kincaid reading level for the stories was 5th grade. The average age of the subjects was 12.88 years. An example of a story is provided below. The information between the slash marks represent a recall section, which would be scored 1 if all the information was correctly recalled and ½ point if partially recalled.

John Smith / lived on an old dirty barge/ in New York harbor/. One day he left the boat/ to go shopping/ for some milk,/ potatoes/ and hamburger/. He entered an alley/ to save time,/ but as he began to exit the alley/ a tall, large man / came over to him / and demanded his money/. John gave the man / the $10 that he had/, as he was afraid/. The man left./ John continued to the store/ in the hope of getting credit/. He found a $20 bill/ on the ground /near a fire hydrant /as he approached the store.

Total Score possible=24 Flesch-Kincaid Reading Grade Level=5.4

---

**Table 2**

National California Achievement test score changes (case #3)

<table>
<thead>
<tr>
<th></th>
<th>Local May-96</th>
<th>Percentile May-97</th>
<th>Change</th>
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<tr>
<td>Vocabulary</td>
<td>31</td>
<td>88</td>
<td>57</td>
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<tr>
<td>Comprehension</td>
<td>28</td>
<td>60</td>
<td>32</td>
</tr>
<tr>
<td>Total Reading</td>
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<td>74</td>
<td>40</td>
</tr>
<tr>
<td>Language Mechanics</td>
<td>99</td>
<td>93</td>
<td>-06</td>
</tr>
<tr>
<td>Language Expression</td>
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<td>83</td>
<td>49</td>
</tr>
<tr>
<td>Total Language</td>
<td>73</td>
<td>90</td>
<td>17</td>
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<tr>
<td>Math Computation</td>
<td>83</td>
<td>98</td>
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<tr>
<td>Math Concepts &amp; App</td>
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<td>88</td>
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</tr>
<tr>
<td>Total Mathematics</td>
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<td>Total Battery</td>
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</tr>
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<td>Word Analysis</td>
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<td>Spelling</td>
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<tr>
<td>Study Skills</td>
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<td>50</td>
<td>09</td>
</tr>
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**Figure 3**
Biofeedback workshops for professionals... by professionals

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The following section presents data on 7 children (under the age of 14) and 7 adults (over the age of 14) whose initial evaluation reading memory values were about or below 1 standard deviation from the norm for their respective group (children or adults). Each group was composed of 4 males and 3 females. The amount of time allotted for evaluation reading task was 100 seconds. The reading memory reassessment times were recorded for 9 of the 14 subjects (4 children & 5 adults). The 4 children averaged 72 seconds for the reassessment stories while the adults averaged 78 seconds. Thus both groups took less time to read the material while improving their memory scores. The data used to determine the final memory score employed the average of the last 2-3 assessments or in one case a complete reevaluation conducted over a year later. The SD change for the subjects employed the SD of the initial evaluation and the final set of data. The calculation for the SD difference from the normative group employed the SD of the normal group. Each of these cases presented a different pattern of problems which, by and large, were addressed successfully.

**Conclusion**

The CAR model of intervention for reading problems shows promise in these three clinical examples and the reading memory data from the 14 subjects. Each subject presented their unique pattern of difficulties. One of the values of the CAR model is its adaptability to individual differences. The limitations of the CAR model are its confinement to cortically-generated electrophysiological patterns, its inability to define subcortical or cerebellar patterns which contribute to task performance; and its inability to measure the effect of the cortical interventions upon involved subcortical function. The Low Resolution Brain Electromagnetic Tomography (LORETA) method could possibly address this limitation.

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**Table 3**

<table>
<thead>
<tr>
<th></th>
<th>Child Norm Value (SD) N=37</th>
<th>Pre-Treatment Value – N=7</th>
<th>Post-Treatment Value</th>
<th>SD Change (a-b)/b</th>
<th>% Change</th>
<th>Initial SD Difference From Norm</th>
<th>SD Difference from Norm at end of Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Short Term Memory</td>
<td>12.3 (7.6)</td>
<td>.307</td>
<td>11.2</td>
<td>2.45</td>
<td>265%</td>
<td>-1.21</td>
<td>- .14</td>
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<tr>
<td>Long Term Memory</td>
<td>12.7 (7.6)</td>
<td>1.79</td>
<td>9.9</td>
<td>2.25</td>
<td>454%</td>
<td>-1.44</td>
<td>- .37</td>
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<tr>
<td>Total Memory</td>
<td>25 (14.5)</td>
<td>4.86</td>
<td>21.1</td>
<td>2.40</td>
<td>334%</td>
<td>-1.39</td>
<td>- .27</td>
</tr>
<tr>
<td></td>
<td>Adult Norm Value (SD) N=45</td>
<td>Pre-Treatment Value – N=7</td>
<td>Post-Treatment Value</td>
<td>SD Change (a-b)/b</td>
<td>% Change</td>
<td>Initial SD Difference From Norm</td>
<td>SD Difference from Norm at end of Treatment</td>
</tr>
<tr>
<td>Short Term Memory</td>
<td>17.9 (9.2)</td>
<td>6.28</td>
<td>11.8</td>
<td>1.79</td>
<td>88%</td>
<td>-1.46</td>
<td>- .82</td>
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<tr>
<td>Long Term Memory</td>
<td>18.7 (10.8)</td>
<td>5.6</td>
<td>11.08</td>
<td>1.87</td>
<td>99%</td>
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<td>Total Memory</td>
<td>36.9 (19.8)</td>
<td>11.86</td>
<td>23.4</td>
<td>1.94</td>
<td>97%</td>
<td>-1.43</td>
<td>- .86</td>
</tr>
</tbody>
</table>

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Continued on page 14
Dear Colleagues,

The senior editors of the Journal of Neurotherapy: Investigations in Neuromodulation, Neurotherapy and Applied Neuroscience hope that you have noticed and enjoyed the increased content of the Journal. We would like to remind all our friends that we are diligently working to get the Journal Medline-indexed. In order to accomplish this goal we need more submissions, especially of high level research. If you or any of your colleagues are thinking about publishing your research we would encourage you to submit to the Journal.

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Randall Lyle, PhD and Martijn Arns, MSc
This is a story about Mozart, therapy dogs, and a six-year old boy who wanted to learn to read...

Introduction
When a child can’t learn to read, might Mozart make a difference? As improbable as it sounds, this was the hypothesis I was being asked to help put to the test. Each summer, our rehabilitation hospital opens the doors of our “therapeutic learning center” for a two-week “reading day-camp.” During the short camp, children from the general community are offered extra help with reading, combined with a healthy dose of summer fun. A highlight for many participants is the opportunity to read to visiting “therapy dogs,” who seldom seem to notice misspelled words and never seem to tire of listening to their favorite children read to them as they quietly lie at their side.

Last summer, our pediatric neuropsychologist, Dr. Paula Ray, sought to enrich the usual summer camp curriculum by offering several children the opportunity to participate in a small pilot study. Her question: might exposing children to an individually tailored program of therapeutic sound positively impact their reading readiness? To complement her neuropsychological outcome data, I offered to conduct an analysis of pre and post training qEEGs for two young study participants. The case history which follows is of one of those children.

The EnListen method of auditory stimulation is a computer-based therapeutic intervention designed to improve auditory processing and enhance performance in children and adults with disorders of communication and learning. Based on the seminal work of French physician Alfred A. Tomatis, the method begins with a computer-based assessment to identify auditory processing strengths and weaknesses. Each child is provided with a customized therapeutic listening program of music and voice, which has been specifically filtered and gated to condition the auditory system to respond to frequencies found to be deficient in their pre-training evaluation. In the present pilot study, each participant received an individually tailored training program, presented via air and bone conduction stereo headphones, for 2 hours daily, for a total of 60 hours, while concurrently engaging in other activities. (Note: Despite my effort at a catchy title, the child’s individualized program may include, but is not limited to, works by Mozart.) For an in-depth review of the historical roots and neuropsychological basis of the method, see (Thompson and Andrews 2000) while a meta-analysis of previous outcome studies is found in (Gilmore 1999).

Research has provided support for the EnListen method in auditory processing disorders (Ross-Swain 2007), while past studies have identified a high incidence of co morbid auditory processing difficulties in dyslexic children (Ramus 2003). Historically, dyslexia was termed “word blindness,” however, functional brain imaging studies over the past two decades have taught us that the most common cause of dyslexia for English speakers is, in fact, a deficit in auditory processing; in particular an impaired ability to efficiently map written word (grapheme) to corresponding spoken sound (phoneme) (Gabrieli 2009). Moreover, evidence is accumulating that interventions targeting auditory system processing mechanisms in children with dyslexia can improve reading, and in the process, promote cortical reorganization of relevant neural networks (Gaab, Gabrieli et al. 2007; Veilllet, Magnan et al. 2007).

In the case history which follows, we present pre- and post training neuropsy-
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psychological and qEEG outcome data for Kevin, a six and one half year-old youngster who had recently completed the first grade. Like many dyslexic children, reading disability ran in his family: both his father and his older brother had received special education and continued to be poor readers. Although he was of average intelligence, pretreatment assessment revealed a large discrepancy between his verbal and performance IQ scores—Kevin was a visual-spatial learner, who was at a distinct disadvantage in a classroom dominated by language-based learning.

QEEG Data

In order to identify regional processing patterns unique to the reading task; we examined Kevin’s task-related brain activation patterns. Utilizing NeuroGuide software, we calculated the absolute difference between qEEG values for 19 sites collected during an eyes-open baseline condition, and corresponding values during a standardized 3-minute reading task, in order to isolate specific activation patterns unique to the reading task itself. The resulting qEEG maps for each frequency band are compared and contrasted below for pre-treatment and post-treatment evaluations.

**Delta**

*Background:* Slow wave activity has traditionally been associated with either deep sleep, or brain pathology. However, recent studies have also shown that discrete regional increases in delta activity during task may reflect neural networks associated with active cognitive processes. Good readers reliably demonstrate discrete task-related increase in 1-4 Hz activity over temporo-central, but not posterior cortical regions during reading (Angelakis, Lubar et al. 2002). By contrast dyslexics demonstrate diffusely elevated delta during resting baseline (Arns, Peters et al. 2007) as well as while reading (Thornton 2002). Penolazzi and colleagues reported greater overall delta amplitude, as well as localized left posterior delta activation during reading, in dyslexics but not in controls (Penolazzi, Spironelli et al.
Angelakis and colleagues postulated that, when a task-related increase in slow wave activity is correlated with absence of activation in faster portions of the EEG spectrum, then that slow wave activity is likely to be a marker of pathology and brain disengagement during task (Angelakis, Lubar et al. 2002).

**Pretreatment Findings:** During reading, Kevin demonstrated task-related increase in delta over left posterior (O1) visual cortex (figure 1a), in precisely the pattern previously reported by Penolazzi’s group for their dyslexic subjects. The significance of this pattern becomes clear when one understands that for skilled readers, but not dyslexics, activation over left occipital cortex during reading occurs primarily in beta band (Thornton and Carmody 2005), and appears to support effortless, rapid (< 150 millisecond) and automatic word-recognition skills (Shaywitz, Gruen et al. 2007; Spironelli and Angrilli 2009). Kevin’s left occipital delta activation continues on page 18.
Jonathan E. Walker, M.D.

- Board Certified Neurologist
- Board Certified Electroencephalographer
- Past 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

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

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

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Mozart
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appears to be a maladaptive substitute for the posterior beta activation seen in good readers, signaling that neural substrates which support rapid automatic word recognition are deactivating during task; for Kevin, word recognition is likely to be a slow, laborious and effortful struggle.

While background theta activity is thought to be a marker of fatigue or mental inefficiency, task-specific increase in theta is well-known to play a crucial role in cognitive processes including verbal learning.

Post-treatment findings: Following 60 hours of listening therapy, striking improvements were evident in the topographical distribution of Kevin’s reading-activation delta (figure 1b). Reading now elicits the desired focal increase in temporo-central delta seen in good readers (Angelakis, Lubar et al. 2002), a pattern which likely reflects task-related engagement of a network integrating bilateral auditory cortex with a midline attention system. Although posterior delta was still evident during reading, it was now lateralized to the right-hemisphere O2 site, signaling deactivation of the system which supports number recognition, but no longer encompasses the O1 site associated with visual word recognition. His new pattern likely signals improved neural selectivity during encoding of words vs. numbers, a skill found to be deficient among dyslexics (Klimesch, Doppelmayr et al. 2001).

Theta
Background: While background theta activity is thought to be a marker of fatigue or mental inefficiency, task-specific increase in theta is well-known to play a crucial role in cognitive processes including verbal learning. For example, oscillatory activity within the 4-7 Hz band is the “language of the brain” supporting memory encoding when learning a story or word list (Klimesch 1999). Good readers demonstrate a greater left vs. right hemisphere theta activation for language-based tasks. By contrast, EEG studies have confirmed that dyslexics readers demonstrate an “inverted theta lateralization” pattern, with compensatory right hemisphere activation substituting for weak or absent left hemisphere activation during language-based tasks such as reading (Spironelli, Penolazzi et al. 2006). For good readers but not dyslexics, task-related increase in theta amplitude during reading is typically observed over left lateral-frontal and occipital regions. At occipital sites, dyslexics show a complete lack of response to pseudo-words (non-words to be sound ed out phonetically) showing their lack of ability to match grapheme to phoneme in order to encode unfamiliar words into visual working memory (Klimesch, Doppelmayr et al. 2001).

Pre-treatment Findings: A reverse-lateralization pattern was evident in Kevin’s pre-treatment theta band qEEG (figure 2a). Kevin’s compensatory “inverted theta lateralization” pattern (Spironelli, Penolazzi et al. 2006) suggested that, prior to training; he inefficiently relied on his non-language-specialized right hemisphere to support encoding of reading input into memory.

Post-treatment Findings: Following 60 hours of therapeutic listening, Kevin’s maladaptive reverse-lateralization was replaced with a bilateral frontal activation pattern (figure 2b), reflecting his increased success in recruiting the language-specific hemisphere to support working memory and encoding during reading. While this was not the normal, left-frontal-only theta recruitment pattern seen among “good readers,” Kevin’s post-treatment bilateral frontal activation pattern did reflect a shift to the pattern seen in “well compensated” young adult dyslexics who have learned to improve reading accuracy, although their speed and fluency continue to be reduced (Shaywitz, Shaywitz et al. 2003).

Alpha
Background: In contrast to the task-related synchronization, or amplitude increase, which accompanies cognitive processes in the theta band, within the alpha band, the inverse is true. Cognitive processing, which is typically accompanied by desynchronization or the suppression of alpha amplitude during task, is the “best case” finding. Confirming a frequently observed clinical finding, Rippon and Brunswick (2000) reported that dyslexics, in comparison to good readers, demonstrate a global absence of task-related alpha amplitude reduction during reading, as compared to normal readers. Klimesch and colleagues sought to clarify the specific role of alpha in reading. They found that task-related desynchronization in an upper alpha band lying above individually determined peak alpha frequency, is associated with encoding of word-meaning during reading, while desynchronization within a lower alpha band reflects general attentional control processes. In the

Continued on page 20
lower alpha band, Klimesch reported that dyslexics demonstrated failure to suppress 8-10 Hz activity during reading at O1, suggesting difficulties in attention control at the initial word recognition stage. In the upper alpha band, good readers, but not dyslexics, demonstrated highly selective desynchronization over left frontal sites (e.g. Fz, F3, F7) which are responsible for extracting meaning from the written word, highlighting dyslexics’ chronic difficulties with these skills (Klimesch, Doppelmayr et al. 2001).

Pretreatment Findings: Consistent with the above studies, Kevin’s pre-treatment reading vs. eyes open difference map for the alpha band (figure 3a) revealed only a weak, localized alpha suppression during reading, suggesting inefficient visual attention for the written word.

Post-treatment Findings: Kevin’s post-treatment alpha band task activation map (figure 3b) revealed a broad and robust posterior alpha desynchronization, which
now included O1, suggesting markedly improved activation of visual cortex to support rapid visual word recognition processes specific to that region. However, in both pre- and post treatment maps, alpha band desynchronization over left frontal (Broca’s) area, reported by Klimesch and colleagues for good readers, was not observed, hinting that Kevin is likely to continue to experience continued difficulties with extracting meaning from the written word.

**Beta**

Background: Within the beta band, significant findings have been reported for both left lateral and occipital cortical regions during reading task. In normal readers but not dyslexics, Klimesch and colleagues noted a topographically specific cortical activation in 14-16 Hz beta band at recording sites corresponding to Broca’s area (FC5) and angular gyrus (CP5, P3) during word encoding, suggesting a specific role for beta in the grapheme-to-phoneme encoding of words (Klimesch, Doppelmayr et al. 2001). Walker and Norman reported that a neurofeedback protocol to increase 16-18 Hz activity at left temporal area (T3) was quite helpful in improving reading speed and comprehension in a series of dyslexic patients (Walker 2006). Flynn and colleagues reported that dyslexic children demonstrated decreased parieto-occipital beta activation during reading as compared to good readers. (Flynn, Deering et al. 1992). Thornton examined qEEG reading-activation in normal readers for both 15 to 30 Hz beta and 30 to 64 Hz beta bands, and found that, during the input stage, “reading is primarily a posterior beta activity” (Thornton 2002). Penolazzi and colleagues investigated the impact of computer administered phonological training on cortical reorganization in dyslexic children. After a 6-month phonological training (10 min/day through PC software), improved reading speed was significantly correlated with beta amplitude at posterior sites; those children who had the greatest reading speed enhancement showed the largest left posterior EEG beta power increase (Penolazzi, Spironelli et al. 2010).

Pretreatment Findings: Prior to training, Kevin demonstrated a maladaptive decrease in left posterior beta. This finding, in combination with the atypical task-related increase observed in delta band, signaled deactivation of the word-form area which supports effortless, rapid word recognition in skillful readers, but which is typically inactive in “persistently poor” dyslexic readers (Shaywitz, Shaywitz et al. 2003).

Post-treatment Findings: Upon completion of the therapeutic listening intervention, Kevin demonstrated a dramatic enhancement of his ability to recruit left occipital cortex during reading. His increased beta amplitude at O1 during reading predicted that he was likely to experience enhanced reading fluency and faster word recognition skills.

Within the beta band, significant findings have been reported for both left lateral and occipital cortical regions during reading task.

**Coherence**

Background: In comparison to normal readers, dyslexic children demonstrate a number of coherence abnormalities, reflecting the functional connectivity problems underlying the disorder.

Dyslexics demonstrated significantly greater coherence within hemispheres as compared to controls, suggesting functional disconnection between the respective hemispheres (Leisman 2002). During phonological tasks, dyslexics demonstrate disrupted functional connectivity between left angular gyrus (P3, T5) and adjacent temporal and occipital sites (Pugh, Mencl et al. 2000).

Excessive right temporo-central coherence likely correlated with the dyslexic’s compensatory over-reliance on their non-language hemisphere (Arns, Peters et al. 2007).

Findings: Prior to the therapeutic listening intervention, Kevin demonstrated pronounced theta hypocoherece within the right hemisphere. While Kevin’s pre-treatment pattern, displayed in figure 5, was somewhat different than that described by researchers in the literature review above, his theta coherence values nonetheless responded well to treatment and were fully within normative limits upon completion of the listening intervention.

**Neuropsychological Findings**

When training was complete, repeat neuropsychological testing did not indicate an across the board increase in Kevin’s reading grade level. However, he did demonstrate significant improvements in several component skills suggesting improved reading readiness. These included gains of 1 standard deviation or more in span of auditory attention, auditory memory, and reading speed/fluency. Together these gains supported the prediction, based on pre-post training qEEG comparisons, that neural substrates of reading efficiency had been enhanced, providing a firmer foundation for future reading instruction upon Kevin’s return to the classroom. While EnListen training will never replace classroom reading education, it may jump start traditional remediation interventions, by providing the student with an enhanced “brain readiness” to benefit from educational interventions.

**Post-script**

While dyslexia is strongly (54 to 75%) heritable, functional neuroimaging has revealed that a new generation of neuroscience-based interventions have a surprising capacity to promote brain plasticity; “re-wiring” and normalizing function in portions of the brain responsible for the disorder (Gabrieli 2009). In the present case history, this investigator was unprepared for both the consistency and specificity with which sound training appeared to impact electrocortical networks known to be associated with effective reading. Might EnListen therapy be a candidate for membership in this newly emerging group of highly effective, neuro-science based interventions? Further research certainly appears to be warranted.

Acknowledgements: I wish to express my gratitude to Dr. Paula Ray, and my sincere appreciation to Dr. Billie Thompson, for their interest and support in introducing me to therapeutic listening, and the EnListen method, and allowing me to discover for myself its potential to make a difference in the lives of the children that we serve.

Dr. Riss has no commercial relationship with or financial sponsorship by Sound Listening Corporation or the EnListen method. For further information www.enlisten.com; www.soundlistening.com

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Impact of Neurofeedback on Neural Substrates of Reading Readiness: A Case History

Roger H Riss, PhD

Introduction

Operant conditioning of qEEG holds promise as one of an emerging family of rehabilitation strategies which complement traditional interventions by directly targeting cortical reorganization. By addressing the neural substrates of reading readiness, neurofeedback may provide a foundation for traditional educational remediation efforts, jump starting the student’s capacity to respond to classroom reading instruction.

Background

When I first met Matt, he was a 16-year-old high school junior. His mother, herself an educator, reported that Matt’s reading disorder was first diagnosed in the 1st grade. Despite 10 years of remedial reading interventions at school, supplemented with mother’s extra help at home, he continued to struggle with reading. As a result, Matt derived little pleasure from the classroom and redirected his energies toward high school football and baseball, where he thrived and excelled. Like many dyslexics, Matt was an active, popular kid, who preferred to relate to his world through hands on experience. At home, he assiduously avoided leisure reading, with the exception of his monthly issue of Sports Illustrated magazine.

Baseline neuropsychological testing confirmed the problem. On the Woodcock Johnson-III Tests of Achievement, Matt’s reading efficiency fell below 90% of his peers. While his full-scale Wechsler IQ fell solidly within average range, his Performance IQ lagged behind Verbal IQ by nearly one standard deviation, highlighting that, like many problem readers, Matt was a right- brain dominant, visual-spatial learner, who was at a distinct disadvantage in a world where instruction relied primarily on left-brain, verbal abilities.

To get a qualitative feel for his reading experience, I asked Matt to read aloud from the Sports Illustrated issue which he had brought with him to our session. His approach to the passage provided additional insight into both his reading struggles and his preferred compensatory strategies. His reading was hesitant and non-fluent. When he encountered a word which he did not recognize, he defaulted to a sight-reading approach, attempting to guess the word from the initial few letters, rather than decoding phonetically. When this strategy failed, Matt preferred to gloss over words which he did not recognize, relying on context and his overall sense of the passage to compensate for any missed words. This strategy had its limits-Matt did best on concrete passages which were easily visualized, and struggled with abstract metaphor. For example, when the sportswriter colorfully attributed his favorite running back’s phenomenal ability to hold onto the ball to “hands like Velcro mitts,” Matt could neither decode the words in front of him nor make sense of the passage.

Initial Assessment:

To further clarify the basis for Matt’s reading difficulties, we collected 19-channel quantitative EEG data during baseline eyes open and eyes closed conditions, for reference to the NeuroGuide normative database. However, baseline resting data, while helpful, provides only indirect information about brain performance characteristics during relevant cognitive tasks. With this in mind, we also collected qEEG data during standardized reading and delayed reading-recall tasks, referenced against a sample of approximately 100 normal readers aged 15 to 60, utilizing EEGStat analysis software (Nahum and Thornton 2005; Thornton and Carmody 2005). Values for EEG relative power, phase delay and coherence, demonstrating > 1.5 S.D. discrepancy with reference to normal readers were identified.

Pre-treatment findings

We inspected Matt’s EC baseline and task-activation qEEG patterns with particular attention to regions of interest relevant to dyslexia (figure 1).

Matt’s qEEG revealed a characteristic pattern of prominent fronto-central 4-7 Hz slowing, suggesting that he was among the 40% of dyslexic children who may qualify for a concurrent diagnosis of ADHD. For discussion of the underappreciated role of attentional mechanisms in reading difficulties, see Shaywitz and Shaywitz 2008.

Several investigators have reported that good readers, as opposed to dyslexics, demonstrate increased posterior task-related activation during reading, which appears to support efficient visual attention and word encoding processes. Increased task-related beta band activity may be critical to support visual attention for initial word-form detection at left visual cortex (O1) region, as well as the grapheme-to-phoneme matching process supported by the angular gyrus (P3, T5) region. Klimesch and colleagues noted a highly specific posterior cortical activation in 14-16 Hz Beta band during semantic word recognition (Klimesch, Doppelmayr et al. 2001). Thornton extended this analysis to examine activation in a 30 to 64 Hz beta 2 band, and found that, during the input stage, “reading is primarily a posterior beta 2 activity” (Thornton 2002). When we examined Matt’s task-activation qEEG, we found the posterior underactivation pattern during silent reading, in both beta 1 and beta 2 bands, which is characteristic of dyslexic, as compared to normal, readers (see illustration, figure 1).

Guided by past neuro-imaging and qEEG studies, we knew that amelioration of anomalies in left lateral hemisphere regions comprising the phonological loop, visual-auditory association cortex and primary visual cortex were likely to be particularly productive (see Neurobiology of Dyslexia, on page 29 for a brief review). Matt’s EC qEEG revealed a striking pattern, reflecting functional disconnection of the entire left hemisphere network responsible for visual-linguistic processing, which was apparent in both absolute power and coherence views.

Training plan

We developed a 3-stage training plan to address these anomalies (see figure 2, SEQUENCE OF TRAINING). During stage 1, we planned to utilize a beta / theta ratio protocol at Cz to address central slowing.
During stage 2, we planned to address the role of primary visual cortex in rapid word recognition, by rewarding a task-related increase in Beta 1 and Beta 2 activity during reading task. In Stage 3: we planned to address coherence deficits across the left lateral cortex. In contrast to traditional neurofeedback interventions, all training was performed under active task conditions.

Course of training

Over the next 12 weeks, Matt completed 25 twice-weekly training sessions, lasting 45 min each. Sessions were divided into three minute runs, consisting of auditory feedback while Matt silently read to himself. Feedback consisted of an auditory tone, where beep rate signified success in maintaining the target brain activation state appropriate to the reading task to match the EEGStat reference sample target values. Matt’s first seven sessions were devoted to theta/beta training; while the remaining 18 sessions focused on training over the left lateral hemisphere as well as left posterior visual cortex.

In a previous paper, Thompson and Thompson advocated for concurrent training in metacognitive strategies as a useful adjunct to neurofeedback training. Advantages of this approach include enhancing student’s level of engagement, and promoting skill generalization to the classroom (Thompson and Thompson 1998). Consistent with this model, Matt received coaching regarding various metacognitive word attack and attention-focusing strategies. Prior to each run, Matt was encouraged to select and test out the effect of using a particular strategy on his success during the run, in order to identify those strategies which optimized his ability to “get in the zone” which reflected activation patterns predictive of efficient reading.

At the end of each 3-minute run, I probed and informally scored Matt’s recall for the passage he had just read. I next reviewed with Matt his % time above training threshold and average score during the run. We discussed his recall success and his feedback performance scores, comparing his success as he trialed various word attack and metacognitive attention strategies during successive runs. Following 3 successive trials to criterion, we proceeded to the next planned training target.

In contrast to traditional Z-score training, which provides guidance only for baseline EC and EO conditions, Thornton’s task-activation reference sample provides the clinician with an exact numeric training target specific to the reading task itself, for each relevant training parameter and training site. Training thresholds were both precise and specific to the performance of efficient readers under task condition, serving as

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guidepost for activation patterns likely to be associated with improved reading efficiency. This method offered a number of compelling advantages, including the capacity to train to link feedback directly to the reading task, enhanced efficiency of training, decreased likelihood of overshooting the training target, and immediate face-valid feedback to the patient re: the impact of various metacognitive reading strategies on his ability to get “in the zone” reflecting the brain activation patterns of efficient readers.

Clinically, an early sign of progress was seen in Matt’s improving story recall ability. Matt demonstrated a clear learning curve across sessions in the number of details he was able to recount from a short passage read during his training runs, suggesting improving comprehension. As training progressed, reading rate became more fluent. Matt began to demonstrate obvious gains in his phonetic decoding skills—he more frequently attempted to sound out unfamiliar words, rather than simply glossing over them. However, neurofeedback alone could not substitute for the years of reading experience that Matt had missed out on. This became obvious when he encountered irregularly pronounced words which defied the usual rules of phonics. As his phonics skills emerged, I would observe him sound out such “irregular” words phonetically, only to be led astray because the word was not pronounced according to regular phonetic usage.

Our decision to terminate training at 25 sessions was guided as much by practical as by clinical considerations. Because of his involvement in high school sports, Matt’s goal was to complete training during the break between fall football and spring baseball seasons. As spring approached, Matt was anxious to trade in his electrodes for his ball and glove.

Outcomes

Post-treatment qEEG findings provided compelling support for the efficiency and precision of the task-activation, z-score guided, training method. Figure 3 illustrates progress toward Matt’s three primary training goals: 1) Fronto-central 4-7 Hz activity was now within normal limits. 2) Task-related beta 1 and beta 2 power during reading over the posterior cortex now approximated reference values for normal readers. 3) Coherence patterns, reflecting local neural networks, indicated that the left lateral hemisphere was no longer being “left out of the conversation.”
Moreover, whereas traditional training techniques often introduce new amplitude and coherence abnormalities, due to inadvertent over-training effects (see Walker and Horvat for discussion), Matt’s post-training maps suggested that, due to the precision and control afforded by the task activation z-score method, Matt’s training had resulted in few iatrogenic anomalies. Moreover, amplitude values over the left hemisphere now approached normal limits, in response to coherence and phase training alone.

Repeat neuropsychological testing (figure 4) reflected robust interval gains, with pre-post training gains on Woodcock-Johnson reading subtests averaging 1 standard deviation, and all post-training reading scores falling in the low average range or better. An additional measure of comprehension for paragraph length material reflected equally impressive gains: following training, Matt’s comprehension for a 10th grade level passage had improved from 20% to 75%.

The relevance of these changes to real-life school performance was confirmed the day that I received a phone call from Matt’s mother. She informed me, with a mixture of delight and incredulity, that Matt’s English literature class was reading Shakespeare-and Matt had received an “A” on his Julius Caesar examination!

Interestingly, retesting on the WISC-III revealed that Matt’s gains were specific to reading. Matt’s VIQ/PIQ differential remained unchanged. This highlights that Matt’s training exerted a highly specific effect on neurophysiological substrates of phonological processing and reading abilities rather than exerting a broader effect on his underlying right brain dominant learning style (figure 5).

Post-script

Matt returned to the clinic at the end of the summer, prior to starting his senior year. Repeat assessment on Woodcock-Johnson reading subtests indicated stable gains. On repeat qEEG, Matt’s left lateral hemisphere amplitude and coherence patterns continued to approximate normal limits, predicting enhanced reading readiness as he entered his senior year. However, central 4-7 Hz activity had returned, suggesting insufficient intensity of previous work on this goal. Results were discussed with Matt and he was invited to return to work on this goal. Matt opted not to do so, due to the approach of the fall football season. He successfully completed his senior year and is planning to start community college this fall.

Dr. Riss is a neuropsychologist whose practice focuses on rehabilitation of neurological disorders and traumatic injuries. He holds a diplomate with the American Board of Professional Neuropsychology, and is a BCIA senior fellow. He can be contacted at rriss@madonna.org.

REFERENCES


Considering ecological, morphological, and perceptual constraints, cetaceans were more likely than primates to develop language. Dolphins vocalize in the same frequencies in which they perceive the world (echolocation), which means they ought to be able to convey what they ‘see’ with sound. Such an overlap of communicative and perceptual processes seems ripe for the evolution of language. Primates, on the other hand, see one thing and hear another. Even humans, the virtuoso of sound processing of the Primate order, can only muster a few dozen sounds reliably (phonemes) and in a limited frequency range. Most of our brain is dedicated to vision, to images, which explains the rise of squiggles to represent our thoughts (writing) but not the rise of speech.

How did primate vocalizations become words? The move from ape to hominid involved numerous morphological, physiological, and behavioral changes, and at the end of the gauntlet of change we encounter a species speaking sentences and writing plays and treatises. Many scholars look for anatomical clues as to how and when this metamorphosis took place. Anatomically our vocal tract is quite similar to the standard-issue simian larynx. “(I)f a chimpanzee larynx could be grafted into an otherwise normal human being and if all the nerves could be connected such a human individual would be able to produce vocalizations and speech hardly or not discernible from the normal ones” Wind (1981). And our hearing is nothing special either: most mammals can hear what we hear, give or take a bit. Human speech occurs in the sweet-spot of the mammalian auditory system in terms of frequency response, which explains why dogs can readily learn spoken commands but not why cats refuse to, which is another matter entirely. Even minks and chinchillas can hear the difference between supposedly subtle human-centric speech properties like voiced and unvoiced phonemes (Kuhl & Miller, 1975). So what triggered the transformation from emotive-vocalizer to speaker of sentences and verbal awareness? When did this change occur? When did the first person speak? Or is this an unfair question, one that ignores the gradual changes which had to accumulate in order for conversation to become commonplace?

Fine control of the primate vocal apparatus was critical to the emergence of speech and this necessarily required an increased blood supply to those areas of the brain controlling the musculature of our lips, mouth, and tongue, not to mention lungs and diaphragm. Endocranial wall impressions of prehistoric human skulls, however, reveal increased vascularization of these brain areas as early as 30,000 years ago; and cultural artifacts suggest an even earlier point of origin. About 80,000 years ago a small group of humans in Southern Africa were fashioning sewing needles and hooks out of bone and shell and drawing abstract designs in ochre, the Picassos of the Pleistocene Era. It is difficult to explain this combustion of creativity and abstract thought without some semblance of words to guide them.

According to evolutionary theory, the radical shift from primate vocalization to full-blown language must have occurred at a time when its benefits outweighed its disadvantages (e.g., greater likelihood of choking). So the question is, when was there enough ecological pressure to reward a sweeping change in primate/hominid communication? We do know that our current look (brain, body, face) is about 100,000 years old, give or take a few years, and that our most recent speciation (to Homo sapiens from a precursor species) coincided with a series of four ice ages. The warm interglacial periods between cold spells probably lacked sufficient ecological challenge to grant any new survival value to speech, but once the glaciers took over the neighborhood, language may have come in handy.

Many scholars believe that language evolved in response to the needs of parent-child communication (e.g., Locke & Bogin, 2006; Gibson, 1985). Others believed that the loss of habitat pushed us into invading an environmental niche (social predator) that did not cater to our primate strengths (e.g., visual/color inspection, hand manipulation, climbing) so we created new strengths to survive (Jerison, 1988). Most social predators (e.g., wolves) navigate and defend an extensive territory using scent (urine cues), but our olfactory system is embarrassingly weak and we would have to rely on visual memory and vocalizations to map and defend new territories. Speech may have been a byproduct of new hunting practices, our attempts to mimic the cries of other species (food calls, mating calls) (e.g., Fischer, 1981). By imitating more and more sounds of prey, we became facile with our vocal apparatus and developed reference and signifiers in our vocalizations, nouns and verbs and adjectives, the basics of propositional thought. This theory is intriguing in that it means that Nature herself taught us to speak.

We may never find indisputable evidence of the event or pressure that got us talking and thinking in words. But we do know that language is critical to not only communication but cognition. When Nature fails to teach someone to speak fully and freely, or when this process is interrupted or impaired by injury or disease, we need to step in and assist. Without language a modern-day person is lost in this world. Language makes us human. Fortunately those with language can readily assist those without, and we now know more about the processes of language in the brain than ever known before.

References
Case Report: Dyslexia Remediated with QEEG-Guided Neurofeedback

Walker

H.L. was 7 years and 4 months old when she first came to our clinic. Her parents’ chief complaints for her were of reading difficulty, ADHD, auditory processing difficulty, and short term memory difficulty. She had fallen off a bed and hit her head at 4 months of age, but did not lose consciousness and had no obvious sequelae. Her developmental milestones (sitting, standing, walking and talking) occurred within normal limits. Review of systems revealed that her sleep onset was delayed and sleep was fitful. She would often awaken several times. Her parents reported that she was anxious much of the time (especially at school), and would occasionally become somewhat depressed. She was in a regular first grade class, but was reading poorly and at a pre-kindergarten level in her reading recovery program. She often had to hear or read things more than once before she understood them. When given 3 things to remember, she might recall one or two, but rarely the third. She was not particularly hyperactive or impulsive, but did have difficulty maintaining focus in the class room (especially with math). She sucked her thumb obsessively, was a poor speller and was not comfortable in social situations including not going to any friend’s house. Her neurological examination was normal.

An IVA Continuous Performance test revealed a full scale attention quotient of 64, with an auditory AQ of 62 and a visual AQ of 75. Her response control quotient was 89, with an auditory RCQ of 77, and a visual RCQ of 101. Her CNS Vital Signs report indicated average psychomotor speed, low average complex attention, memory and neurocognitive index (NCI). Reaction time and cognitive flexibility were low.

Her raw EEG was within normal limits for her age. A reading difference topograph (Walker and Lawson, 2007) revealed an increase in delta activity at PZ and O1, an increase in theta activity at O1 and O2, and an increase in beta activity at O1 and O2, all during the reading condition when compared to the eyes open /not reading condition. These increases are paradoxical and abnormal, suggesting probable difficulty with visual processing and thinking speed while reading. Abnormal QEEG findings using the Thatcher Neuroguide data base were as follows:

Consistent with (Walker, et al, 2007)

<table>
<thead>
<tr>
<th>Absolute power</th>
<th>Increased 1-4 and 10 Hz at FP1 and FP2</th>
<th>ADHD</th>
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<tr>
<td></td>
<td>Increased 1-2 Hz and 10 Hz at F7 and F8</td>
<td>Poor verbal and emotional expression</td>
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<tr>
<td></td>
<td>Increased 10 Hz at T3</td>
<td>Poor short term memory</td>
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<td></td>
<td>10 Hz at O1</td>
<td>Increased</td>
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<td></td>
<td></td>
<td>Poor visual processing</td>
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<tr>
<td>Relative power</td>
<td>Increased 1-2 Hz at F1 and F8</td>
<td>Poor verbal and emotional expression</td>
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<tr>
<td></td>
<td>Increased 2 Hz at F4</td>
<td>Poor left hand function</td>
</tr>
<tr>
<td></td>
<td>Increased 21-30 Hz at F3 and C3</td>
<td>Anxiety and OCD</td>
</tr>
<tr>
<td>Coherence</td>
<td>Decreased delta and theta coherence at T3/C3</td>
<td>Disconnection between memory and right hand function</td>
</tr>
</tbody>
</table>

Initial neurofeedback consisted of 5 sessions each of 2 channel training in the following sequence:

1. decrease 1-5 Hz at O1 + O2 while reading, to improve visual processing
2. decrease 1-2 Hz at F7 + F8 to improve verbal and emotional expression
3. decrease 1-5 Hz at FP1 and Fp2, to improve attention, hyperactivity and impulsivity
4. decrease 10 Hz at T3 and F4, to improve verbal memory and left hand function
5. decrease 21-30 Hz at F3 and C3, to reduce anxiety and obsessive behavior

A repeat QEEG was then done, which revealed normalization of all the focal slow and fast abnormalities and coherence abnormalities. Her parents reported significantly improved auditory and visual processing and in her ADHD symptoms. They did not feel she had experienced any depression or anxiety. She no longer sucked her thumb. They reported that her reading ability was now “phenomenal.” She was reading at or above grade level, even one book on the 5th grade level. She was falling asleep easily and sleeping through the night. We did not repeat the IVA or the CNS post-training.

Addendum

We have now done QEEG-guided neurofeedback training on 40 children (7-12 years of age), 10 adolescents (13-18 years of age) and 15 adults (19-50 years of age). All have experienced significant improvement in their reading ability. All of the children now read at or above grade level. The average number of sessions required is 35 (range 20-46). Details of our procedure may be found in our original dyslexia paper (Walker & Norman, 2006).

References

Developmental dyslexia is a learning disorder marked by persistent difficulty in learning to read in children who otherwise possess normal intelligence, motivation and educational opportunity (Ramus 2004). It is strongly hereditable; up to 50% of siblings and parents, and 68% of identical twins will have the disorder (Gabrieli 2009).

Over the past two decades, data from laboratories around the world has emerged to reveal a neural network. Figure 1 illustrates three neural systems for reading within the left hemisphere. System #1 (shown in green, left inferior gyrus, Broca’s area) supports motor speech, and the child’s first efforts to sound out new words by vocalizing aloud. This region also encompasses a working memory system which helps the child to encode what has been read into memory. System #2, (shown in red, in the parieto-temporal region, angular gyrus) is pivotal in helping the child to map the visual word, or grapheme, to its corresponding sound, or phoneme (this is where phonics lives in the child’s brain!).

As a child first learns to read, systems 1 and 2 develop first, and reflect effortful decoding. System #3 (shown in yellow, in the occipito-temporal region), the last of the three systems to evolve, develops only with practice. Termined the visual word-form area, it supports rapid (150 ms) effortless and automatic word recognition processes and is of particular importance for skilled, fluent reading.

Convergent evidence from brain imaging has revealed a specific pattern of differences in brain activation between good and poor readers that may represent a “neural signature” for reading difficulties experienced by dyslexic readers (see figure 2). Researchers have identified a pattern of relative under-activation in the back of the brain, coupled with relative over-activation in the anterior left and right hemispheres systems. Adapted with permission.

Continued on page 31
QEEG / TOPOGRAPHIC BRAIN MAPS:
Generalized Anxiety Disorder Subtypes

- **High Beta Subtype:** Anxiety, Insomnia, Alcohol / Drug Abuse
- **High Alpha Subtype:** Anxiety, Depression, ADD
- **Low Alpha Subtype:** Anxiety, Insomnia, Alcohol / Drug Abuse

Cingulate Dysfunction: Anxiety, Rumination, Obsessive Compulsive Disorder

High Mean Frequency Beta: Anxiety, Alcoholism, Insomnia

High Mean Frequency Alpha: Anxiety, Insomnia

**AVAILABLE SERVICES**

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<td>03) Neuroguide - R. Thatcher Normative Database</td>
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<td>04) Neurorep - W. Hudspeth QEEG Analysis System</td>
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<td>05) Thatcher TBI Discriminant Analysis and Severity Index</td>
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<td>06) Thatcher Learning Disabilities Discriminant Analysis and Severity Index</td>
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<td>07) Clinical Correlations and Neurotherapy Recommendations by Bob Gurnee</td>
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<td>08) Conventional Medical EEG - Read by Neurologist</td>
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<td>09) EureKa3! – Nova Tech EEG LORETA Analysis - Eyes Open-Non Database</td>
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<td>10) Neurorep - W. Hudspeth QEEG Analysis System: Task</td>
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<tr>
<td>11) Supervision and Training Hourly Rate</td>
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<td>12) Extra set of Printed Maps sent priority mail</td>
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</table>

Total value: $630
in front of the brain. In comparison to normal readers, dyslexic readers fail to recruit posterior regions of the brain supporting efficient reading (Shaywitz, Shaywitz et al. 2002; Spironelli and Angrilli 2009), and must over-rely on anterior regions (e.g. F7; F3) which reflect conscious, effortful processing, and reliance on rote memory vs. phonetics to support word recognition. Good readers, by contrast, demonstrate robust activation of a left occipito-temporal reading system (anterior to O1) to support rapid, effortless word recognition, and a parieto-temporal (e.g. P3; T5) reading system encompassing left angular gyrus, supramarginal gyrus and posterior aspects of the superior temporal gyrus, to map the visual word to its corresponding sound.

Adding to their disadvantage, dyslexic readers must rely upon compensatory activation of their non-language, right hemisphere during reading to partially offset their reduced capacity to recruit left-hemisphere, language-specialized areas of the brain (see figure 3).

REFERENCES

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Dear ISNR Members—
The Public Relations Committee is in need of your assistance to vastly extend the range of their efforts in two simple ways:

1. Providing suggestions of media targets for the PR Committee and Board of Directors to address with letters and other types of contact. The working definition of “media target” would be
   • national television programs,
   • radio programs,
   • professional organizations,
   • patient or organizations concerned with neurofeedback potential disorders
   • continuing education organizations for various professional fields that should be knowledgeable about or referring for neurofeedback,
   • prominent individuals who have either written about a disorder that neurofeedback improves or celebrities who themselves have gone public with a disorder or bothersome symptom that neurofeedback likely would successfully address
   • science or other journalists that might be interested in neurofeedback

2. Taking the initiative and a few minutes to send individual faxes, e-mails, or hard copy letters to media targets that are locally based or of particular interest to you as individual providers. As an assist, the Committee has written a form letter that you can tailor to your own style to fit specific situations of which you become aware. Contact Grayce Stratton at DrGrayceStratton@aol.com for further assistance.

The Committee will compile a listing to be made available to the entire membership when complete.

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Grayce Stratton
Cindy Perlin
Dianne Roberts Stoler
Kathy Abbott
Tom Collura and Cynthia Kerson [ad hoc as President and Executive Director (respectively)]]
Mozart
continued from page 21

REFERENCES


Helping the Brain Help Us
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Support the ISNR Research Foundation ADD Study
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Gamma Level
Betty Reedy $150

Clinician Fundraising Program (see above)
Richard E. Davis $400
Cynthia Kerson $100

BRODMANN BOOKLET SALES:
5 x $20 = $100
Thank you to the authors: Michael Thompson, James Thompson and Wu Weng Qing

Multi-Component Treatment for PTSD Book Sales:
35 x $49 = $1,715
Thank you to the author: John Carmichael

Total = $25
Daniel Kuhn $25

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As always, your donation is tax-deductable
From a clinical perspective, excessive anger/irritability (AI) is interesting for a number of reasons. First, it is one of the symptoms for about 17 conditions outlined in DSM-IV (American Psychiatric Association, 1994) including my specialty of Post-Traumatic Stress Disorder (PTSD). Second, when AI is one diagnostic element in a condition, the commonly-held clinical presumption is that treating the syndrome will result in a decrease in all component symptoms including AI. Third, the position of many clinicians today is that medication and/or aspects of cognitive behaviour therapy represent the optimal treatment choices for affective conditions.

As measured by a variety of methods including use of the Trauma Symptom Inventory (TSI; Briere, 1992), about 70% of my police and military clients achieve complete remission of their PTSD with what I have referred to as my basic approach. The TSI is a standardized psychological test with good psychometric properties. It yields an overall PTSD statistic, scores for each of the three clusters that comprise the diagnosis of PTSD, and information on a variety of other variables such as AI. Scores above 63 are considered to be clinically significant. Following a careful assessment, my basic treatment approach includes utilizing relevant stabilization strategies, teaching methods of self-regulation for both autonomic nervous system variables and selected muscle tension via clinical psycho-physiology, therapeutic attention to improving sleep, and implementation of specific elements of cognitive behavioural therapy as required. Additional information is found in my 350-page book Multi-Component Treatment of Post-Traumatic Stress Disorder Including Strategies from Clinical Psycho-physiology and Applied Neuroscience (Carmichael, 2010). The book is published by ISNR and all monies from the sale of this book are directed to the ISNR Research Foundation in the furtherance of their agenda.

In addition to improvements such as reducing excessive AI into the normal range for most clients. However, to date three military veterans and one retired police officer were exceptions.

All four had completed my assessment and basic approach. WG and AB were both taking SSRI medication as prescribed when we met but CS and GR were not on any psychotropic regimen. Three clients (WG, CS, and GR), veterans of the Canadian Forces, had been deployed to UN peacemaking duties in the former Yugoslavia. Although without symptoms previously, they developed PTSD from such frequent deployment experiences as being under armed attack, patrolling potentially dangerous locations, and witnessing many acts of aggression and inhumanity from one side or the other in the conflict. At the end of their deployment, they retired from the army. Like most of the military veterans I see, the three came to my attention about fifteen years later. Again similar to other veterans, between deployment and seeing me they had not sought out any professional assistance for their psychological symptoms.

AB had retired from decades of frontline work with the Royal Canadian Mounted Police (RCMP), Canada’s national police force. As such he had witnessed scores if not hundreds of traumatic events in his career as well the experience of physical actions against him by suspects. I saw him during the time leading to his retirement and afterwards in regards his diagnoses of PTSD and MDD (major depressive disorder), both of very long duration. Additionally, he had obstructive sleep apnea in the severe range, had undergone surgery for this, had been prescribed a continuous positive airway pressure device (CPAP), but had not been able to tolerate it.

Following my basic treatment approach, my assessment combination (clinical impression, client verbal report, information from significant others, and scores on the valid TSI) indicated that AI remained in the range of clinical significance.

Consequently, using the DeyMed Truscan, I administered a qEEG to all four, webmailed the data to Dr. Bob Thatcher who inspected the data as well as subjecting it to analysis using both the NeuroGuide and LORETA software. Then he forwarded the resulting brain maps to me along with his NFT recommendations. As it transpired, each client’s qEEG differed significantly from the normative samples such that three NFT protocols were required for each of them. Hence, weekly NFT sessions were arranged wherein 15 minutes was allocated to each of the three protocols. As well, time was available during each session for a variety of purposes such as encouraging maintenance of the basic approach learned previously and managing stressors as they arose. Consequently, session-duration ranged between 1.25 and 1.50 hours. All clients were funded by Veterans Affairs Canada as all had received disability status...
for their psychological condition(s). VA Canada is the federal government agency responsible for both RCMP and military veterans.

As measured by a variety of methods including use of the Trauma Symptom Inventory (TSI; Briere, 1992), about 70% of my police and military clients achieve complete remission of their PTSD with what I have referred to as my basic approach.

Client WG
WG had frequent outbursts of verbal anger, throwing things, and punching holes in walls and windshields. After episodes of anger, typically WG had no recollection of the incident. Alcohol played no role. The triggering component for episodes of AI was the behaviour of his wife who would keep at him during frequent disagreements and not accept a cooling off period; when he called a time-out and walked away, she would follow him and continue pressing her point. Additionally, he found her to be very critical of both he and his son. Anger episodes did not occur at his place of employment, during recreational activities with his friends, or when with his children.

Following basic treatment, WG’s AI score remained elevated although I have not been able to locate it. Brain maps indicated electrophysiological deregulation; insufficient amplitudes in the 13 Hz to 20 Hz range at P4 and C4, again in the 14 Hz to 20 Hz range at C3, and low coherence between P3 and the linear combination of Pz, O1, and O2 in the 8 Hz to 12 Hz frequency range. After 25 sessions of NFT protocols during which electro-physiological characteristics moved in the desired direction, AI decreased to 54 (remission). In the last two months of NFT he had but one anger outburst (overturned a table) in spite of the continuing provocative behaviour of his wife. Also, during NFT he decided to limit alcohol intake even though the topic had not arisen during our discussions. Additional to NFT, during the last number of sessions considerable time was spent in examining his marital situation resulting in the development of a number of options. During the last month of treatment he was promoted to shift supervisor at work. We continue to meet monthly for follow-up which includes booster NFT sessions such as when indicators suggest that levels of electrophysiological variables are moving back towards baseline.

Client CS
The anger outbursts by CS could best be described as road rage which included verbal outpourings, fingering, horn honking, and vehicle manoeuvres against other drivers during his full-time work as a long-distance trucker in charge of a very large rig. Additionally, he had a history of verbal attacks on his teen-aged daughter as well as at least one incident of administering a significant and prolonged physical beating to his dog following a minor melee. His explanation for all episodes was that once the anger arose, he was unable to stop himself even though he experienced prolonged guilt after each episode. His TSI score for AI was 64 after completing the basic approach although specific anger outbursts had continued.

Analysis of the qEEG indicated amplitude excesses in the 1 Hz to 4 Hz range at Pz, insufficiencies in the 15 Hz to 20 Hz range in most posterior sites, and low coherence in the 20 Hz to 30 Hz range between Fp2 and P4. Data collected during the last 27 NFT sessions indicated that desired changes were made according to the electrom-physiological variables of interest. At the conclusion of our sessions, AI was in remission with a TSI score of 46 and he reported no episodes of anger on the road. Moreover, he was able, with clear thinking and without anger, to resolve the issues between himself and his daughter Additionally, although in remission previous to NFT, significant continuing improvements were noted on TSI scales for anxious arousal, intrusive re-experiencing, self concept, and depressed affect. Also, he was able to attend the first reunion of his army unit without any negative effect; previous to this, even news of or thinking about things military would result in an increase in symptoms. Finally, in spite of a life-long difficulty with math, he was able to learn enough so as to pass an algebra exam which led to a desired job change. He chose to not continue with follow-up sessions.

Client GR
GR limited his anger to verbal assaults on his wife for such things as not keeping the house clean and free of clutter to his standards, not helping with home renovations, and not getting a job to help with their finances. Also, he had frequent outbursts directed at some co-workers in the ambulance service where he worked as a paramedic when they failed to replace...
AB was a tall and muscular man who produced elements of road rage when he witnessed illegal or dangerous driving by others even after retiring from the police force. Relatively, post-retirement on a number of such occasions he would stop his car, get out, go over to the offending driver, and...
consistent with my clinical experience with both PTSD and MDD. I have yet to find two identical patterns. Such a conclusion runs counter to the approach by some colleagues in their search to find a single protocol or limited number of them that will be effective for all clients with a particular presentation. Thus, my clinical interventions with NFT will continue to be based on normative analysis of each client’s qEEG.

Second, there is objective evidence that electro-physiological variables such as microvoltages and coherence percentages do change in the desired direction as qEEG-based NFT sessions continue. I monitor and record levels of these variables during each NFT session and then average the end-of-session statistics in three-session intervals. From session to session, variables for some protocols change, others do not, and sometimes levels move in a direction opposite to the previous session. However, with very few exceptions, there is evidence of desired changes such that the averages for the last three weeks are significantly better in comparison with the averages recorded in the first three meetings.

Third, in this case series, there was a corresponding decrease in AI associated with improvements in electro-physiological changes during NFT. With clients who have PTSD, my results indicate that adding NFT to my basic approach increased remission rates from 70% to 90%. In this series, NFT was added for those who no longer met the diagnostic criteria for PTSD or MDD but who nevertheless still had AI scores in the range of clinical significance after completion of the basic approach. However, following NFT, AI reached remission levels for all of the clients (100%). These four clients are the only ones I have seen to date where NFT was required specifically for their AI.

References
Dr. John A. Carmichael has a sole private practice in clinical, police, and military psychology. His office is located in Kamloops, British Columbia, Canada. His practice is restricted to those whose psychological presentations are the result of traumatic incidents. More than 90% of his clients are from police or military backgrounds. His e-mail address is: dr.john@telus.net
Conferences and Calls for Papers


**Society of Applied Neuroscience.** Thessaloniki, Greece. May 5-8th, 2011. Enquiries to: .gruzelier@gold.ac.uk ; bamidis@med.auth.gr ; [http://www.applied-neuroscience.org/](http://www.applied-neuroscience.org/)


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- **Frequent interaction with nationally and internationally known instructors** by phone & e-mail.
- **All courses are conducted at your own pace** – you start when you are ready.
- **CE credits for each course** are available through the American Psychological Association, the National Board of Certified Counselors, & California’s Boards of Psychology and Behavioral Sciences. These groups have reviewed our courses for quality – as have over 400 students.
- **Most courses are approved by BCIA for certification and recertification.**
- **Most courses can be taken in parts** if you just need a few credits for recertification or license renewal.
- **Courses cost $550 each.** Scholarships are available. See our web site for details.

**Distance based courses include:**

- Neurofeedback (EEG biofeedback), General Biofeedback, Anatomy and Physiology / Human Biology, Behavioral Interventions for Pelvic Floor Disorders, Psychopharmacology for Behavioral Medicine Practitioners, Hypnosis for Behavioral Medicine Practitioners, Neuromuscular Reeducation, Neuropsychophysiology, & Pain Assessment and Intervention From a Psychophysiological Perspective.

**Also Available:**

- Mentoring and hands-on training for certification:
  - Doctoral Program in Clinical Psychophysiology given through the University of Natural Medicine.
  - **See our web site for full details: [www.behavmedfoundation.org](http://www.behavmedfoundation.org)**

For further information, call us at **(360) 452-5020** or e-mail us at **rsherman@nwinet.com**.
WHY? THE ANSWER IS SIMPLE. We create the products you want and need to help your clients and improve your practice. Things designed as a system work together more efficiently. And if there should be problems, you can talk directly to the people who created your hardware and software in the first place.

BrainMaster is a leader in EEG biofeedback. We upgrade and improve our products continually. Over the years, we have brought you the BrainMaster 2E, 2EW, Atlantis I & II, the MINI-Q, the original Live Z Score, DC and Slow Corical Potentials, Event Wizard, Session Wizard, supervised web-based remote training capability, 3.0 software, MultiMedia DVD Player Package, and now the Discovery 24E (EEG biofeedback and QEEG).

We strive to provide the best support and service worldwide.

Because we think about you and your practice, we go out of our way to make our products the highest quality and affordable. In addition to fair-pricing, we offer the ability to trade in and trade up and our lease-to-own program keeps you ahead of the curve — for less.

There is no substitute for knowledge. Stress Therapy Solutions, Inc. offers a broad range of workshop courses and training in the how, why, and when of EEG biofeedback, QEEG and general biofeedback related topics. The equipment of choice is BrainMaster. Made in the USA.

NEW! DISCOVERY* 24ETM
24-channel EEG is an outgrowth of the BrainMaster 2E Atlantis series of EEG and biofeedback devices. Train 2-19 channels. Offers Neurofeedback, Biofeedback Training, and QEEG acquisition and training live! Compatible with industry standard software packages that read standard EDF, Lexicor, or ASCII files.

THE ORIGINAL LIVE Z-SCORE TRAINING
BrainMaster introduced this method in 2006 and has continued to develop advanced methods. Only BrainMaster provides flexible training of multiple Z-Score targets (%ZOK), target biasing, and automated review of up to 248 Z-Scores from 4 channels. Add to Atlantis or Discovery 24 systems. Train 2, 4, or up to 19 channels (coming soon) of BrainMaster Live Z-Score. 19 channel Real Time Z-Score currently available.

HOME NEUROFEEDBACK TRAINING
Clinician controlled, website based.

upcoming workshop events*

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<td>Live Z-Score EEG Training Method (4-13 channels and above)</td>
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<tr>
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<td><a href="http://www.brainmaster.com">www.brainmaster.com</a> /sales@brainmaster.com</td>
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*Final field-testing stage.
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