NEURO CONNECTIONS Newsletter

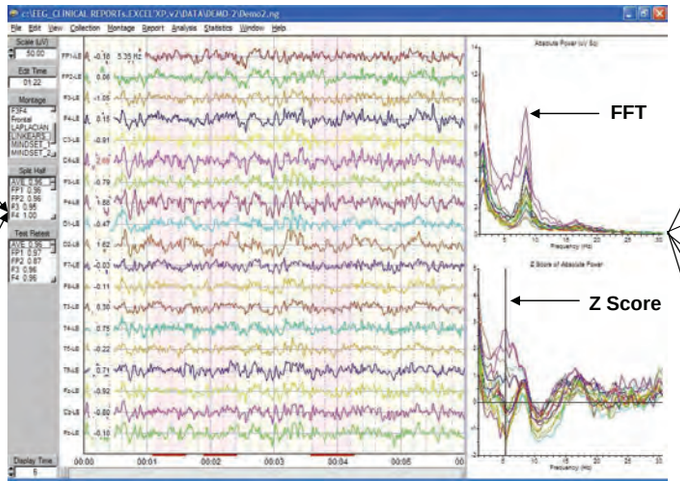
Fall 2009

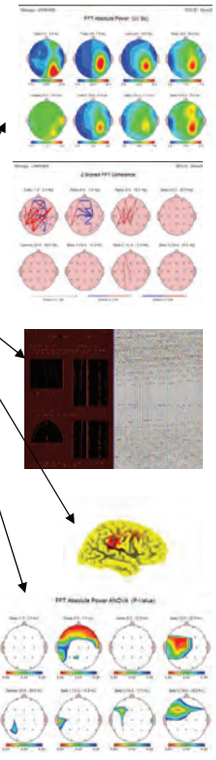
A joint newsletter from the ISNR
& the aapb Neurofeedback Division

NeuroGuide by Applied Neuroscience, Inc.

The most affordable and comprehensive EEG system available: Dynamic EEG Databases, LORETA, Discriminant Functions, Power, Coherence, Phase, JTFA, Burst Metrics, Phase Reset, Statistics, Instantaneous Coherence & Phase Reset, Phase Locking & Phase Shift Duration, Batch Processing, Bi-Spectra & Neurofeedback

BESA
BioSemi
BrainMaster
Cadwell
CapScan
DeyMed
Lexicor
MindSet
Mitsar
NeuroNav
NeuroPrax
NeuroScan
Nicolet
NuAmps
ProComp
XLTEK
& Many More





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

Take a test drive – download a FREE NeuroGuide Demo at:
www.appliedneuroscience.com. qeeg@appliedneuroscience.com 727-244-0240

ISNR
International Society for Neurofeedback & Research

ISNR Research Foundation

ISNR Research Foundation
Thank you to all who have contributed to the Foundation to date
2009 RFP Available Online
www.isnr.org/ResearchFoundation.cfm

Become an ISNR Member!

Membership Advantages

- Subscription and online access to the *Journal of Neurotherapy*
- Subscription to *NeuroConnections*
  Joint newsletter with the AAPB Neurofeedback Division
- Listing your Member Profile on our Site
- Discounts to ISNR-Published books and DVD purchases
- Discounts to the annual conference registration
- Corporate Memberships Available

http://www.isnr.org for fees and application

ISNR 2009

Thank you to all who attended and/or presented to make it one of the best conferences this year!

ISNR Indianapolis 2009

www.isnr.org (800) 847-4986

NeuroConnections
ISNR Co-editor: Merlyn Hurd, PhD
merlynh@aol.com
AAPB Neurofeedback Division Co-editor: Roger H. Riss, PsyD
rriss@madonna.org
Managing Editor: Cynthia Kerson, PhD
office@isnr.org
Journalist for MindFull: David Kaiser, PhD
davidkaiser@yahoo.com
Student Editor: Kimberly Weeks, MS
breetheasy@earthlink.net
Publisher: International Society for Neurofeedback and Research
Design: Rosalie Blazej
rblazej@pacbell.net

NeuroConnections is published four times a year and will consider all materials pertaining to the practice and/or promotion of neurofeedback.

Direct all correspondence and inquiries, including commercial advertising information and classified ads to:

ISNR
1925 Francisco Blvd. E. #12
San Rafael, CA 94901
Phone: (800) 488-3867 Fax: (415) 485-1348
Email: *office@isnr.org*

NEURO CONNECTIONS

A joint newsletter from the ISNR & the aapb Neurofeedback Division

Contents

Letter from ISNR President

This has been an encouraging period of time. The ISNR is developing a constructive relationship with CHADD, the largest advocacy organization for people with ADHD. ISNR representatives have been invited to CHADD's annual meeting and we're inviting CHADD leadership to our annual meeting. We think this is the beginning of a very beneficial dialogue.

We are also hearing numerous reports of neurofeedback reducing symptoms of Autistic Disorder, as discussed in this issue of NeuroConnections. I've worked with a number of children and young adults with Asperger's, PDD-NOS and Autistic Disorder - the "Autism Spectrum Disorders" (ASD) - over the last several years. This has been very rewarding work, particularly when we hear of improved self-management of anxiety and improved social relations. We use combinations of neurofeedback, slow paced respiration with heart-rate variability biofeedback and skin conductance biofeedback. Whether the coherence problems that seem frequent in frontal regions or the hypercoherence that we often see in posterior regions can truly be altered, with concomitant clinical improvement will require careful controlled studies. On an individual basis in my clinic we train these functions and behavior as well as emotional control improves. We also frequently – but not in all children – see major excesses of high frequency beta (18 – 30+ Hz). This usually appears over central and parietal regions. We've found training this

Continued on page 6

Letter from AAPB President

QEEG-Guided Neurofeedback for Personality Disorders

At last count there are 200 listservs on Yahoo in which people discuss neurofeedback or EEG, from professionals comparing protocol differences to the parents of a child receiving services to the hobbyist engineer building a better amplifier. Add to these dozens of commercial and non-profit groups with private discussion lists such as ISNR, EEG Spectrum International, EEG Info, Neuroguide, BrainMaster, LENS, and SKIL (my company). And add to this the many temporary email groups that form and unform weekly over single issues or events, and add in the other common carriers like AOL, Google, and MSN, and what we have is a cacophony of opinion and discussion. It's amazing anything ever gets accomplished or standardized.

We have three major scientific societies that do help standardize our process and our technology, which act as keels to our ship, improving the science of quantitative EEG assessment and neurofeedback and distributing empirically-validated information. These societies are AAPB, ISNR, and ECNS (EEG and Clinical Neuroscience Society) and in terms of mission and membership, AAPB is a mix of autonomic nervous system (ANS) training and central nervous system (CNS) training, whereas ISNR and ECNS focus on CNS training. We also have splinter or daughter groups from the larger

Continued on page 6

Letter from ISNR Co-Editor

Dear Readers,

Welcome to the fall edition which you are receiving with the Journal of Neurotherapy. The combining of the two in one mailing will be the method from now on. Hope this is useful to you.

In this issue the focus is on Autism and Asperger's. A number of highly expert clinicians and researchers have contributed comprehensive articles that have information to help you in your work. Personally, I am very excited regarding this issue!

Martijn Arns, Werner van den Bergh and Jay Gunkelman provide an article on theory-driven approach to QEEG. The authors came together as they began to examine similarities between EEG Phenotypes and Vigilance Stages which they saw as dynamic variants of the EEG as a function of time occurring in the same subject. I urge you to read this article to obtain more insight into the information that is being revealed to us about the function of the brain and the impact on our clinician work.

Michael Thompson M.D., Lynda Thompson PhD, James Thompson PhD and Andrea Reid provides an overview of the biofeedback interventions for Autisitc Spectrum Disorders (ASD). They review findings that point to the anterior cingulate Brodmann area 24 as possibly correlated with difficulties with affect modulation. Their review of theories for understanding the symptoms of ASD that support Neurofeedback interventions cover Mirror Neuron System; Theory of Mind; Weak Central Coherence; Polyvagal Theory and Executive Dysfunction. Well worth reading and

ISNR Mission Statement

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

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

AAPB Neurofeedback Division Mission Statement

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

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

seeing the strong rationale for combining neurofeedback and biofeedback.

Estate (Tato) Sokhadze, Ph.D., Joshua Baruth, M.S., and Manuel Casanova, M.D., have provided an article re: Neuropathological Theories and EEG Gamma Oscillation Abnormalities in Autism. They state "the available neuropathological and structural imaging data suggest that autism is the result of a developmental lesion capable of affecting normal brain growth. Currently our laboratory is looking into the therapeutic effects of low-frequency (i.e., inhibitory) repetitive transcranial magnetic stimulation (TMS) and neurofeedback on the excessive high-frequency EEG activity characteristic of individuals with autism." This article is extremely informative and will help to understand autism parameters and treatment.

Penijean Rutter, M.A., C.R.C., L.M.H.I, a very talented clinician, has provided a case study of a young child receiving Z-score training. The child has Profound Autistic Spectrum Disorder. Her study illuminates the patience and creativity that one needs to work with these children. After 40 sessions they observed a decrease in his hypocoherence across both eyes open and eyes closed conditions. The article will give you more insight into the autism spectrum and Z-score training with children with the disorder.

Bojana Knezevic MA, Lynda Thompson PhD. and Michael Thompson M.D. provide an article of "Using the Tower of London to assess improvement after Neurofeedback training in clients with Asperger's Syndrome." They looked at the changes in executive planning. This is a pilot project at the ADD Centre to measure changes in which metacognitive strategies and Neurofeedback were combined. Again the high quality of research provides us with more information on the types of work that can be done in our offices and with our clients with success.

Robert Coben Ph.D. and Kevin McKeon M.A. have provided a case study regarding children with Autism disorder and seizures. They note that the total cost of providing treatment and additional resources for the children/adults with Autism Disorder is $35 billion annually. Certainly the help that Neurofeedback and biofeedback has shown over and over again would help to reduce this cost and we need to help the medical world embrace the treatments. Their report is very useful to all of us.

David Kaiser PhD provides his usual humorous and deeply profound article, this time of the neuroscience of consciousness. Do have a good time reading his latest musing in this issue.

Daniel A. Hoffman M.D. has provided us with a thoughtful article on the myths, fears and reality of washing a patient off their medication for QEEG assessment. Being an QEEGer, I can relate to the worry of having the Psychiatrist or Neurologist take the client off medication so that the EEG is as "real" as possible. His article will help to relieve our concerns on this issue. Having recently had the opportunity to use the CNS Response of which Dr. Hoffman is Chairman, I can state the information they provided for the psychiatrist, client and myself was very , very helpful.

Finally, we welcome Michael Gismondi, LMHC with his interview with Dr. Robert Thatcher on the evolution of his 19 channel live Z-score and LORETA training system. Well worth reading to see what is the next system we will probably want to add to our repertory of treatments.

Having written this letter listening to Hugh Jackman sing all the songs in The Boy from OZ, I feel very hopeful and joyous that we are providing caring help to so many and for ones that are so needing the treatments!! Keep up the good works!!

See you ar the ISNR Conference in September!!

Merlyn Hurd PhD, BCIAC/EEG Fellow
ISNR Co Editor

Letter from ISNR ED

It took us a little longer to get this issue to you because we are retiming its mailing to coordinate and mail along with the Journal. The issues will now be sent with each journal. If you've missed us, this is why.

This season, we have been busy with PR projects. We set up the *Helping the Brain Help Us* campaign; I hope you're enjoying your free t-shirt from the conference. See page 32 for a sample of the poster

We hope to encourage your patients and clients to support sorely-needed research for neurofeedback.

that you can display in your practice. We hope to encourage your patients and clients to support sorely-needed research for neu-

Continued on page 7

Letter from AAPB Co-Editor

"One in 166." In 2006 the Centers for Disease Control and Prevention updated their best estimate of the prevalence of Autism Spectrum Disorder (ASD) to between 2 to 6 per 1,000; or as high as one in 166 American children. This estimate reflected an astonishing increase from the previous figure of one in 2,500 that autism researchers had accepted for decades. Across a single decade—1993 to 2003—statistics from the U.S. Department of Education revealed a 657 percent increase in the reported rate of autism within US schools. While the explanations for this apparent increase continue to be widely debated, what cannot be argued is the profound lifelong impact of this disorder, and the crying need for a new generation of effective interventions.

Much of the debate has focused on the causes of autism, as research continues into the role of genetics, environmental toxins and early developmental influences, with research extending even to the potential impact of increased rates of television viewing in infants! But for children and families who live with the effects of this disorder, the more immediate questions are closer to home. What can be done to help my child? Who is having success with children like mine? The search for answers for these parents and children has led to a blossoming of clinical innovation and research among neurofeedback practitioners which is increasingly drawing the attention of mainstream neuroscience. This is the theme of the current issue. I tip my hat to my co-editors, Merlyn Hurd and Cynthia Kerson, who have brought together a groundbreaking group of contributors to update readers on this compelling story.

Roger Riss, PhD
AAPB Co-Editor

Letter from AAPB ED

AAPB's University Outreach Initiative

AAPB's University Outreach Task Force Chair, Constance Schrader, said it best in her recent report to the AAPB Board of Directors. Here is what Schrader had to say: "…we are in the midst of a powerful paradigm shift in our conception of the human body. This shift is evident in the attention given to health in the media, in legislative actions, in our organizational initiatives (well workplaces) and in personal lifestyle choices." She continued, "… this shift is occurring in spite of interests and agendas of so-called 'traditional' medical practices that are embedded in industries whose profits rely on the embrace of these practices. Those who are going to investigate human potential as doctors, researchers, and health practitioners are our current students."

AAPB's University Outreach initiative is designed to specifically address Ms. Schrader's observations. Here is how AAPB has defined the charge of this task force:

- Identify universities who use and or/ teach biofeedback but are unknown to us (through equipment and other vendors) in order to acquaint them with AAPB, ISNR, and BCIA's educational blueprint for certification.
- Target selected schools that don't teach biofeedback or applied psychophysiology in their curricula, and encourage them to do so.

We need to be prepared to take advantage of the paradigm shift that Ms. Schrader noted so eloquently above. There are few initiatives that we see as more important than obtaining recognition for biofeedback and neurofeedback in college and university curricula. It offers real benefits to growth of our field and, we all know the benefits that both modalities offer to the wellbeing of human mind and body.

We are looking for your help. If you are affiliated with a college or university, you could play an important role in helping us meet the objectives of this task force. We encourage you to visit our website at www.aapb.org,access our Committee Volunteer Form under "About AAPB," and sign up to participate on this task force. You will be making an important contribution to our field and helping to ensure that this new paradigm shift becomes a reality!

David L. Stumph, IOM, CAE,
Executive Director

ISNR President
Continued from page 4

down at the same time as we train for improved HRV and lower skin conductance is very effective for anxiety and creates a calm feeling that the child can replicate outside the clinic. Given the tendency of kids with ASD to have "meltdowns" easily when faced with social demands, unexpected changes in routine or disappointment, I think the development of anxiety management skill is really important.

I suspect one net effect of the various brain electrical abnormalities we see in people with ASD is to make the Yerkes-Dodson "inverted U" narrower for a given task. Consider the task of "cooperation." Cooperation often requires 1) stopping something one would rather do; 2) interpreting a request; 3) possible negotiation; 4) imagining the potential positive consequences of cooperating—or the negative effects of not; 5) executing a behavior, be it simple or complex. So even "simple" cooperation is really a set of tasks. The Yerkes-Dodson Law predicts that elevated arousal beyond a certain level will make the performance of a given task decline and then cease abruptly (the inverted U-shaped curve). Any kind of brain impairment may in effect "narrow" the Y-D arousal window for optimal task performance of a given task. We see this phenomenon, commonly called "flooding," in traumatic brain injury. At higher levels of arousal, simpler tasks run better. Complex cooperation will run better at lower levels of arousal where more complex thinking and behaving is possible. We hear reports that our kids with ASD will spontaneously report using their anxiety management skills at home and in school when they feel themselves getting ramped up. They report this proudly to parents or to us at the clinic. Parents report improved cooperation and fewer and less intense meltdowns. I see our clients learning to keep themselves in the "performance window" where their better behavior is possible. The development of this ability to relax and handle a situation deliberately—as a conscious choice—is very exciting to see. I think there's also a background alteration in the default brain state that results from training that isn't particularly deliberate or conscious. This is certainly the case in my own experience of blood pressure control. I trained myself 30 years ago to lower my blood pressure, first silently in a non-demanding situation, then during talking and finally during exposure to increasingly demanding and competitive situations (e.g., playing pool). The result is that I have the background habit of low normal blood pressure (no meds) that is quite unconscious; I also can very deliberately lower my blood pressure when the occasional extreme situation arises. I think this is exactly what I see my patients with ASD and other disorders learning to do. I hope ISNR members who work clinically will develop the necessary training and experience to understand and work with people with ASD, applying their clinical skills coupled with neurofeedback and biofeedback. And I hope our researchers will continue their efforts to create excellent scientific studies that will gradually illuminate the best methodologies for helping people with ASD.

John K. Nash, Ph.D., L.P., Fellow,
BCIA-EEG
President, ISNR

AAPB President
Continued from page 4

entities, such as BFE (Biofeedback Foundation of Europe) and SABA (Society for the Advancement of Brain Analysis) which further provide support to our larger mission of brain modification.

I created one of the first list serves in our field in 1996. This was a time when kids fresh out of college controlled the World Wide Web, when e-mail was hyphenated, and AOL was considering whether to litter the biosphere with CDs or not. Unfortunately for those who missed the origins of our field's email lists, I checked back in recently to a few of the lists and although the conversations have changed, they haven't changed as much as I had hoped in the intervening decade. There is more science in the discussion, but the proportion of opinion and confusion remain high. Some days I believe the written word was created solely to foster miscommunication, removing the face-to-face encounters, and without this social constraint, people tend to exaggerate, or lie as Dr House from TV might say, or

even make up fake people and emails to support their opinions.

Science is a set of rules to keep us from lying to each other but with neurotherapy, and with any other clinical science, the clinical comes first and this means opinions and anecdotes often trump general principles. We are working with humans, not molecules, and with humans, variation is the rule, not the exception. So my point is that we will be hard-pressed to wring out opinion from our listservs, and we would be doing it a disservice. Surely we treasure evidence over conviction, but from outsiders come truth, less often from insiders. The truth starts at the periphery and moves in, revising older ways.

With this in mind, the Neurofeedback Division of the AAPB created a listserv for its members and I hope we might focus it on the strengths of this division, which is training both ANS and CNS, co-registering peripheral signals with brain activity. The first order of business for many a person is to connect heart to head and this can be done physiologically, if not metaphorically, nowadays. But how it is to be done is still a question. With the rise of interpersonal biofeedback, couples therapy with physiological assistance, we have a fresh new world to discuss and develop. This list may serve those who come up with new methods, test out those methods, and assist others in constructing a whole-person physiology trainer.

We also need discussions on dollar items—the need to improve the financial health of our practitioners, including methods for receiving third party reimbursement, commercializing technology, and advancing and maturing certification and mentoring programs. This newsletter is a great place for clinicians to have their voice heard above the fray and lay out their beliefs and evidence for all to see and adopt (or ignore), but keep in mind that ideas never stay put. They squirm around, expand and constract, and can always improve, be heard by more people, and a listserver serves that purpose as well.

For more information to join the AAPB Neurofeedback Division and its listserver, visit http://www.resourcenter.net/Scripts/4Disapi9.dll/4DCGI/join/intro.html

David Kaiser, PhD

ISNR ED

Continued from page 5

rofeedback. We also created brochures that inform potential clients about neurofeedback and potential members about ISNR.

I want to extend infinite appreciation to **Sarah Prinsloo**, our PR committee chair, who has been working hard to see the tasks set forth by the committee come to fruition. These tasks include:

- Sending letters to the appropriate congresspersons that encourages them to consider neurofeedback in their health care reform efforts (you can link to it from our home page, edit it and send it to your congressperson as an individual),
- Sending a letter to the 50 attorneys general that state that the unwillingness to pay for biofeedback is actually a restraint of trade. This letter, which was first composed by the Northereastern Regional Biofeedback Society, can be linked to from our home page (www.isnr.org),
- Coordinating with our members in finding out what other organizations they belong to and which other conferences they attend and/or present at in an effort to determine where ISNR may be best suited for creating a presence to inform others about neurofeedback and about our society,
- Creating a DVD about neurofeedback and ISNR that can be distributed via the Web, and
- Creating a template letter that can be used to respond to newspaper and magazine articles that provide or fail to provide information about neurofeedback when applicable.

I also want to thank **Anita Myer**, our Professional and Government Affairs committee chair who helped Sarah with getting the above-mentioned letters to the correct congresspersons in a timely and most useful way and **Cindy Perlin**, another PR committee member, who is working to see that the letter to the attorneys general is properly distributed.

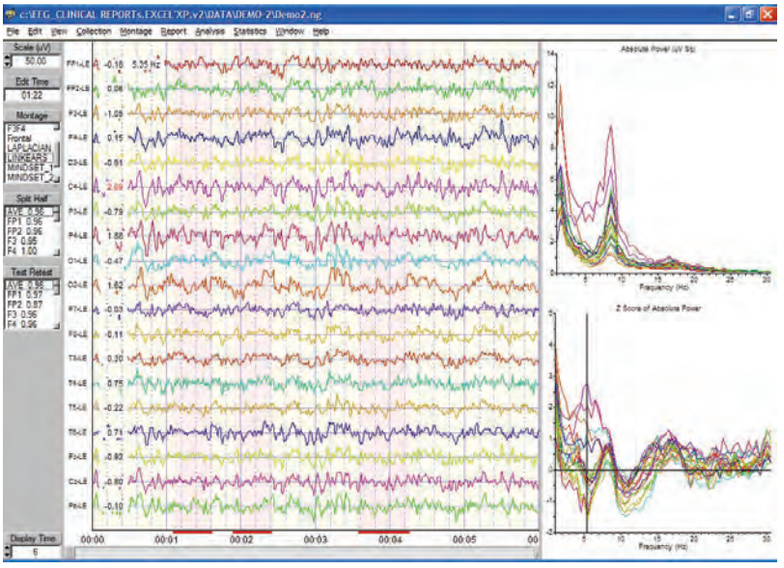
The new members of the Board of Directors will have been voted in and the changing of the guards will have occurred after this writing. In the interest of being as effective as possible, the 2008-09 Board of Directors has worked with respect and fairness to each other, in spite of some disagreements. It is because of this that we accomplished as much as we did and will continue to do so. I would like to extend thanks and appreciation to **John Nash** for leading us with initiative and insight into what the field and association need to do now and in the near future as we grow and become more recognized. And, I welcome **Tom Collura** as this year's president. Tom is one of the most clear-minded, creative and reasonable persons I know and I look forward to working with him.

Until next time,

Cynthia Kerson, PhD, BCIA-EEG
Executive Director, ISNR

19 Channel NeuroGuide Neurofeedback

Seamless Integration of QEEG and EEG Biofeedback

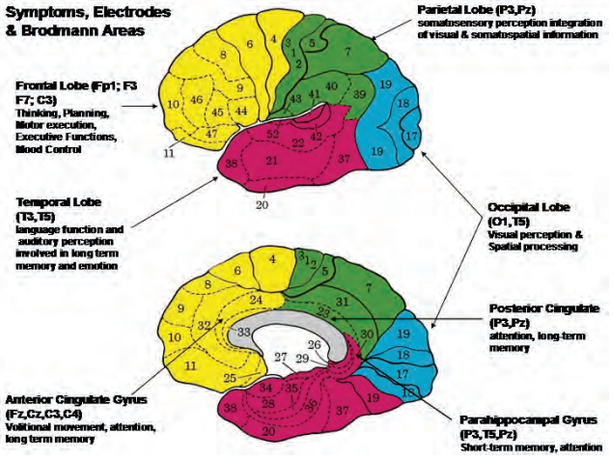


Available to users of

Deymed

BrainMaster Discovery

Neuropulse



Including

Real-Time Z Score

Symptom Check List

Discriminant Functions

Power

Coherence

Phase

JTFA

Burst Metrics

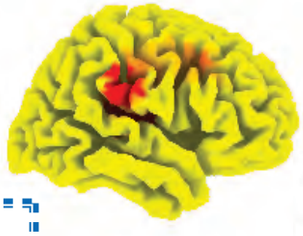
Phase Reset

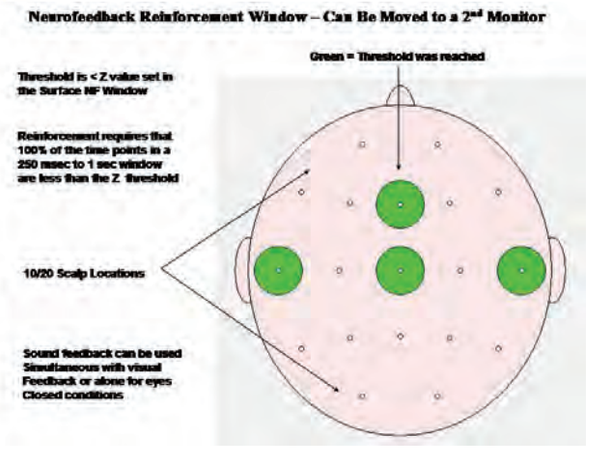
Statistics

Instantaneous Coherence & Phase Reset

Phase Lock and Phase Shift Duration

3-D LORETA Z Score Biofeedback.





Take a test drive – download a

FREE NeuroGuide Demo at:

www.appliedneuroscience.com

Contact US at:

qeeg@appliedneuroscience.com

727-244-0240

Biofeedback Interventions for Autistic Spectrum Disorders: An Overview

Michael Thompson, M.D., Lynda Thompson, Ph.D., James Thompson, Ph.D., Andrea Reid, M.A.




Introduction

When working with Autistic Spectrum Disorders (ASD) a multimodal approach is typically used (Green et al, 2006). The senior author has worked with these disorders for 40 years and had the very good fortune to work and co-author a chapter in a child psychiatry textbook with Milada Havelkova, a child psychiatrist and true pioneer in the treatment of Autism (Thompson & Havelkova, 1983). Havelkova's work began in the 1940s and was devoted to the investigation of educational, behavioral, psychotherapeutic and medication approaches to working with the most seriously disturbed children with ASD. (See Sloman, 2005, regarding medication use.) In the last fifteen years, there has been the experimental addition of EEG Biofeedback also called neurofeedback (NFB) and biofeedback (BFB), especially diaphragmatic breathing and heart rate variability training, to this mix of interventions. Most experts in the autism field are appropriately skeptical of NFB due to the lack of controlled research. Our experience leads us to be enthusiastic, but everyone should also be very cautious not to 'over-sell' our hypotheses and our clinical observations of improvements. In particular, if we are to keep the respect of experienced clinicians in this field, we should be carefully reporting observations but not making any pretense that these observations necessarily correspond to 'sub-types' of ASD. Clinically derived hypotheses can, however, lead to careful future research concerning this complex diagnostic entity.

Autistic Spectrum Disorders include both autism and Asperger's Syndrome (Asperger, 1944). Autism is a disorder of neurodevelopment resulting in pervasive abnormalities in social interaction, communication, and imagination, usually combined with repetitive behaviors and restricted interests. The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) criteria for autism and Asperger's Disorder are very similar with the main difference being that there are no significant delays in language development or cognitive development in Asperger's syndrome (AS) (APA, 2000; Macintosh et al. 2004; Simpson, 2004; Wing, 2001). Language proficiency constitutes a main feature of those with Asperger's, as contrasted to children with autism who have severe language limitations. High functioning autism (HFA) can seem close to AS so the two terms are often used almost interchangeably. In the authors' experience, clients with AS are quite different from those with autism in terms of their emotional responsiveness and interest in others. The term Pervasive Developmental Disorder (PDD) should be reserved for those few children who truly have a "pervasive" disorder in virtually all areas of functioning. Such children are described well in older literature on childhood psychoses and autism (Thompson & Havelkova, 1983).

Despite good language proficiency, those with Asperger's Syndrome (AS) do have communication difficulties in the practical applications of language, such as in conversations. They talk about their interests too much and fail to read the nonverbal cues of the person they are talking to. In addition, there may be differences in their speech, such as the use of pedantic phrases or a voice that is monotone and lacks prosody (intonation, loudness variation, pitch, rhythm). The latter symptoms may be termed a motor aprosodia (Ross, 1981) and, in the electroencephalogram (EEG), we may observe EEG differences from the normal data base at F6 (electrode position of the 10-10 electrode placement system (Chatrian, Lettich & Nelson, 1985)), an area in the non-dominant right frontal lobe that is the homologous site to F5, which is near Broca's area in the left hemisphere. These are frontal sites identified as mirror neuron areas. In autistic children we may see EEG differences, as compared to a normative data base, at both F3 and F4, the 10-10 sites closest to the mirror neurons. Figures 1a, 1b, and 1c (generated from NeuroGuide Software System, Applied Neuroscience, St. Petersburg, Fl.) illustrate typical findings in the EEG of a fourteen-year-old student with Asperger's syndrome who had been treated with special education placement, speech and language interventions, and stimulant medication (Ritalin) since he was in primary school. The quantitative EEG (QEEG) findings can guide NFB training, which was recommended as an addition to the interventions already in place for this teenager.

The EEG of a sixteen-year-old male client diagnosed with Autism is shown in Figure 2. It is a pattern also seen in Asperger's syndrome because the decreased alpha and increased beta activity above 19 Hz is often seen in people with anxiety and in clients in the autistic spectrum. Adults with AS have often been first diagnosed with anxiety or panic disorder. Using LORETA (Pascual-Marqui et al, 2002), Brodmann Area 24 in the anterior cingulate is often identified as the source of activity found to be outside the data base norms when there is anxiety as a main symptom, as illustrated in Figure 3. Thus the CZ electrode site in children and FCZ in adults have been the primary sites for NFB training in these clients.

Emotional regulation is poor in those with ASD. Even in their teens, those with AS may suddenly over-react emotionally, going from placid to tears, or even extreme anger. Others observing the behavior may feel the precipitating incident was quite trivial. Anxiety may be most apparent with any transition or change in routine. We postulate that our consistent finding of anterior cingulate BA 24 being greater than two standard deviations (2 SD) from the NeuroGuide data base (usually in the 4-8 Hertz

Continued on page 10

Autistic Spectrum Disorders continued from page 9

range and/or 13-14 Hertz and high frequency beta above 20 Hertz) may correlate with difficulties with affect modulation (Devinsky et al, 1995). Understanding of emotions is the other side of this. At our centre, children with AS were compared to a normal school group. Subjects completed an adjective check-list describing their mood before and after reading a happy passage. Those with Asperger's did not show the shift towards positive emotion found in the control group. However, the six AS children who completed NFB training in this pilot study identified more adjectives that signified positive mood in the same way as matched, normal controls (Martinez, 2003).

Neuroanatomical Findings

Right frontal and right parietal-temporal junction abnormalities may correlate with aprosodias as mentioned above; that is, deficient expression of emotion in their voice, facial expression, and gestures (motor aprosodia) plus difficulty reading social cues, gestures, and tone of voice (sensory aprosodia). Shamay-Tsoory and his colleagues (2005) have hypothesized that prefrontal brain damage may result in impaired social behavior, especially when the damage involves the orbito-frontal and/or ventromedial areas of the prefrontal cortex (but not dorsolateral areas in this research). These authors note that prefrontal lesions resulted in significant impairment in the understanding of irony and faux pas. In contrast to the patient who has damage to the amygdala, who cannot correctly understand the significance of another person's anger or aggressive behavior, the patient with orbital frontal damage recognizes the significance of other people's emotions but may fail to modulate their behavior as the social situation changes. This kind of impairment could lead to difficulty in correctly recognizing the intentions of others and thereby lead to inappropriate behavior (Bachevalier & Loveland, 2006). In their paper, Bachevalier and Loveland

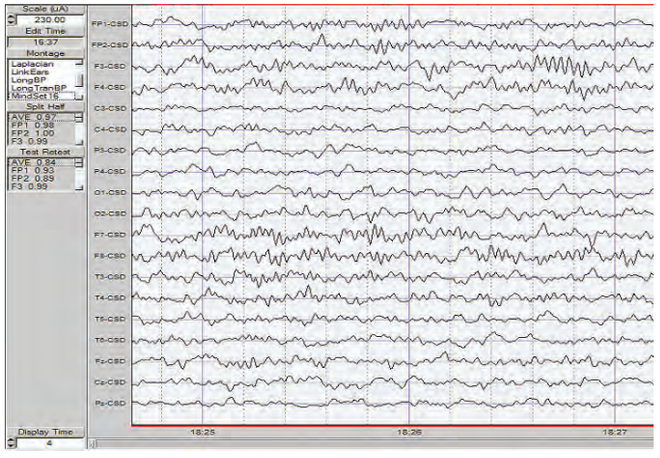


FIGURE 1a: *Child, MC, with Asperger's syndrome. Laplacian Montage, 19 channel EEG. Spindling beta seen repeatedly at F3 (as close as we can be with 19 channel EEG). With single channel we were able to confirm origin between F3 and F5.*

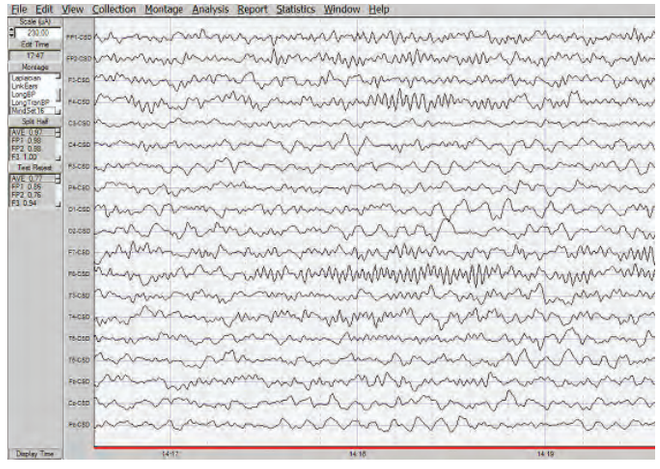


FIGURE 1c. *Child, MC, with Asperger's syndrome, Laplacian Montage, 19 channel EEG. Spindling beta at F4 and F8 possible origin is near F6. Hypothesize that dysfunction in this area (F6) might correspond to observed motor aprosodia.*

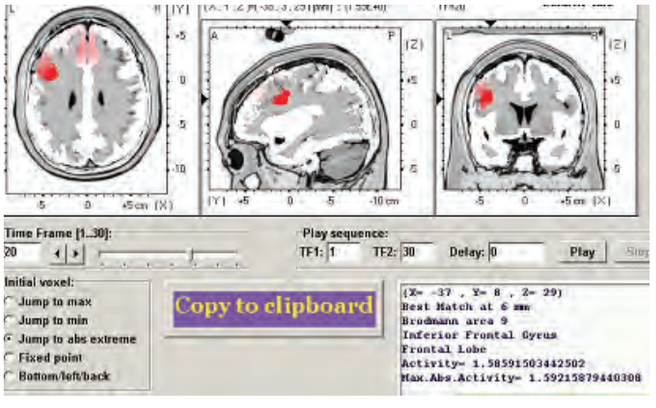


FIGURE 1b. *Child, MC, with Asperger's syndrome. The LORETA image shows bright red, 20 Hz, F5 area, which is at an important mirror neuron site in the left frontal lobe.*

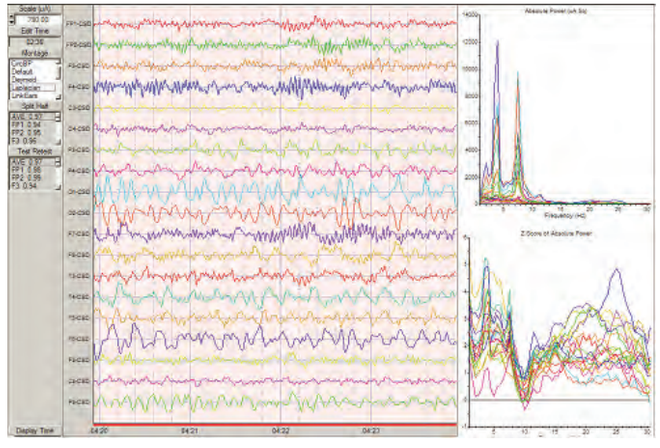


FIGURE 2. *DU, autistic male, aged 10.9, Laplacian montage ec, spindling synchronous beta at F4 (25 Hz 5sd) Fp2, & F7 (20 Hz 3.4sd), C4 (25 Hz 3sd). and high amplitude slow wave at T6. The general pattern shown by this child is a frequently found pattern in both anxiety disorders & ASDs demonstrating high amplitude 2-5Hz, low amplitude 8-10 Hz, high amplitude 11-12 Hz & 12-16 Hz, plus high amplitude higher frequency beta at various frequencies between 17–36 Hz. The specific frequency ranges & sites vary from child to child.*

posit that developmental dysfunction of the orbito-frontal-amygdala circuit is a critical factor in ASD. Imaging studies have shown differences, as compared to neurotypical children, in the density of gray matter at the junction of the amygdala, hippocampus and entorhinal cortex. LORETA consistently shows EEG abnormalities in these regions. The fusiform gyrus and the superior temporal lobe are noted in many papers to be involved in ASD and Porges polyvagal theory specifically notes that these areas may not be inhibiting the central nucleus of the amygdala and this can lead to sympathetic drive increase and a decrease in a myelinated vagal sense of a "safe" environment. Both hearing (as mediated by the strapedius muscle in the middle ear) and facial expressiveness are influenced by vagal innervation.

The importance of noting the above areas that are reported as deviant in the general literature on ASD is that these areas are all found to be outside the data base norms using LORETA (Thompson & Thompson, 2009).

Theories for Understanding the Symptoms of ASD support NFB interventions

Mirror Neuron System (MNS): The MNS is postulated to be involved in the imitation of movements, and perhaps also in copying appropriate social interactions, as well as being critical to understanding and predicting the behavior of others. Important MNS areas in the left hemisphere (there are corresponding areas on the right) include frontal near F5, the temporal pole, the temporal-parietal junction, and activities in the anterior insula and the anterior cingulate gyrus. Each area is postulated to have mirror functions that correspond to the functions of that area of the cortex. Mirror neurons have strong connections to the limbic system including the anterior cingulate (AC) (Iacoboni & Dapretto, 2006). The cingulate and the insular cortices both contain mirror neuron cells (Ramachandran & Oberman, 2006). A functional magnetic resonance imaging (fMRI) study demonstrated that activity of the MNS is correlated with empathic concern and interpersonal competence (Pfeifer, Iacaboni, Mazziatta & Dapretto, 2005). It has also been shown that children with ASD have reduced activity in MNS regions during tasks that require the child to mirror facial expressions of different emotions (Dapretto et al, 2006). The reason for reminding the reader of this system is that our findings with 19-channel QEEG and LORETA source locations show these areas to be almost consistently involved (>2SD from data base means from normal children) in our ASD clients (Thompson & Thompson, 2009).

At the Association of Applied Psychophysiology and Biofeedback (AAPB) meeting in April 2009, Jaime Pineda reported on his work using neurofeedback to increase 8 – 13 Hz activity at C4 in children with ASD. Increased activity in these frequencies was associated with improved facial recognition and it was posited that the training had an influence on the mirror neuron system in the right hemisphere. (The intervention was based on findings concerning lack of "Mu" suppression in children with ASD when they viewed videos of children moving their fingers and hands. Mu, which is measured at C3 and C4 across the motor strip, is found in conjunction with motor quiescence and is attenuated when there is movement of the contralateral hand, or thinking of moving the hand, or watching someone else moving their hands. Mu was not identified by its morphology in this study.)

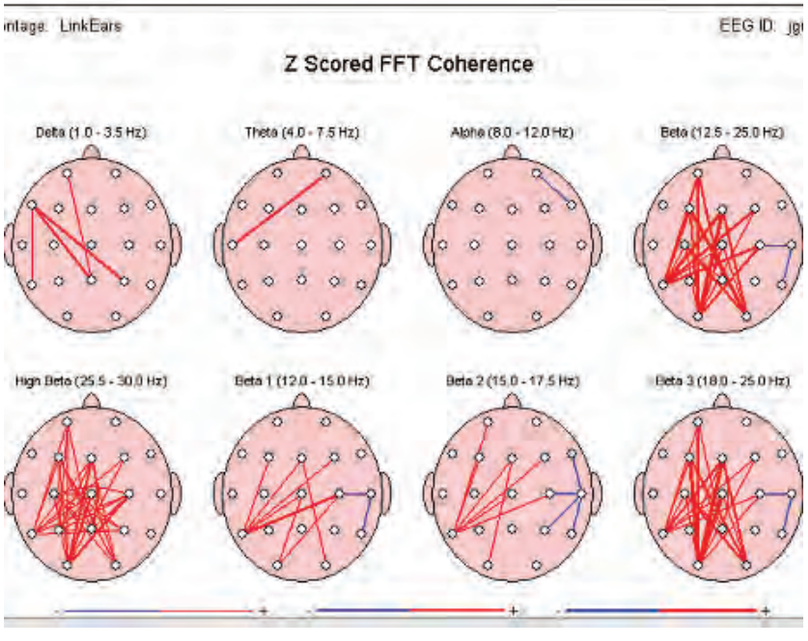


FIGURE 3: *Eyes open, linked ears montage. Deep red lines are >3SD from NeuroGuide data base mean for age and sex. Figure shows hyper-coherence between occipital, parietal, temporal and frontal regions. This shows mainly left hemisphere hyper-coherence but we see cases with autism that have only right hemisphere hyper-coherence and ASD cases where hypo-coherence is the dominant pattern. We must be very careful not to make attempts at this early stage to say the EEG can distinguish different subtypes of ASD.*

Theory of Mind (ToM) (Hill & Frith, 2003)**:** Theory of mind (which is sometimes more accurately called "theory of others' minds") involves the ability to "mentalize about both the self and others" (Abu-Akel, 2003). This model implicates the posterior brain (parietal and temporal regions) in representational thinking, the prefrontal regions for the application and execution of theory of mind, in addition to the medial prefrontal cortex (anterior paracingulate cortex), the temporal-parietal junction, and the temporal poles. ToM proposes that a fault in any component of these aspects of the social brain can lead to an inability to understand aspects of social communication. Intuitive understanding of others, especially understanding what they are feeling or thinking, has always been understood to be a core deficit of the autistic spectrum disorders (Thompson and Havelkova, 1983). The reader will note that these are also areas referred to in the above discussion of **mirror neurons.** The amygdala is also implicated (Adolphs, 2003; Adolphus et al, 2007) and the reader will see the overlap here with the **salience-landscape** theory (Ramachandran & Oberman, 2006). They also mention findings of less connectivity between the occipital and temporal regions and that is a finding that we observe using coherence (a measure of phase-locked connectivity) analysis in the EEG with these subjects (Figure 3).

Weak Central Coherence: The weak-central-coherence theory seeks to explain the need for 'preservation of sameness' and also the special interests and talents of those with AS. The child with ASD seems to be flooded

Continued on page 12

Autistic Spectrum Disorders
continued from page 11

with inconsequential details and/or, memories, without grasping the context or the Gestalt. Defensive behaviors may include rigidity, repetitive movements and obsessive and even preservative behaviors. Weak central-coherence probably involves a lack of appropriate connectivity between areas of the brain. Connectivity in this discussion refers particularly to connections between the posterior sensory processing areas of the brain (including lingual gyrus) and the frontal areas that modulate responses to the sensory input ('top-down' modulation). Hill states that one cause of this deficit could be a failure of normal developmental 'pruning' in early life that eliminates certain brain connections and optimizes the coordination of neural functioning. Resulting perceptual overload may, in turn, be partly responsible for their 'autistic' withdrawal. A reasonable, researchable hypothesis is that our findings of gross EEG coherence abnormalities with clients who have an ASD and our observations of symptomatic improvements corresponding to normalization of these coherence abnormalities corresponds to Hill's weak central coherence theory. Once the thalamus is properly gating the incoming sensory information (the result of sensory motor rhythm (SMR) training changing thalamo-cortical firing patterns) we hypothesize that the person may be less overwhelmed by sensory stimuli.

Executive Dysfunction: This third cognitive theory was advanced to explain features that do not appear to be subsumed under the former two theories. Executive functioning (including attention, planning, inhibition and mental flexibility) appears to be impaired in those with ASDs. The Tower of London Test (ToL) can evaluate many aspects of executive functioning and those with an ASD score poorly on this test. It requires the subject to inhibit immediate responses, plan, shift mental-set, use working memory, initiate a thought-out response and then monitor and evaluate the results of that response. The required cognitive functions all depend on good prefrontal functioning, and the prefrontal region is an area also seen to be outside EEG database norms in our clients with Asperger's. Improvement in performance on ToL has recently been reported in children with AS who received neurofeedback training (Knezevic, 2007). We hypothesize that these improvements could be related to our training over the left dorsolateral frontal cortex (a site midway between F3 and F7) and over the anterior cingulate (CZ).

Polyvagal Theory: Stephen Porges has noted that flat facial expression, poorly modulated tone of voice, and poor listening skills are related to the neural pathways that regulate the striated muscles of the face and head and that reduced muscle tone in this circuit correlates with less expressiveness in voice and face, less eye contact (eyelids droop), and slack middle ear muscles that makes distinguishing human voices from background noise more difficult. In addition, Porges has discussed the neurophysiological interactions between what he terms the Social Engagement System and the hypothalamic-pituitary-adrenal (HPA) axis, the neuropeptides of oxytocin and vasopressin, and the immune system (Porges, 2003, 2004, 2007). He has noted that training of myelinated vagal tone should be helpful in the ASDs (Porges 2003). We would add that this BFB "bottom-up" training synergistically combines with our NFB "top-down" training to potentially decrease a key symptom of anxiety in addition to other symptoms observed in ASDs, which we presented as a Systems Theory of Neural Synergy at the International Society for Neurofeedback and Research (ISNR) annual conference in 2008. We hypothesize that heart rate variability (HRV) training or, in the early days, just teaching effortless, diaphragmatic breathing, may have been important in our success with clients along the autistic spectrum and Porges' polyvagal theory provides a rationale for adding the biofeedback to neurofeedback.

Neurofeedback Training

As noted above, our understanding of the functional significance of different areas of the brain, as elucidated in the small booklet that correlates Brodmann areas to 10-20 sites (Thompson, Thompson, & Wu, 2008), combined with theories concerning brain dysfunction in those with ASD, as outlined above, and 19-channel QEEG assessment, sometimes combined with a stress assessment of psychophysiological variables, has led to interventions that combine EEG feedback with BFB. Normalizing the EEG can involve using one or two-channel NFB training, such as training at FCZ (to influence the AC) and at T6 (to decrease the sensory aprosodia symptoms). EEG training can be combined with BFB, usually HRV (Gevirtz, 2007). We usually increase sensorimotor rhythm (SMR), which may have a stabilizing effect on a cortex that is unstable and easily kindled (Sterman, 2000). When enhancing SMR be careful to identify a site on the sensory motor strip where there is no high amplitude spindling beta in the 13 – 15 Hz range. In addition to the low activity observed at T6, another factor that may, in future, prove to be a helpful 'marker' for ASD could be the 'mu' rhythm response. In ASD there is evidence of a reduction in mu rhythm suppression during action observation (Oberman, Hubbard, McCleery, Altschuler, Ramachandran, & Pineda, 2005). In our experience, however, mu is not observed in the majority of clients. Therefore using this as a training parameter for NFB, as suggested in an article in *Scientific American Mind* (Ramachandran & Oberman, 2006), would not be our initial approach, although Pineda has reported some success when 8 – 13 Hz was enhanced at C4. (Whether the training was increasing mu, alpha, SMR, or some combination of those brain waves is an interesting question that could be researched). Abnormalities in coherence are commonly found in those with ASD and training for normalizing connectivity between the parietal lobes and the temporal and frontal regions may prove to be beneficial. Coben (2005) has reported on normalizing coherence using single channel, sequential training of the sites found to be deviant on QEEG analysis. We have observed normalization after amplitude training, as Joe Horvat used to teach in his workshops. Some clinicians have reported on the emergence of difficult behaviors when treating purely based on QEEG z-score findings (James Thompson, personal clinical experience). This should not be surprising for some of the abnormal findings may be related to compensatory mechanisms. In addition, any professional well versed in psychosocial development in early childhood will recognize that these children are actually beginning to emerge from an arrested early development (Thompson & Patterson, 1986, Thompson, 1990). These factors and the importance of a staged approach to treatment that begins by working with the child's anxiety will be discussed in more detail in an upcoming chapter (Schwartz & Andrasik, in press). Briefly, our overall approach is to initially do neurofeedback with biofeedback to alleviate anxiety. Then the NFB parameters are changed to placements and frequencies designed to decrease impulsivity while increasing externally oriented attention span. This is a logical initial approach because

Continued on page 14

Biofeedback workshops for professionals... by professionals

Earn the most didactic hours for BCIA certification while learning from highly experienced instructors and receive hands-on training using the two leading biofeedback systems, NeXus and Infiniti®. Advance your career with the finest and most established workshops from Stens, now in our 33rd year and once again breaking new ground with cutting-edge NeXus wireless bluetooth technology.

5-Day Professional Biofeedback Certificate Program

Chicago, IL	July 18-22
San Francisco, CA	August 15-19
Austin, TX	September 19-23
Los Angeles, CA	October 24-28
Ft. Lauderdale, FL	November 7-11
San Francisco, CA	December 5-9

4-Day EEG Neurofeedback Certificate Program

Anaheim, CA	June 25-28
Chicago, IL	July 23-26
Austin, TX	September 24-27
Ft. Lauderdale, FL	November 12-15
San Francisco, CA	December 12-15

Stens CORPORATION

The leading supplier of biofeedback equipment and training

Call us at 800-257-8367 or register online at www.stens-biofeedback.com

CEs for APA, CNA, BBS. Stens Corp is approved by the APA to offer CE for psychologists and maintains responsibility for the program

Infiniti is a registered trademark of Thought Technology, Ltd.

Autistic Spectrum Disorders
continued from page 13

these are the difficulties that cause the client with an AS disorder to withdraw and / or to act out in situations that involve interacting with others. As sessions progress and the client becomes relaxed, calm, and focused, the NFB will be combined with cognitive and social strategies which place more emphasis on executive functions and social awareness. The sites for training and the frequencies used will correspond to the brain areas involved in these processes and the QEEG assessment findings.

Remember that other interventions are typically being used in conjunction with NFB, such as speech and language training, dietary approaches, and other behavioral training. At a center treating children with autism in Edison, New Jersey, they have documented significant improvements after hyperbaric oxygen treatments (James Thompson, personal observations of clinical outcomes at this center). For each individual client, the right mix of interventions has to be created for optimal improvement.

Conclusion

Over the past dozen years, a few papers and presentations about interventions using neurofeedback for clients with AS have appeared (Coben, 2005, 2006, 2007; Jarusiewicz, 2002; Linden, 2006; Reid, 2005; Solnick, 2005; Thompson & Thompson, 1995, 2003, 2006, 2007, 2009). These papers all note favorable clinical outcomes in case series, some with large numbers of cases, such as 159 clients reported on at the Biofeedback Foundation of Europe annual meeting in 2009 (Thompson & Thompson, in press). Of particular interest for NFB providers are findings that EEG differences, augmented by LORETA analysis of QEEG data, identify many of the key areas reported in the literature to be abnormal in those with ASD (Thompson & Thompson, 2009). Since neuroanatomical differences reported in numerous studies parallel EEG differences, it provides an impetus for systematic investigation of the question of whether correcting amplitude, phase and connectivity differences (identified through comparisons with a normative data base) can lead to normalization of symptoms in people who have diagnoses along the autistic spectrum. Other interesting research questions involve the efficacy of combining NFB with BFB. The latter approach is supported by Stephen Porges' demonstrating a relationship between vagal nerve output and symptoms observed in ASD. There does appear to be a strong rationale for combining neurofeedback and biofeedback, particularly heart rate variability training, since both promote calmness and better self-regulation, which are pre-conditions for successful social interactions.

References

Abu-Akel, A. (2003). A Neurobiological Mapping of Theory of Mind. *Brain Research Reviews, 43*, 39-40.

Adolphs, R. (2003). Is the human amygdala specialized for processing social information? *Annals of the New York Academy of Sciences, 985*, 326.

Adolphs, R., Gosselin, F., Buchanan, T.W., Tranel, D., Schyns, P., Damasio, A.R., (2005). A mechanism for impaired fear recognition after amygdala damage. *Nature 433*, 68.

American Psychiatric Association. (2000) *Diagnostic and statistical manual of mental disorders - 4th edition – Text Revision*. Washington, DC.

Asperger, H. (1944). Die "Autistischen Psychopathen" im Kindesalter. *Archiv fuer Psychiatrie und Nervenkrankheiten, 117*, 76-136. English translation (1991) Autistic psychopathy in childhood. In U. Frith (Editor and Translator) *Autism and Asperger's syndrome*. Cambridge, United Kingdom: Cambridge University Press. 37-92.

Bachevalier, J. & Loveland, K. A., (2006). The orbitofrontal-amygdala circuit and self-regulation of social-emotional behavior in autism. *Neuroscience and Behavioral Reviews, 30*, 97-117.

Chatrian, G.E., Lettich, E., Nelson, P.L. (1985). Ten percent electrode system for topographic studies of spontaneous and evoked EEG activity. *American Journal of EEG Technology, 25*, 83-92.

Coben, R. (2005). Assessment guided neurofeedback for autistic spectrum disorder. Presentation at the Society for Neuronal Regulation 13th annual conference, Denver, Colorado, September.

Coben, R. (2006) Panel on autistic spectrum disorders at the 36th annual meeting of the Association for Applied Psychophysiology and Biofeedback, Portland OR.

Coben, R. (2007). Connectivity-guided neurofeedback for autistic spectrum disorder. *Biofeedback, 35*(4), 131-135.

Dapretto, M., Davies, M. S., Pfeifer, J. H., Scott, A. A., Sigman, M., Bookheimer, S. Y., & Iacoboni, M. (2006). Understanding emotions in others: mirror neuron dysfunction in children with autism spectrum disorders. *Nature Neuroscience, 9*(1), 28-30.

Devinsky, O., Morrell, M., & Vogt, B. (1995). Contributions of Anterior Cingulate Cortex to behavior, *Brain, 118*, 279-306.

Gevirtz, R. (2007). Biofeedback Training to Increase Heart Rate Variability. In Lehrer, Paul M.., Woolfolk, Robert L., & Sime, Wesley E. (Eds.) *Principles and Practice of Stress Management, 3rd Edition*. New York: Guilford Publications.

Green, V. A., Pituch, K. A., Itchon, J., Choi, A., O'Reilly, M., & Sigafoos, J. (2006). Internet survey of treatments used by parents of children with autism. *Research in Developmental Disabilities, 27*, 70-84.

Hill, E. L. & Frith, U. (2003). Understanding Autism: Insights from mind and brain. Theme Issue *'Autism: mind and brain'. Phil. Trans. of The Royal Society London*, Bulletin 358, 281-289.

Iacoboni, M. & Dapretto, M. (2006). The mirror neuron system and the consequences of its dysfunction. *Nature Reviews and Neuroscience, December 2006*, 942-951.

Jarusiewicz, E. (2002). Efficacy of neurofeedback for children in the autistic spectrum: A pilot study. *Journal of Neurotherapy, 6*(4), 39-49.

Knezevic, B. (2007). Pilot Project to Ascertain Utility of Tower of London Test (ToL) to Assess Outcomes of Neurofeedback in Clients with Asperger's Syndrome. Student Award paper presented at the International Society for Neurofeedback and Research, Annual Meeting, San Diego, CA.

Linden, M. (2006) Panel on autistic spectrum disorders at the 36th annual meeting of the Association for Applied Psychophysiology and Biofeedback, Portland OR.

Macintosh, K. E & Dissanayake, C. (2004). Annotation: The similarities and differences between autistic disorder and Asperger's disorder: A review of the empirical evidence. *Journal of Child Psychology & Psychiatry. 45*(3), 421-434.

Martinez, Y. (2003). Unpublished thesis done as part of the requirements for an Honours B.A. degree. Copy on file at the ADD Centre, Toronto.

Neuroguide Delux, 2.3.7 (2007). Robert Thatcher, Applied Neuroscience Inc. (www.appliedneuroscience.com)

Oberman, L. M., Hubbard, E. M., McCleery, J. P., Altschuler, E. L., Ramachandran, V. S., & Pineda, J. A. (2005). EEG evidence for motor neuron dysfunction in autistic spectrum disorders. *Brain Research & Cognitive Brain Research, 24*, 190-198.

Pascual-Marqui, R. D., Esslen, M., Kochi, K., & Lehmann, D. (2002). Functional Imaging with Low Resolution Electromagnetic Tomography (LORETA): A review. *Methods & Findings in Experimental & Clinical Pharmacology, 24C*, 91-95.

Pfeifer, H., Iacoboni, M., Mazziotta, C., & Dapretto, M. (2005). Mirror neuron system activity in children and its relation to empathy and interpersonal competence. In Abstract Viewer/Itinerary Planner. *Soc. Neurosci. Abstr. 660*(24).

Porges, S. W. (2003). Social engagement and attachment: A phylogenetic perspective. *Annals of the New York Academy of Sciences, 1008*, 31-47.

Porges, S. W. (2004). The Vagus: A mediator of behavioral and physiologic features associated with autism. In M. L. Bauman and T. L. Kemper (Eds.) *The Neurobiology of Autism*. Baltimore: Johns Hopkins University Press, 65 – 78.

Porges, Stephen W. (2007). The Polyvagal Perspective. *Biological Psychiatry, 74*, 116-143

Ramachandran, V. S. & Oberman, L. M. (2006). Broken Mirrors. *Scientific American, 295*(5), 62-69.

Reid, A. (2005). Autistic Spectrum Disorders: Assessment and Intervention Results after Neurofeedback in 146 Cases. Student Award Presentation, International Society for Neuronal Regulation annual meeting. Denver, Colorado, September, 2005.

Ross, E. D. (1981). The Aprosodias: Functional-Anatomic Organization of the Affective Components of Language in the Right Hemisphere. *Archives of Neurology, 38*, 561-569.

Shamay-Tsoory, S. G., Tomer, R., Berger, B. D., Goldsher, D., & Aharon-Peretz, J. (2005). Impaired "Affective Theory of Mind" is associated with right ventromedial prefrontal damage. *Cognitive & Behavioral Neurology. 18*(1), 55-67.

Simpson, D. (2004). Asperger's syndrome and autism: Distinct syndromes with important similarities. In Maria Rhode & Trudy Klauber (Eds) *The many faces of Asperger's syndrome. The Tavistock Clinic Series. xviii, 302*, 25-38. London, England: Karnac Books.

Sloman, L. (2005). Medication use in children with high functioning pervasive developmental disorder and Asperger syndrome. In K. Stoddart (Ed.) *Children, Youth and Adults with Asperger Syndrome: Integrating multiple perspectives*. London: Jessica Kingsley Publishers.

Solnick, B. (2005). Effects of electroencephalogram biofeedback with Asperger's syndrome. *International Journal of Rehabilitation Research, 28*(2), 159-163.

Sterman, M. B. (2000a). Basic concepts and clinical findings in the treatment of seizure disorders with EEG operant conditioning. *Clinical Electroencephalography, 31*(1), 45-55.

Thompson, M. and Thompson L. (in press). Autistic Spectrum Disorders and Neurofeedback combined with Biofeedback. In Mark S. Schwartz, & Frank Andrasik (Editors) *Biofeedback: A Practitioner's Guide (4th edition)*

Thompson L. & Thompson M. (1995). Exceptional Results with Exceptional Children. *Proceedings of the Society for the Study of Neuronal Regulation*. Annual Meeting: Scottsdale, Arizona.

Thompson, L. & Thompson, M. (2006). Panel on Autistic

Continued on page 22

BrainMaster lets you serve clients quickly, easily, and affordably

WHAT MAKES BRAINMASTER THE WISE CHOICE?

The answer is simple. We create the products you want and need to help your clients and improve your practice. When you can obtain the latest and most-sought hardware and software, all invented, designed and created by the same manufacturer, think how much your learning curve is reduced. 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 consistent world-wide leader in EEG biofeedback. We upgrade and improve our products continually. Over the years, we have brought you the Atlantis, the MINI-Q, Live Z Score Multi-Variate Training,* supervised web-based remote training capability, 3.0 software, MultiMedia Player Package, Real World Real Time Training with the BrainTrack, and now the Discovery QEEG. Of course, we 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 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 so through an affiliate corporation (Stress Therapy Solutions, Inc.) we offer a broad range of courses and training in the how, why, and when of EEG and peripheral biofeedback, bringing you acclaimed authors, instructors, and industry leaders.

When it comes to EEG, QEEG, and general biofeedback, there is only one choice. BrainMaster Technologies, Inc.

*pat.pend.

NEW!

DISCOVERY™

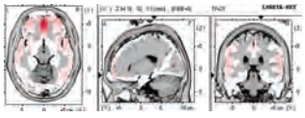
The Discovery 24E 24-channel EEG is an outgrowth of the same technology used in the Atlantis series of EEG and peripheral biofeedback devices. It provides up to 24 channels of 24 bit, DC-coupled EEG using a standard electrode cap and cables, and includes auxiliary channels for EEG external signals, peripheral biofeedback, or system expansion. Compatible with industry-standard software including NeuroGuide, SKIL, WinEEG, and other packages that read standard European Data Format (EDF) files. Direct, real-time interface to NeuroGuide also available.

ATLANTIS™

Atlantis continues to be a leader in EEG and peripheral biofeedback. Up to 10 channels of EEG including DC and Slow Cortical Potentials with no additional hardware. General biofeedback options presently include temperature, near infrared hemoencephalography (HEG), passive HEG, photo-oximetry, and heart rate feedback. Fast, accurate 24-bit acquisition, simultaneous continuous impedance monitoring, in a safe, reliable magnetically and optically isolated USB-powered device. Flexible training protocols enable feedback using multimedia, DVD, or CD feedback, MIDI sounds, or Flash Games. Control external devices using the BrainTrack interface, for BCI (Brain-Computer Interface) development.

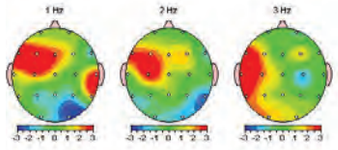
DC AND SLOW CORTICAL POTENTIALS

Monitor, record, and train up to 4 channels of DC and Slow Cortical Potentials using standard sensors and leads. Flexible training protocols enable feedback using multimedia, DVD, or CD feedback, MIDI sounds, or Flash Games. Control external devices using the BrainTrack interface, for BCI (Brain-Computer Interface) development.



LIVE Z-SCORE TRAINING

BrainMaster is continuing to evolve and advance Live Z-Score Training. BrainMaster introduced this method first, more than 2 years ago, and has continued to develop advanced methods. Only BrainMaster provides flexible training of multiple Z-score targets, target biasing, and automated review of up to 248 Z-scores from 4 channels.



SUPPORT TEAM

Our support team is available to help you make everything work smoothly. Just call or visit our website and KnowledgeBase.

AFFILIATE MEMBERSHIP

This is your opportunity to reduce your expenses and expand your influence. Your Affiliate Membership offers you free website advertising, technical support, warranty, list server, preferred discounts, workshop discounts, and more.

REMOTE TRAINING

Our product line, ***designed and manufactured in the USA***, is the only one that allows clinician-controlled, website-based, remote training.

BrainMaster Technologies, Inc.
From the Decade of the Brain, Into the New Millennium

195 WILLIS STREET
BEDFORD OH 44146
440-232-6000, ext 200, 201
SALES@BRAINMASTER.COM
WWW.BRAINMASTER.COM

BRAINMASTER TODAY.
WHERE THE INDUSTRY WILL BE NEXT YEAR.

Jonathan E. Walker, M.D.

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

---

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

Neurotherapy Center of Dallas

12870 Hillcrest, Suite 201, Dallas, Texas 75230

Call us: 972-991-1153, Ext. 14
Email us: admin@neurotherapydallas.com
Visit our Web site: www.neurotherapydallas.com

EEG Assessment and Treatment of Seizures in Children with Autism Spectrum Disorder: A Case example

Robert Coben Ph.D. and Kevin McKeon M.A.

Autism is a neurodevelopmental disorder with possible genetic and environmental influences. The Center for Disease Control and Prevention indicates that the current prevalence of the Autism Spectrum Disorder (ASD) is 1 in 150 (CDCP, 2007). Furthermore, the U.S. Department of Education reported that from the 1992-1993 to 2001-2002 school years the rate of Autism increased 528%. Monetarily, in the United States approximately $3.2 million is spent to care for a single individual with Autism over the course of his or her lifetime, which in turn equates to a total cost of $35 billion annually. Beyond the financial costs, countless other collateral effects are felt by family members and caregivers of those with ASD. Parents with an Autistic son or daughter have been found to have increased complaints of anxiety disorders, higher instances of obsessive-compulsive illness, and poorer sleep quality as well as quantity. Children with a sibling with ASD have been found to have more behavior disturbances, increased reports of loneliness, and suffer more peer related problems. These detrimental ancillary effects compounded with the financial burdens associated with this disorder serve to provide clear evidence that ASD is an apparent problem in society warranting increased treatment efficacy research.

Seizures and Autism

Multiple neuroimaging studies have demonstrated brain anomalies in autistics compared to healthy controls (McAlonan et al., 2004; Page et al., 2006). Consistent with this, seizures and epilepsy have been commonly observed in autistic samples. Recent analyses have estimated the prevalence of seizure disorders in autistic series at anywhere from 20% to 46%. Based on recent analyses, the prevalence of seizure disorders in autistic series is estimated at about 36% (Danielsson, Gillberg, Billstedt, Gillberg, & Olsson, 2005; Hara, 2007; Hughes & Melyn, 2005; Parmeggiani et al., 2007). In fact it has been reported that the autistic population has about 3 to 22- fold increased risk of developing seizure disorders as compared to the normal population (Volkmar & Nelson, 1989). Increasing cognitive/intellectual disability appears to be associated with seizure disorders in Autism. Sub-clinical seizure activity or paroxysmal discharges occur in an even higher proportion of autistics, but the significance of these remain uncertain (Hughes & Melyn, 2005; Parmeggiani et al., 2007). Ray, Tao, Hawes-Ebersole, and Ebersole (2007) have suggested that the initial phase of cortical spikes may relate to underlying intracranial foci. Other work has suggested that EEG spikes may reflect underlying morphological brain abnormalities (Shelley, Trimble, & Boutros, 2008) and/ or metabolic disturbances (Kobayashi, Bagshaw, Grova, Dubeau, & Gotman, 2006).

Recent estimates suggest that approximately one-third of all autistic children experience a regression in speech or behavior early in life (Canitano, 2007). Tuchman and Rapin (1997) were unable to relate early regression to seizure disorders, but suggested that the EEG is abnormal in a greater proportion of autistic children that regress than those that do not. Abnormal electroencephalogram (EEG) recordings are also present in the majority of autistic children with seizure disorders (Hughes & Melyn, 2005). For these reasons, experts in the field have recommended the use of routine and sleep EEGs in the evaluation of autistic disorders, especially when there has been regression or there are signs of possible seizures.

EEG Assessment

The electroencephalogram (EEG) is a premiere tool to assess neural dysfunctions due to its non-invasive nature, availability and temporal resolution. Moreover, when EEG assessment is processed and analyzed with

Continued on page 18

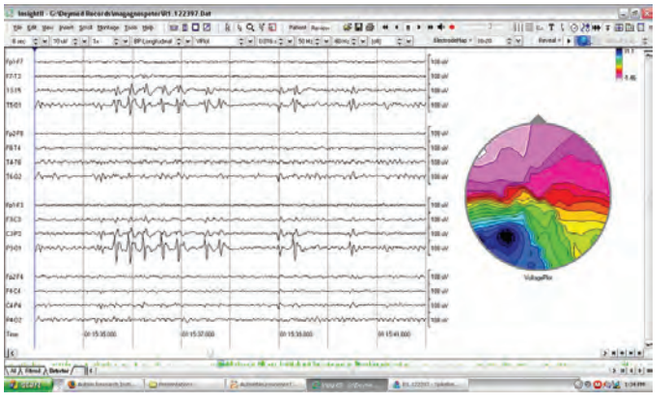


FIGURE 1. *Spike and wave pattern detected by the Persyst/reveal–spike and seizure detection system (longitudinal montage) with voltage mapping.*

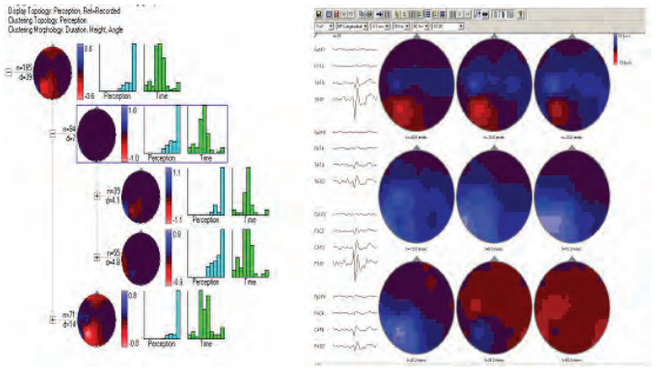


FIGURE 2. *Summary of spike reveal/reveal with component mapping, time, and perception (left side of figure). Spike review propagation mapping (right side of figure).*

EEG in Children with Autism Spectrum

continued from page 17

the most advanced techniques it can be invaluable in screening for possible seizure activity. In fact, seizure detection has been the primary role of the EEG for decades.

The following exemplar is presented as a demonstration of the utility of such an assessment. This patient presented to our clinic in 2006 and was diagnosed by a pediatric neurologist with Pervasive Developmental Disorder-Not Otherwise Specified at the age of 3. He was delivered at 36 weeks gestation due to gestational diabetes and had high liver enzymes. There was no history of regression, but a developmental delay in speech/language abilities and a complete inability to identify letters or read words. At the time of his assessment he was taking no medication, was receiving speech, occupational, and physical therapies at school, and his intelligence was considered low average (WISC-IV FSIQ = 80). A clinical EEG (resting) conducted in our clinic at intake resulted in the following. Resting eyes closed and open data was acquired with the Deymed 32-True Scan acquisition system (Deymed Diagnostic, 2004) and viewed/analyzed with the Persyst/Reveal–spike and seizure detection system

(Persyst, 2004). This included 19 cephalic, ground, and reference channels. Figure 1 shows 8 epochs (seconds) of data during which the patient clearly evidenced paroxysmal activity consistent with a focal spike and wave pattern (Fisch, 1999; Goldensohn, Legatt, Koszer, & Wolf, 1999). On the right side of this figure is a voltage topographic map suggestive of a left occipital-parietal-temporal localization.

Figure 2 is the spike review of all detected events seen during this single EEG recording. There were a significant 165 events detected over the 20 minute recording time. Analysis of individual components of such activity showed five separate patterns with similar areas of morphological disturbance. These all involve the left posterior region likely including regions near the left occipital and left temporoparietal junction. The spike review propagation maps show a deactivation followed by a hyperactivation of this region which is often associated with seizure activity. Interestingly, these regions of the brain are responsible for some of his difficulties including comprehension, speech/language functions and his alexia (inability to read).

EEG Biofeedback as a Treatment for Seizures

Human EEG biofeedback was first attempted in the 1960s by Joe Kamiya at the University of Chicago (Kamiya, 1969). Early investigations focused on operant conditioning of alpha brain waves primarily to facilitate deep relaxation and meditation. Subsequently, M. Barry Sterman at UCLA developed a form of EEG biofeedback involving the training of the sensorimotor rhythm (SMR) (12-15 Hz frequency range) developed from operant conditioning of cats' EEG. Sterman and colleagues found that cats trained in the middle to high frequency range from a previous unrelated experiment had greater latency to seizure onset, and a higher threshold for seizure onset, than those of untrained cats. These results were then replicated in humans in some twelve research centers, encompassing over twenty studies. Finley et al. (1975) found evidence supporting these findings by reporting that training humans in the way prescribed by Sterman resulted in reduced seizure frequency in even the most severely epileptic patients. In 2000, Sterman performed a meta-analysis of 30 years of research conducted on epilepsy. The results showed that 82% of subjects achieved clini-

cal improvements with about 60% showing significant reductions in seizures intensity and frequency (Sterman & Egner, 2006).

More recently, Walker and Kozlowski (2005) have proposed a novel approach to epilepsy that utilizes EEG biofeedback focused on reducing abnormalities of power and coherence in an attempt to decrease and potentially eliminate seizure likelihood. Prior to this investigation no studies of coherence abnormalities in patients with epilepsy had been conducted. The basis of this approach is to tackle the most statistically abnormal power or coherence anomalies first, followed by training of successively less severe abnormalities in the latter course of treatment. Moreover, these abnormalities are located by use of advanced statistical techniques, including methods such as Quantitative EEG analysis (QEEG). Walker & Kozlowski (2005) reported a successful case of a 31-year-old man with recurrent partial complex and secondary generalized seizures with frequent episodes of status epilepticus. At the time of assessment the patient was on nine different anticonvulsant medications and was not a candidate for surgery due to the independence of the foci of onset in the left and right temporal lobes. Further, a vagal nerve stimulator was previously implanted which provided little relief. A QEEG revealed several neural anomalies and each was trained by order of severity. Training was performed three times per week and by the end of the first protocol there were no further episodes of status epilepticus. Additionally, generalized seizures were limited to one episode per week. By the completion of treatment all seizures had ceased. Further, both the patient's speech and memory improved and he returned to working full time and began to drive again.

Case Study

To begin the process of determining a neurofeedback treatment plan for the subject noted above we further analyzed his EEG with various unique quantitative methods. While quantitative EEG usually focuses on artifacting records so as to eliminate non-eeg related signals and spike or seizure events, we have started performing a unique type of analysis. In this approach, we highlight all instances in which seizure activity occurred from the original EEG recording, and then merge these separate pieces of data together for analysis via quantitative methods. In this case study, we processed his EEG in this fashion. Once all EEG data that included spike events was compiled we were then left with 2 minutes and 22 seconds of analyzable data from an approximately 15 minute original recording. These data were then analyzed for absolute power values across various EEG frequencies as shown in Figure 3. This analysis revealed large elevations within the delta and theta bands in the left posterior region mainly over electrode sites O1, T5 and PO3. These low frequency elevations correspond to the slow wave component and the spike and wave complex. Similarly, there were also elevations in the alpha and beta bands from about 8-15 hz. which correspond to the spike component of this paroxysmal activity.

The next steps in this data analysis were designed to understand the contribution of coherence analyses to these power elevations. We processed this same data set with the NeuroRep coherence analysis system (Hudspeth, 2009). This system uses pairwise coherence data and through a principal components statistical analysis converts the data into 3-dimensional multivariate coherence eigen images. The analysis for this case is shown in Figure 4 with only the horizontal views displayed.

This analysis revealed low coherence in the left hemisphere over all frequency bands, most prominently in the delta range. There appears to be diminished coherence at these low frequencies that spans almost the entire left posterior quadrant of his brain. Importantly, this finding indicates that at the time when these spike and wave episodes occur they are associated with low

Continued on page 21

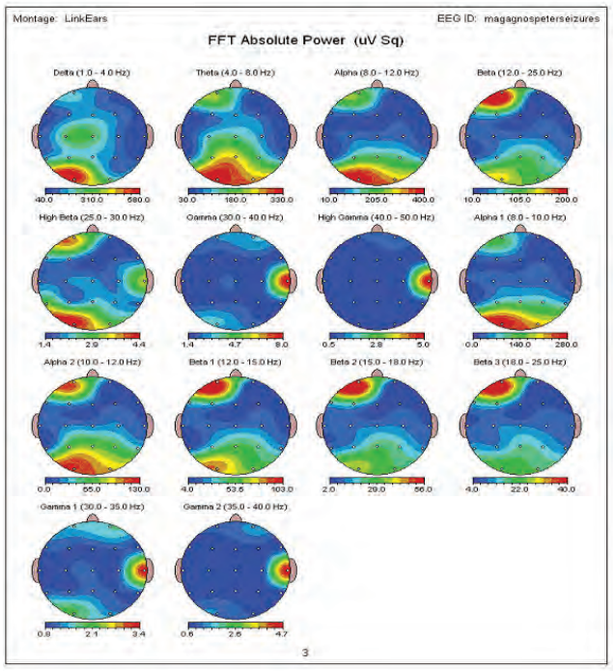


FIGURE 3. *QEEG analysis of Absolute Power for this patient's EEG data composed of spike and wave paroxysmal discharges.*

FIGURE 4. *Horizontal Eigen images in delta, theta, alpha, beta and total frequencies based on NeuroRep coherence analysis.*

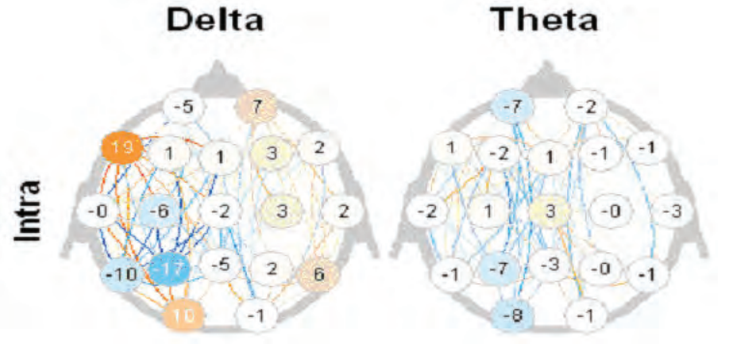


FIGURE 5. *Findings from Compare for differences in delta and theta coherences comparing high – low spike and wave events processed in Spectral Sort for O1/1-4 hz/absolute power.*

OchsLabs, Inc. is pleased to announce: The LENS is now FDA-certified as a Class-II Medical Device 510-K Exempt.

Foundations Trainings

Aug 31-Sept 2, 2009 by Cathy Wills R.N.,M.S.N., C.N.S. Indianapolis ISNR. Registration through www.ISNR.org.

Sept 25-27, 2009 by Cathy Wills R.N.,M.S.N., C.N.S. Rockville, MD. Registration through www.ochslabs.com or (707) 823-6225.

October 9-11, 2009 by Cathy Wills R.N.,M.S.N., C.N.S. and Len Ochs, Ph.D. Sebastopol, CA. Registration through www.ochslabs.com or (707) 823-6225.

January 15-17, 2010 by Cathy Wills, R.N., M.S.N., C.N.S. and Len Ochs, Ph.D. Sebastopol, CA. Registration through www.ochslabs.com or (707) 823-6225

Advanced Trainings

Aug 31-Sept 2, 2009 by Len Ochs, Ph.D. Indianapolis ISNR. Registration through www.ISNR.org.

Sept 25-27, 2009 by Len Ochs, Ph.D. Rockville, MD. Registration through www.ochslabs.com or 707-823-6225.

November 13-15, 2009 by Len Ochs, Ph.D. Sebastopol, CA. Registration through www.ochslabs.com or (707) 823-6225.

December 4-6, 2009 by Len Ochs, Ph.D. New Paltz, NY. Registration through 845-658-8083.

The LENS system can **dramatically decrease the number of treatments for many of your clients.** You can learn more about the Low Energy Neurofeedback (LENS) approach in *The Healing Power of Neurofeedback*, by Stephen Larsen, and issue 10/3-4 of the *Journal of Neurofeedback* devoted entirely to the LENS. OchsLabs, Inc. offers a number of trainings throughout the year for both the beginner and the experienced practitioner. The LENS Foundations Training is designed to teach assessment, introductory treatment planning, and software operation. The LENS Advanced Training is designed to teach advanced assessment, reassessment, and treatment planning.

OchsLabs, Inc 503 South Main St., Sebastopol, CA 95472 www.ochslabs.com (707) 823-6225

Z-Score™
Mini Suite

Z-Score Biofeedback Made Easy

Thought Technology's products for z-score assessment and training combine to create the most easy to use, powerful, and flexible Z-Score Biofeedback system on the market.

Try it today, and find out why BioGraph® Infiniti, now with Z-Score Biofeedback Technology, is the most widely used biofeedback software in the world.

Simple Z-Score Assessment & Training

Everything you need for 2 and 4-channel Z-Score Biofeedback

- **No more guessing.** Train using simple indeces that report on multiple z-score values simultaneously. Use any of 4 preset indeces, modify them easily or make your own.
- **Measure standard bands of EEG** activity at the same time.

Quick Start Icon

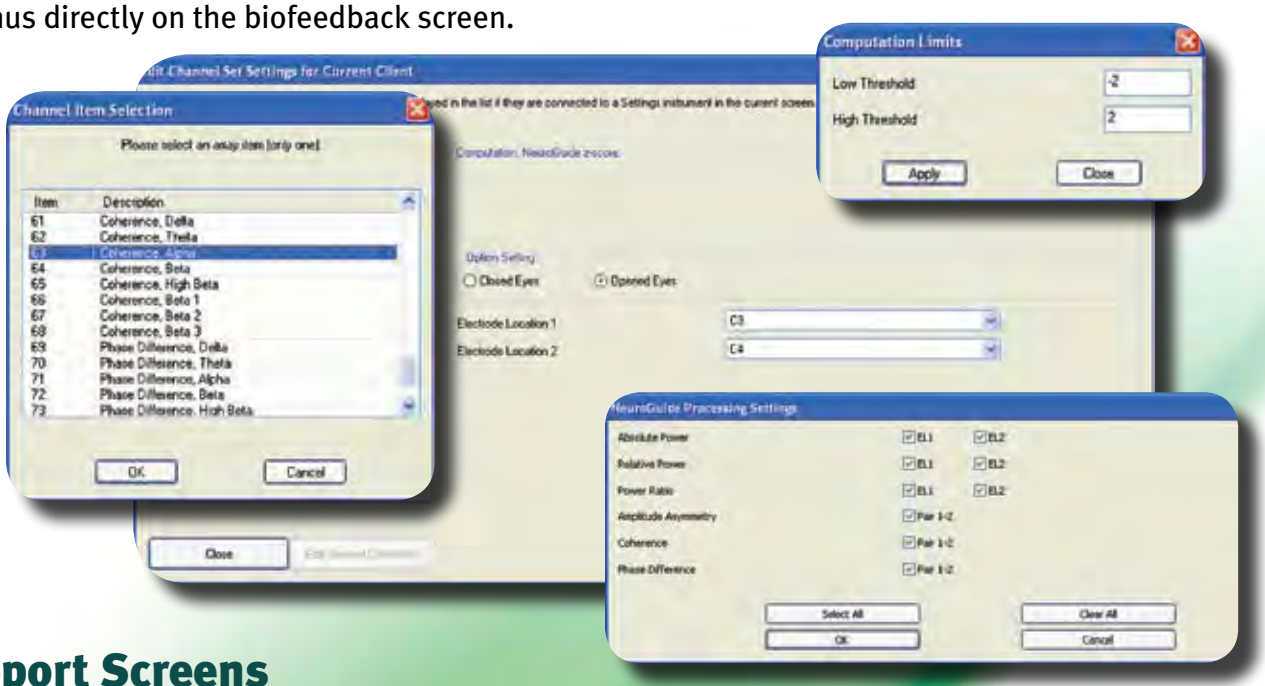
Try the new **one-button session loading** that includes everything you need:

- Assessment screens, training screens, statistics, and review screens.
- Access all this directly via a single desktop icon!

- Use **bright, colorful screens** that include popular BioGraph® Infiniti features such as single and dual monitor displays, DVD, animations & sound feedback, and powerful real-time trending of means in-session and post-session.

Easy Access to Settings

Enjoy easy access to simple Graphical User Interfaces. Access graphical settings via right click menus directly on the biofeedback screen.



Report Screens

Review and report using screens that include efficient graphical representation of session trends.

BioGraph® Infiniti Platform

- **Stability.** BioGraph® Infiniti is the most reliable and accurate software in the biofeedback market. Never lose data again, with our exclusive crash-proof technology and pause-on disconnect features.
- **Flexibility.** A simple right click allows you to change settings on the fly - such as bandwidths, sound feedback and animations. Observe changes in other physiology, using any of our other sensors by customizing your screens with our FREE Developer Tools.

Electrode kits tailored for Z-Score Biofeedback

... and ready for all your EEG applications

Thought Technology's new line of EEG electrodes delivers high quality performance, ease-of-use and maximum flexibility. Use efficient 2 and 4-channel kits designed for Z-Score Biofeedback.

- All electrodes are available in gold or sintered silver-silver/chloride*. Several colors available!
- Ready-to-use kits have been designed with your applications in mind and allow for multiple connection configurations
- Individual cables are also available to create any configuration you like
- All cables feature standard DIN connectors for compatibility with Infiniti and other EEG systems
- Kits include new reference ear clip.
  - * Comfortable, sturdy easy to apply and quick to clean. Includes standard DIN connector for use with any EEG equipment.
  - * Also available separately as a useful add-on to third party electrode caps!

Two and four-channel connectivity kits

- Apply fewer electrodes and record more accurately
- Use single or linked ears reference, common ground and multiple configurations.
- All with the same cable set.
- Ideal for Z-Score Biofeedback!

* Required for all DC recordings to minimize electrode offset and drift.

Thought Technology Ltd.
Tel: 1-800-361-3651 • 514-489-8251 Fax: 514-489-8255
http://www.thoughttechnology.com

MAR937-00

EEG in Children with Autism Spectrum
continued from page 19

coherence or synchronization of brain activity over these regions. Interestingly, these are the same regions of the brain where the spike and wave events were manifested.

This same data was then analyzed in the program Spectral Sort (SS), a component of the NeuroRep qEEG software suite. Spectral Sort is designed to test the hypothesis that large amplitude EEG signals arise because of failed connectivities. Thus, when EEG signals range between small and very large amplitudes, there are correlated changes in connectivities that serve to modulate amplitude. SS traverses through the time history of an EEG recording, computing the FFT magnitude, in a specific frequency range at specific electrode sites. The sample voltages are sorted by amplitude and then a median value is computed. Two new recordings are extracted from the original recording: one is based on epochs in which the sorted values are larger than the median and the second is based on the epochs less then the median. The two records are then subjected to coherence analysis and the connectivity metric is then put though statistical analyses to discover the significance of any observed coherence differences between these two files or conditions. For this case presentation, his EEG file composed only of spike and wave events, was analyzed in SS highlighting the O1 electrode site for the frequency between 1 – 4 hz. in absolute power. The median value over this site was 432.4 uv/sq. with epochs above this value being placed into a "high" record and epochs below this value being placed into a "low" record. These two records, high and low, were then analyzed in the NeuroRep program Compare for differences in power and coherences with a special emphasis on the latter. Figure 5 shows the results of this analysis for delta and theta coherences between when these paroxysmal discharges are larger vs. when they are smaller. These findings show a decrease in coherence over O1, T5, and P3 when these spike and wave events become larger or are released or discharged. This coincides with the same regions of the brain where these events are observed and correlates with many of his symptoms.

Based on these analyses of his paroxysmal activity a neurofeedback treatment plan was designed. This involved two-channel coherence training between electrode sites O1 and T3; active sensors were placed over these sites referenced to his left ear and grounded on the right ear. Coherence was rewarded for increases from 1 – 7 hz. and amplitude inhibits were applied simultaneously over these same regions for frequencies including 1 – 4 hz. and 8 – 13 hz. These latter values correspond to the amplitude values of the spike and wave events themselves. The hypothesis underlying this training was that these coherence anomalies are what lead to these losses of control with paroxysmal discharges being the outcome. As a result, the goal of treatment was to enhance coherence across the region of the brain involved in these paroxysmal events with the idea that this would allow for greater neuronal regulation and diminished seizure activity. This treatment continued with gradual improvements evident over a period of approximately one year.

By the completion of these sessions the subject's neuropsychological findings were all within normal limits for his age, including receptive language skills which were previously impaired. His reading ability had progressed to within one year of his age, parents ratings of autistic symptoms diminished as did his seizure activity. As may be seen in Figure 6, epileptiform paroxysmal discharges decreased from 165 to practically none over the duration of treatment. He was not taking medications to reduce such activity and it is presumed that the neurofeedback training was responsible for these changes. During the same time frame, his parents' ratings of his autistic symptoms decreased by approximately 82% and at the completion of his sessions his level of symptoms would be considered to be within the normal range of behaviors.

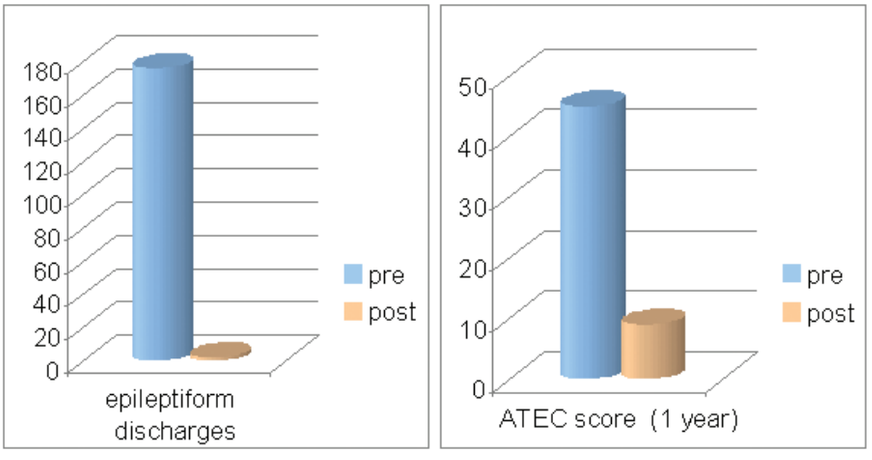


Figure 6. Changes in epileptiform/paroxysmal (left side of figure) discharges and parents ratings of autistic symptoms (right side of figure) before and after neurofeedback training.

Summary

Research indicates seizures and subclinical paroxysmal discharges to be prevalent in autistic disorders. This case presentation was provided as an exemplar for how EEG data may be analyzed in a way to detect, conceptualize and provide impetus for intervention related to these complex difficulties. Through such an application, EEG biofeedback may be utilized to treat seizure activity in children with ASD over a short treatment span and without significant side effects. Clearly, more empirical data is needed to validate this approach as an evidence-based form of intervention for seizures in ASD.

References

Canitano, R. (2007). Epilepsy in autism spectrum disorders. European Child and Adolescent Psychiatry, 16(1), 61-66.

Centers for Disease Control and Prevention (2007). Prevalence of the autism spectrum disorders (ASDs in multiple areas of the United States, 2000 and 2002). MMWR, 56 (SS 1–2).

Danielsson, S., Gillberg, I.C., Billstedt, E., Gillberg, C., & Olsson, I. (2005). Epilepsy in young adults with autism: A prospective population-based follow-up study of 120 individuals diagnosed in childhood. Epilepsia, 46(6), 918-923.

Deymed Diagnostic (2004). TruScan 32 Specifications. Retrieved March 24, 2006, from http://www.deymed.com/truscan32.asp.

Finley, W.W., Smith, H.A., Etherton, M.D., Andrews D.J., Schonfeld, W.H., & Ellerston, B. (1975). Reduction of seizures and normalization of the EEG in a severely epileptic following sensorimotor biofeedback training: preliminary study. Biological Psychology, 2(3), 189-203

Fisch, B. J. (1999). Fisch and Spehlmann's EEG Primer: Basic principles of digital and analog EEG (3rd ed.). New York: Elsevier.

Goldensohn, E. S., Legatt, A. D., Koszer, S., & Wolf, S. M. (1999). Goldensohn's EEG Interpretation: Problems of overreading and underreading. Armonk, NY: Futura Publishing.

Hara, H. (2007). Autism and epilepsy: A retrospective follow-up study. Brain Development, 29(8), 486-490.

Continued on page 22

EEG in Children with Autism Spectrum
continued from page 21

Hudspeth, W. J. (2009). Multivariate connectivity within a spherical brain space. Unpublished manuscript.

Hughes, J.R., & Melyn, M. (2005). EEG and seizures in autistic children and adolescents: Further findings with therapeutic implications. Clinical EEG and Neuroscience, 36(1), 15-20.

Kamiya, J. (1969). Operant control of the EEG alpha rhythm and some of its reported effects on consciousness. In C.T. Tart (Ed.), Altered States of Consciousness. New York: Wiley.

Kobayashi, E., Bagshaw, A. P., Grova, C., Dubeau, F., & Gotman, J. (2006). Negative BOLD responses to epileptic spikes. Human Brain Mapping, 27, 488–497.

McAlonan, G.M., Cheung, V., Cheung, C., Suckling, J., Lam, G.Y., Tai, K.S, Yip, L., Murphy, D.G.M., & Chua, S.E. (2004). Mapping the brain in autism: A voxel-based MRI study of volumetric differences and intercorrelations in autism. Brain, 128 (2), 268- 276.

Page, L.A., Daly, E., Schmitz, N., Simmons, A., Toal, F., Deeley, Q, Ambery, F., McAlonan, G., Murphy, K.C., & Murphy, D.G.M (2006). In vivo H-magnetic resonance spectroscopy study of amygdala-hippocampal and parietal regions in autism. American Journal of Psychiatry, 163, 2189-2192.

Parmeggiani, A., Posar, A., Antolini, C., Scaduto, M.C., Santucci, M., & Giovanardi- Rossi, P. (2007). Epilepsy in patients with pervasive developmental disorder not otherwise specified. Journal of Child Neurology, 22(10), 1198-1203

Persyst. (2004). Spike and Seizure Detection System Online User's Guide. Retrieved January 4, 2006, from http://www.eeg-persyst. com/web/OnlineUsersGuide.html

Ray, A., Tao, J. X., Hawes-Ebersole, S. M., & Ebersole, J. S. (2007). Localizing value of scalp EEG spikes: A simultaneous scalp and intracranial study. Clinical Neurophysiology, 118, 69–79.

Shelley, B. P., Trimble, M. R., & Boutros, N. N. (2008). Electroencephalographic cerebral dysrhythmic abnormalities in the trinity of nonepileptic general population, neuropsychiatric, and neurobehavioral disorders. Journal of Neuropsychiatry and Clinical Neuroscience, 20, 7–22.

Sterman, M.B., & Egner, T. (2006). Foundation and practice of neurofeedback for the treatment of epilepsy. Applied Psychophysiology and Biofeedback, 31(1), 21-35.

Volkmar, F.R. & Nelson, D.S. (1989). Seizure disorders is autism. The American Academy of Child and Adolescent Psychiatry, 29(1), 127-129

Walker, J.E. & Kozlowski, G.P. (2005). Neurofeedback treatment of epilepsy. Child and Adolescent Psychiatric Clinics of North America, 14(1), 163-176.

Autistic Spectrum Disorders
continued from page 14

Spectrum Disorders. 36th Annual meeting of the Association for Applied Psychophysiology and Biofeedback, Portland, Oregon, April.

Thompson, M. & Thompson, L. (2007). Autistic Spectrum Disorders including Asperger's syndrome: EEG and QEEG findings, results, and neurophysiological rationale for success using neurofeedback training. Presented at the 11th Annual Meeting of the Biofeedback Foundation of Europe, Berlin, Germany. Abstract reprinted in *Applied Psychophysiology and Biofeedback, 32*(3-4), 213-214.

Thompson, M. G. G. & Havelkova, M. (1983). Childhood Psychosis. In Paul Steinhauer & Quentin Rae-Grant (Eds.) *Psychological Problems of the Child in the Family*. New York: Basic Books, Inc.

Thompson, M.G.G.; Patterson, P.G.R. (1986). "The Thompson- Patterson Scale of Psychosocial Development: I; Theoretical Basis," - Canadian Journal of Psychiatry, Volume 31, Number 5, June.

Thompson, M.G.G. (1990). Developmental Assessment of the Preschool Child. In *Difficult Diagnoses in Paediatrics*, Ed. Stockman, J.A., W.D. Saunders, 1990.

Thompson, M., Thompson, J., & Wu, Wenqing (2008). Brodmann Areas (BA) correlated with 10-20 sites and their primary functions. San Rafael, CA: International Society for Neurofeedback and Research.

Thompson, M. & Thompson, L. (2003). Neurofeedback for Asperger's Syndrome: Theoretical Rationale and Clinical Results. *The Newsletter of the Biofeedback Society of California, 19*(1).

Thompson, M. & Thompson, L. (2007). Neurofeedback for Stress Management. In P. Lehrer, R. Woolfolk, & W. Sime (Eds.) *Principles and Practice of Stress Management, 3rd Edition*. New York: Guilford Publications.

Thompson, M., & Thompson, L. (2009). Asperger's Syndrome Intervention: Combining Neurofeedback, Biofeedback and Metacognition. In Thomas Budzynski, Helen Budzynski, James Evans, & Andrew Abarbanel (Eds.), *Introduction to Quantitative EEG and Neurofeedback: Advanced Theory and Applications (Second edition)*. New York: Academic Press/Elsevier, 365-415.

Thompson, M., Thompson, L., & Reid, A. (in press). Neurofeedback Outcomes in Clients with Asperger's Syndrome. *Journal of Applied Psychophysiology and Biofeedback.*

Tower of London Test. 1982. Colorado Assessment Tests. www.catstests.com/prod03.htm

Wing, L. (2001). The Autistic Spectrum: A parent's guide to understanding and helping your child. Berkeley, CA: Ulysses Press.

Using the Tower of London to assess improvement after Neurofeedback training in clients with Asperger's Syndrome

Bojana Knezevic, Lynda Thompson, and Michael Thompson



Asperger's Syndrome (AS), a developmental disorder on the autistic spectrum (Klin, McPartland, & Volkmar, 2005), is characterized by difficulties in correctly interpreting social innuendo, an inability to use emotionally appropriate vocal intonation and volume, as well as stereotyped, rigid, and repetitive patterns of behaviour, activities, and interests (Ross, 1981; Wing, 2001). Individuals with AS have difficulty with cognitive switching, word retrieval strategies (Kleinhans, Akshoomoff, & Delis, 2005), and free recall (Bowler, Gardiner, & Berthollier, 2004). On the Wechsler Intelligence Scales (WISC and WAIS), most clients with AS obtain high IQ scores with significantly better Verbal IQ than Performance IQ (Alvarez, 2004). Furthermore, these clients often display inappropriate social boundaries, such as excessive friendliness and openness with strangers (Thompson & Thompson, 2007). AS occurs more frequently in males (Lawson, Baron-Cohen, & Wheelwright, 2004). However, as a result of numerous controversial issues regarding the diagnosis of AS, its prevalence and an exact cause are still not firmly established (McPartland & Klin, 2006).

Commonly tried interventions for the treatment of AS are psychotherapy, behaviour therapy, social training, group therapy, medications, and speech therapy (Green et al., 2006). Nevertheless, findings regarding their effectiveness remain equivocal. Over the past 12 years, a few papers and presentations have emerged and reported favourable clinical outcomes using neurofeedback (NFB) as an intervention for clients with AS (Coben, 2005, 2007; Jarusiewicz, 2002; Reid, 2005; Solnick, 2005; Thompson & Thompson, 1995; 2003; 2007). Specifically, researchers reported amelioration of AS symptoms based on the single channel electroencephalographic (EEG) assessments when NFB was applied at the central locations (Cz and FCz) with a goal to train down frequencies that were high in amplitude as compared to the rest of the client's EEG (theta 3-7Hz or low alpha 8-10Hz, and/or high frequency beta 20-35Hz) and to train up sensorimotor rhythm (SMR, 12-15 or 13-15Hz; Thompson, Thompson, & Reid, 2008). These clients were also introduced to various problem solving approaches that were each tailored to their area of difficulty.

Our small pilot project at the ADD Centre investigated the utility of the Tower of London—Drexel University (ToL^{DX}) test as an additional measure of changes obtained after 40 NFB sessions combined with metacognitive strategies in clients with AS. ToL^{DX} is an individually administered neuropsychological instrument designed to assess higher-order problem solving—specifically executive planning abilities—in children and adults (Culbertson & Zillmer, 2001). On this test, there is a board with three posts and the subject must move coloured beads from one post to another in order to copy a model shown by the examiner, who changes the pattern between the ten trials that make up the test. According to Lezak (1995), one must be able to look-ahead, respond impartially, generate and select alternative options, and sustain attention in order to achieve a goal. These are the characteristics of executive planning measured by the ToL^{DX}. Numerous studies have indicated that the prefrontal lobes, interacting with other cortical and subcortical structures, are involved in executive planning and that injuries to these areas and/or deficits can have profound effects on behaviour (Cummings, 1993; Grattan & Eslinger, 1991; Shallice & Burgess, 1991). Recently, Thompson and Thompson (2005) have identified high amplitude slow waves (3-10Hz) and/or very low amplitude beta (13-18Hz) and/or high amplitude beta spindling in these regions in children with AS. Other researchers reported impaired performance of children with Autism on the ToL^{DX} as compared to their typically developing counterparts (Hughes, Russell, & Robbins, 1994). It was thus logical to investigate whether executive functioning related to frontal lobe activity, as measured by ToL^{DX} performance, would improve in clients with AS after they received NFB training. Scores on the ToL^{DX} test were compared before and after NFB training. The goal was both to document changes in executive functions and to ascertain whether the ToL^{DX} could be useful for that purpose in a clinical setting.

Methods

The subjects for this pilot project varied in age from 7 years and 6 months to 21 years and 5 months and were mixed in terms of racial backgrounds, countries of origin, and socioeconomic status. They were 19 consecutive clients (16 males and 3 females) recruited over a two and a half year period (see Table 1) who met the criteria for a diagnosis of AS and who participated in at least 40 NFB sessions. A psychologist (LT) made the diagnosis based on a complete clinical assessment including history taking, continuous performance tests (CPTs), behaviour rating scales, intellectual testing (the WISC or WAIS if not done within the past two years), as well as single channel EEG recordings at Cz. After obtaining parental consent, the client with AS did the ToL^{DX}, which was administered both before and after the completion of 40 NFB sessions. Each NFB session lasted approximately 50 minutes during which 10-15 minutes of the training was done on task, that is, time was spent introducing new thinking and learning strategies and the client practiced the strategy while maintaining the alert and focused state as indicated by the computerized feedback. The client was rewarded once he or she increased SMR (13-15Hz) and/or problem solving (15-18Hz), depending if the deficits were present in either one or both, and decreased slow brain wave ac-

Continued on page 24

Tower of London

continued from page 23

tivity, such as theta (4-8Hz) or some other relevant frequency range, such as 3-10Hz or whatever band showed excessive magnitude at the time of initial assessment.

Clients were evaluated on six quantitative variables, which were the scores for executive function assessed by the ToL^{DX}: (a) total moves (TMS; number of moves needed to solve all the test items), (b) total correct (TCS; number of test items solved with the minimum amount of moves), (c) total rule violations (TRV; number of times the client either moved two beads at a time or placed an extra bead on a peg), (d) total time violations (TTV; number of times the client exceeded one minute per item), (e) total initiation time (TIT; the time it takes the client to execute the first move), and (f) total execution time (TET; the time required to solve the problem after the first move). Each raw score was converted to a standard score for statistical analysis. Following completion of all ten test items, clients were rated by the experimenter on a five-point Likert scale with respect to their problem solving approach, attention and activity, and personal-emotional-social regulation.

Table 1
Descriptive Statistics for Demographics and Executive Functions Initial Standard Scores

Variable	(N)	Minimum	Maximum	Mean (M)	Standard Deviation (SD)
Age	19	7.50	21.50	11.97	3.67
IQ	18	82.00	124.00	105.72	13.75
ToL^{DX}					
TMS	19	60.00	108.00	85.37	12.22
TCS	19	74.00	108.00	90.74	9.64
TRV	19	60.00	104.00	72.63	19.69
TTV	19	60.00	114.00	96.95	12.69
TIT	19	80.00	108.00	90.21	6.63
TET	19	60.00	110.00	88.42	15.12

Note. ToL^{DX} – Tower of London – Drexel University

Table 2
Quantitative ToL^{DX} Mean Standard Scores Pre- and Post-40 Neurofeedback Sessions

Variable	PRE	POST	p value
Total Move (TMS)	85.37	94. 11	.027
Total Correct (TCS)	90.74	93.89	.392
Total Rule Violations (TRV)	72.63	92.74	.007
Total Time Violations (TTV)	96.95	101.89	.331
Total Initiation Time (TIT)	90.21	95.68	.066
Total Execution Time (TET)	88.42	99.68	.002

Note. ToL^{DX} – Tower of London – Drexel University

Table 3
Qualitative ToL^{DX} Mean Raw Scores Pre- and Post-40 Neurofeedback Sessions

Variable	PRE	POST	p value
Systematic	3.32	2.26	.003
Deliberate	4.00	2.63	.002
Persistent	2.47	1.42	.000
Flexible	3.84	2.42	.000
Alert	3.58	1.79	.000
Attentive	3.42	1.63	.000
Motor	1.05	1.00	.331
Cooperative	2.21	1.16	.000
Confident	3.63	2.11	.000
Relaxed	4.00	2.26	.000
Frustration	3.42	1.74	.000
Support	3.52	1.58	.000

Note. ToL^{DX} – Tower of London – Drexel University

Results and Interpretation

This clinical outcome study extends our understanding of planning, working memory, cognitive flexibility, and inhibition in clients with AS; partially replicates previous studies that have provided evidence that NFB and training in metacognitive strategies produce positive clinical outcomes in clients with AS; demonstrates the utility of the Tower of London test to further improve the diagnosis of AS and plan treatment for incoming clients; and reveals that systematic data collection can be carried out in a private educational setting. In addition to these general findings, there are a number of specific conclusions that can be drawn from the data obtained.

There were significant improvements on measures of executive planning, inhibition, cognitive flexibility, and planning efficiency (see Table 2). After the training, clients were more likely to work at an increased speed through the problems and violate fewer rules. As such, their problem-solving pattern showed an improved ability to shift set and inhibit prepotent moves (Culberston & Zillmer, 2001). Following 40 NFB sessions, clients with AS seemed more systematic and organized, appeared more attentive during the task, and presented with increased self-confidence (see Table 3). Furthermore, according to our findings, there do not appear to be significant age or IQ effects on changes in ToL^{DX} performance. Clients' raw scores were converted to standard scores relative to their age matched peers. The availability of norms for standard scores means that ToL^{DX} is a useful tool when assessing performance of clients of all ages as it takes into account that executive functions emerge in early childhood and continue to develop over the years until adulthood is reached (Dennis, 1991). As a measure of executive function, the association between intellectual level and ToL^{DX} performance should not be significant. Accordingly, our findings did not indicate any significant IQ effects on clients' performance. As such, ToL^{DX} appears to be a true measure of frontal lobe function.

In summary, ToL^{DX} provides standardized measurement of numerous, although overlapping, cognitive skills. It provides quantitative measures regarding an individual's performance level as it compares each

client's performance to that of age matched peers. The qualitative measures can be used to further clarify interpretation of the quantitative information, such as directing and controlling of executive planning through verbalizations, as well as speed at which it is executed. In light of the previous research that has indicated dysfunction in the frontal lobes in clients with AS (Kaiser, 2006; Thompson & Thompson, 2007), paired with research on frontal lobe functions being linked to ToL^{DX} performance (Owen, 1997), our current findings suggest that ToL^{DX} may be a useful tool in clinical settings. It can enhance accuracy of the diagnostic process, improve treatment planning, and track client progress across time.

Finally, we were able to conduct qualitative observations with two clients who were tested after 40 and 60 NFB sessions. Client 1 seems to have done worse after 40 NFB sessions, but his performance improved after 60 NFB sessions (see Figure 1). After further training he appears to engage in more efficient executive planning, violate fewer rules, and solve problems faster than at the initial assessment. The second client seems to continue to improve his ability to hold information on-line and to become a more efficient problem solver as he progresses through training (see Figure 2). He also seems to maintain gains, such as increased speed of problem solving and reduced impulsivity, observed after 40 NFB sessions. Finally, according to the researcher's observations, these clients continue to advance their problem-solving approach, attention, and social interaction over time (see Figure 3). Perhaps clients with AS may require more than 40 NFB sessions in order to make significant gains on tests of executive functions. Therefore, tracking their progress with tools such as ToL^{DX} may provide useful information to help tailor the training, especially with respect to indicating whether more training is needed and thus help ensure that sufficient training is done.

In addition to a number of limitations, such as a small sample size, lack of establishing inter-rater reliability, and testing and retesting at different times of the day, the mechanism of improvement remains far from clear. The noted increase in confidence and possible decrease in impulsivity would contribute to clients becoming more reflective and better at test-taking. They may have also developed a more positive attitude and an increased interest in pleasing others after 40 NFB sessions. Despite the five-month gap between the two testing sessions, familiarity with the tests and the relationship with the examiner may have played a role. Future studies would ideally include a control group and random assignment to either NFB or an active placebo. This would help distinguish between nonspecific effects and specific effects attributable to the NFB intervention, as well as address the question of practice effects on the ToL^{DX} test. Nevertheless, our findings suggest that 40 NFB sessions coupled with training in metacognition do result in positive effects on executive functioning in clients with AS, including planning efficiency, speed, and ability to switch sets and inhibit certain responses. In particular, clients with AS may realize that proper planning is essential to everyday success and may become better at identifying subgoals and organizing them into a sequence, self-monitoring during the process, and developing a final action plan in order to achieve a goal. Furthermore, employing measures of planning and organizing abilities in clinical settings seems to provide diverse and important information about clients' initial functioning and ongoing progress over the course of training. Such knowledge may be useful to clinicians when determining the diagnosis and devising a subsequent treatment plan for each client.

References

Alvarez, A. (2004). Issues in assessment: Asperger's syndrome and personality. In M. Rhode & T. Klauber (Eds.) *The many faces of Asperger's syndrome. The Tavistock clinic series.* London: Karnac Books, 113-128.

Bowler, D. M., Gardiner, J. M. & Berthollier, N. (2004). Source Memory in Adolescents and Adults with Asperger's Syndrome, *Journal of Autism & Developmental Disorders. 34*(5), 533-542.

Coben, R. (2005). *Assessment guided neurofeedback for autistic spectrum disorder.* Presentation at the International Society for Neuronal Regulation 13th annual conference, Denver, Colorado, September.

Culbertson, W.C., & Zillmer, E.A. (2001). *Tower of London – Drexel University (TOL^{DX}).* New York: Multi-Health Systems Inc.

Continued on page 39

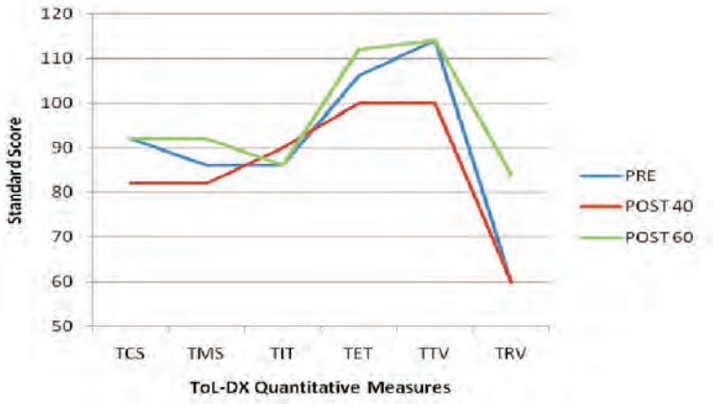


FIGURE 1. *ToL^{DX} standard scores on six quantitative measures at three time points (pre-, post-40, and post-60 NFB sessions) for Client 1.*

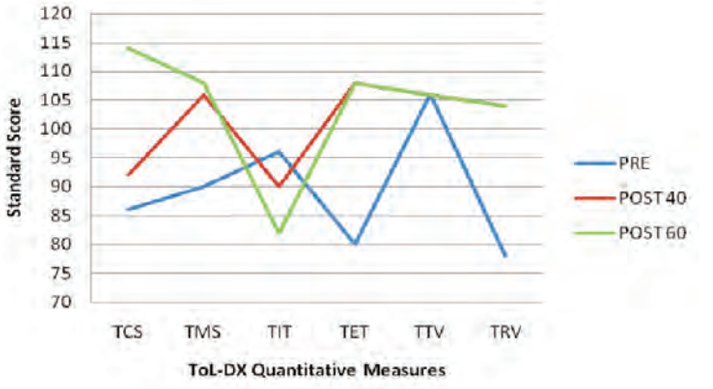


FIGURE 2. *ToL^{DX} standard scores on six quantitative measures at three time points (pre-, post-40, and post-60 NFB sessions) for Client 2.*

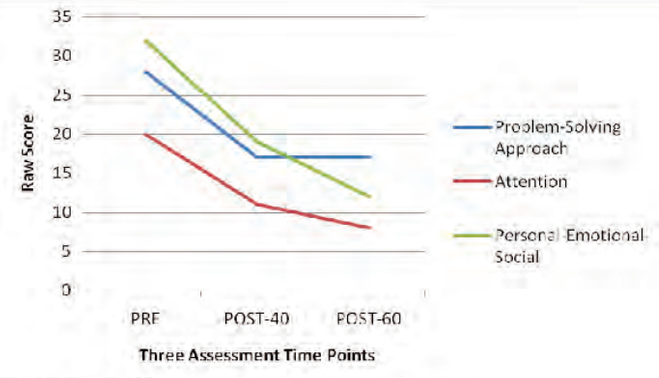


FIGURE 3. *ToL^{DX} raw scores on three groups of qualitative measures at three time points (pre-, post-40, and post-60 NFB sessions) for Clients 1 and 2 combined.*

Myths, Fears and Reality of Washing a Patient Off Their Medication For QEEG Assessment

Daniel A. Hoffman, M.D., Medical Director, Neuro-Therapy Clinic
President & Chief Medical Officer, CNS Response

Introduction

It is a common occurrence for a patient in a mental health practice to present for QEEG evaluation while on numerous, concurrent psychotropic drugs. Medication stacking, particularly in the treatment resistant patient, may cause adverse reactions, over-activation or even neurotoxicity.[4] Equally distressing is the patient who may actually no longer need any medication but remain on psychotropic drugs for years.[3, 6] But often the task requested of the Neurotherapist is to understand the patient's EEG, yet medications are known to affect brainwaves making the assessment results murky, unless a clean naïve brain can be evaluated.

In order to accomplish this task effectively, washing out medication is necessary in order to obtain a true baseline state. This is not done without trepidation, regardless of who orchestrates the taper schedule. Since each medication, drug, or supplement requires 5 to 7 half lives in order to clear the brain, most health care practitioners are understandably uncomfortable washing a patient out of their chemicals. To make matters worse, while the taper decision needs to be done by the prescriber, there is often skepticism about the validity of QEEG and Neurofeedback, thus starting the process off with initial resistance.

A search of the literature revealed several articles on the risks of getting patients off medication or switching from one drug class to another, but no articles were discovered recommending a therapeutic washout except one by this author.[6]

Most prescribers would welcome the opportunity to "start over" with some patients by washing out their current drugs. In fact, most would consider it good medicine, particularly when the patient has been on meds for decades or when they were initially put on psychotropics in their adolescent years prior to the completion of the brain's myelination. On the other hand, patients and prescribers alike are reticent to take the risk of the patient becoming clinically worse or even decompensating, (particularly where one is unlikely to be able to washout or taper in a hospital setting). Washing out some medications, like benzodiazepines, should be considered with caution due to potential for addiction and withdrawal.[2]

This article is based on the combined experiences of several practicing psychiatrists who routinely washout patients in order to perform a Referenced-EEG (rEEG™) test to predict medication response.[7-14]

However there are several factors that should be noted which determine actual brain clearance. LSD, for example, theoretically clears in 2.5 hours according to the five half-life rule, yet the CNS effects continue well beyond that period. Doses of medications and length of time using the drug also affect brain clearance.

For purposes of this reporting, 2,008 patients were followed for medication tapering off both their psychotropic and other medications (including vitamins, herbs, OTC meds, alcohol, caffeine, etc) under the author's guidance. This included the patients at two substance abuse clinics and several leading academic institutions involved in randomized studies for rEEG™--all occurred without significant incidence.

A carefully orchestrated washout of medications was successful without any major incidence.[15, 16] Most of our cases were seen in outpatient settings of non-psychotic patients. In addition to the goal of taking patients off medication in order to get clean rEEG™ testing results, other substantial benefits were observed, not the least of which were patients feeling better.[6] Over this period of several years, based on a survey of other investigators, only15 patients (0.75%) presented with adverse reactions due to medication withdrawal yet elected to continue the process until the rEEG™ test could be performed. An additional 5 (0.25%) patients were unable to complete their washout and chose to go back on their previous medications.

There were no known correlations with the patients who had trouble washing out. Symptoms, history, quantitative EEG variables, or motivation failed to predict difficulty in getting off medication. Previous response to getting off psychotropics was the only variable which helped to determine ease of washout, as discussed below.

Recommendations:

While there are many reasons to contemplate a washout, testing for a new evidence-based biomarker was the motive for these particular patients. Referenced-EEG is a test using a clean "digital EEG" submitted to a database of pattern recognition correlated to clinical outcomes of most psychotropic drugs other than antipsychotics.[7-14]

In the process of using this new test, the author gained experience in tapering and washing out medications and suggests several valuable principles to minimize risks.

1. Preparation and proper informed consent gains the patient's confidence and support. Initially it consists of the patient borrowing from the posture and expertise of the provider recommending the washout. Proper management of expectations can make the difference between a successful taper and a potential crisis.

   Part of the preparation includes the patient understanding that there may be discomfort over the next few weeks. In fact, anticipating discontinuation symptoms from SSRIs of dizziness, dry mouth, insomnia, nausea, nervousness, sweating, anorexia, diarrhea, somnolence, and sensory disturbances (shock like electrical sensations or "zaps") educates the patient as to realistic expectations.

2. Depending on the reason for washout, allowing the patient to have input into the decision of taper speed helps confirm a sense of teamwork, avoiding anger if they experience symptoms. Whereas the more typical taper speed for SSRIs, for example, may be to half the dose every two weeks, it has been my experience that when given this choice for patients doing a Referenced-EEG, almost uniformly they'll elect a more rapid, yet still appropriate, taper schedule in order to reduce the length of time for any potential discomfort.

3. Asking patients if they've ever missed any doses of their medicine and, if so, what symptoms they experienced[6] helps identify whether a slower or more aggressive taper should be attempted.

4. Medications that have long half-lives can temporarily be switched to similar drugs in the same class with shorter clearance (e.g., escitalopram (Celexa) for fluox-

etine (Prozac) or quetiapine (Seroquel) for aripiprazole (Abilify).

5. Written taper schedules for each medication can help alleviate anxiety, especially when a patient is at home questioning their memory of your instructions. Compliance is as much an issue tapering off medications as it is remaining on them.

6. If discontinuation symptoms do develop and cause a significant problem for the patient, allowing the use of natural products to help relieve symptoms of agitation may be beneficial. Typically augmenting with choline, inositol and/or whey protein isolate can ease anxiety and irritability – as long as they are off these products for the last 2 days if they are preparing for rEEG™ testing.

7. If the current schedule of medication withdrawal is causing too much discomfort, the patient should be instructed to immediately return to the previous dose until the prescriber can see them again. In my experience, this is rarely necessary.

8. Before attempting to washout a patient with significant psychiatric symptoms, gathering their support system to gain consensus is not only important for the patient's preparation, but also for their safety. The members of the support environment must agree to watch the patient for suicidal ideations or attempts, mood swings or any other potentially life threatening worsening of symptoms. They must also be prepared not to leave the patient alone. If there is not an adequate support system in place, the discontinuation may be delayed until such support is available.

9. Patients who become extremely ill (e.g., suicidal, homicidal, extremely depressed) or who have medical conditions requiring their continual use of prescription drugs (active cardiac, asthma or seizure disorders, for example) should not be taken off their medications due to the risk of serious medical sequelae. The person prescribing for the medical condition should always make the decisions about the degree of risk of tapering off their medication.

Conclusions:

As reported in an earlier paper[6], several patients experienced significant relief from merely ridding themselves of the toxic effects of medications they didn't need. Others experienced decreased side effects such as sedation, sexual dysfunction and/or cognitive disturbances. Many reported feeling worse, either due to the return of symptoms or to acute side effects of discontinuation. Those patients improved when their medications were reinitiated after successful completion of the rEEG™ assessment.

The purpose of this paper was to ease the concerns of many clinicians who feel it may, for one reason or another, be a good therapeutic approach, yet have concerns about how "risky" it might be. In my direct personal experience combined with cases I've supervised, while there may be a week or so of unpleasantness, irritability and in some cases, even significant mood changes, when done correctly and with an adequate support system, there are many good therapeutic reasons to try a washout and taper in order to adequately assess a brain's QEEG. Juxtaposed to the increasing number of patients on multiple psychotropic medications, many of which are being used to treat a side effect from a previous drug, there are times when a fresh look at the brain's neurochemistry makes sense, and the fear of washout need not stand in the way.

References:

1. Mitchell, AJ. Understanding medication discontinuation in depression, Psychiatric Times, 2007: 24(4):
2. Ramadan, MI., Werder, SF., Preskorn, SH. Safe use of benzodiazepines, buspirone, and propranolol. Journal of Family Practice. 2006: 5(5):

Continued on page 30

Z-score Training with Profound Autistic Spectrum Disorder: A Case Study

Penijean Rutter, M.A., C.R.C., L.M.H.I
Clinical Services Director at Cognitive Neuro Sciences, Inc., Tampa, FL

Brain-based biofeedback with severely compromised clients can be uniquely challenging. When Billy's mother brought him in for an evaluation, I was unsure we would be able to record his EEG without sedating him which would render the data unusable. Billy was an active, large-framed 12-year-old with profound autistic spectrum behaviors and the cognitive function of a small child. He met DSM-IV criteria for Autistic Disorder with the following symptoms:

- Delay in development of verbal and non-verbal communication
- Lack of social or emotional reciprocity
- Stereotyped and repetitive motor manners, impaired fine motor skills
- Tourette's-like physical spasms, and involuntary high-pitched shrieks
- Failure to develop peer relationships appropriate to developmental level, or to spontaneously engage peers in play activities
- Interrupted sleep patterns, and nocturnal enuresis
- Aggressive behavior toward peers and service providers, including hitting, scratching, grabbing and biting

Billy's mother reported her concern with his level of aggression, stating that his increasing tendency to hit and bite people as he entered puberty was becoming problematic and she was worried that the staff members at his school were exacerbating his aggressive behavior by responding to it poorly. As she described his sensitivity to light and sound, and his habit of biting first and asking questions later, I became even less optimistic about a QEEG recording and analysis, much less doing traditional neurofeedback training with him (Sichel, et al. 1995)

Our first attempt at putting an electrocap on Billy more closely resembled a three ring circus than a therapeutic intervention. Billy's mother, one of our clinic technicians, and I worked to get him hooked up as quickly and efficiently as possible while he flailed his arms, shrieked, and tried to bite us. After the second time he pulled the cap off his head, and threw me into a nearby wall, I turned all of the lights in the room down very low, put on some soothing nature sounds, and found an empty plastic water bottle that he could bite down on when he became agitated instead of my arm. Trying to record with minimal muscle artifact required our collective innovation to calm and occupy Billy who had particular difficulty keeping his eyes closed, and would tic even more when he felt stressed or nervous.

An analysis of Billy's EEG recordings revealed a number of significant findings. (See Figures 1 and 2) We did not observe any epileptiform or paroxysmal activity in his raw waves, but statistical analysis of spectral activity according to NeuroGuide v. 2.3.8 and SKIL v.3.0 indicated significant dysregulation of both activity and connectivity measures compared to healthy functional children his age.

The most significant findings in my opinion were Billy's alpha hypocoherence and his high phase activity in the frontocentral lobes because they replicated across databases, showing up in the most recent versions of both SKIL and NeuroGuide, and appearing in both recording states. His excess activity in high beta was also replicated between databases and recording states, then localized to 23-27 Hz using single Hz bins (See Figure 4). Because EMG artifact in the beta ranges is diffuse across the entire spectrum and not localized to specific bandwidths (Nunes, et at., 2004), we determined that Billy's beta activity reflected EEG instead of muscle artifact.

The eyes closed recording showed a significant reduction of alpha activity in frontal and temporal regions that corresponded with the only instance of slow phase activity between frontal lobes and occipital lobes, and excess beta asymmetry (Baving 2002).

Other findings that we noted for monitoring during treatment were Billy's low amplitudes in delta, particularly in eyes open, and his high amplitudes in beta at homologous sites, most notably over the motor strip in eyes open.

Once we had looked at the data, my colleague, William A. Lambos, Ph.D., and I discussed whether Billy was trainable using traditional neurofeedback methods, and if so, which training approach would be most beneficial There was some hesitancy on our part to take his case because we were concerned that he would be unable to sit in the chair, leave the wires on his head, and engage in the task for long enough periods of time for training to actually occur. At the time, we did not have other interventions, such as LENS or Neurofield, that require much less engagement from the client, and we relied on our client participation.

Ultimately, we decided to take Billy's case for a couple reasons. He was a prime physical candidate for treatment because his mother had put considerable effort into providing Billy with excellent nutrition, including supplements believed to assist with the raw materials necessary for brain health and function. She had also visited homeopathic or natural practitioners, and tried alternative methods to help Billy improve his functioning. When he came to us for treatment, he was medication free.

Another factor that weighed heavily into our decision was the opportunity to gather data that would contribute to our internal attempts as a clinic to construct a working paradigm of how neurofeedback impacted our clients. We had been debating between ourselves whether or not our clients need to cognitively understand what they were trying to accomplish for their brains to correctly interpret the feedback they received from our training programs and for function to improve. This question dovetailed with our interest in how much placebo effect or therapeutic interaction with the trainer contributed to the reduction in symptoms that our clients experienced (Budzynski 1996). Billy was an ideal opportunity for us to observe the individual capability of a child with severely compromised functioning to engage in the training tasks without a full cognitive grasp of the purpose behind them, which would affect our perception of not only placebo effect, but how the brain perceives reward and uses feedback to reorganize itself.

We decided to train Billy using the Z-score training program on BrainMaster equipment because the normative database used by the Z-score DLL is based on the database used in Thatcher's NeuroGuide EEG analysis software (Thatcher 2003), and the

connectivity measures in all of Billy's maps indicated severe dysregulation across both recording states.

We created a training protocol as if he were a high functioning client to give his brain the most comprehensive and efficient feedback we could devise regardless of what his cognitive limitations appeared to be (Banaschewski & Brandeis, in press). I was operating from the premise that the brain is a reward seeking machine, and we could potentially condition him to associate the visual and aural feedback from the training program with verbal and social approval from me and his mother, thereby reinforcing him cognitively or emotionally as his brain was rewarded for meeting training criteria.

I trained him with a ground lead behind the right ear, two linked ears for reference, and then four active training electrodes that recorded the live EEG which the program used to create the feedback. I placed the four training electrodes at F3, F4, C3 and C4 because much of the pattern of dysregulation from his brain maps was concentrated in his frontal and central lobes (see Figures 1-2).

There are a variety of different approaches to using Z-score training in a clinical setting, and I chose to train Billy using the "percentZOK" setting wherein the training program monitors the activity at each of the four EEG channels (F3, F4, C3, C4) in absolute power, relative power, and power ratios, and then measures the asymmetry, coherence and phase between each site pairing. This produces 248 variables at which to measure and observe Billy's EEG at those four sites compared to the activity contained in the normative database being accessed.

Creating the actual feedback consisted of setting the standard deviation parameters and telling the program what percentage of the 248 Z-scores needed to be within the set standard deviation criteria simultaneously for Billy to get a reinforcement, which consisted of musical tones or a visual that activated when he met criteria for reinforcement.

The first few training sessions were particularly challenging as Billy and I acclimated to each other and created a routine. I learned to watch his body language and determine when he was becoming agitated enough to act out. Billy and I conditioned each other as our rapport developed, although his continual attempts to grab and bite me extinguished any inattentive behaviors I might have had, far more quickly than my soothing vocal tones and musical rewards shaped his attention. It was helpful to hook him up with very low lighting, and then to train him completely in the dark with the computer monitors dimmed and the musical notes turned down very quietly because of his sensitivities to sound and light.

To distract him as I put on his electrodes, I made up a song about the electrode placement that detailed each step of what I was doing and what I was going to do next to facilitate his comfort level with the procedure and to help him routinize the sensations. I hooked him up in exactly the same order each time and within five or six sessions he would hum along and tilt his head for the next placement.

The involuntary muscle tics and upper body spasms were an issue of concern as his EEG readings were riddled with EMG artifact and I was unsure he would be able to relax enough to produce accurate feedback. The first session, Billy trained for 24 minutes, during which he was able to keep his eyes open and his body still for 20-30 second stretches about 6 times over the course of the session. I monitored his feedback parameters throughout training and adjusted them to keep his reward percentage high enough that he was challenged but low enough that he could receive sufficient reinforcement to keep his brain engaged. Using the percentZOK program, this meant keeping his reward levels between 40-60%.

Over the first 20 sessions, Billy seemed intermittently calmer and less agitated. He ticced less during training, and he sat still and appeared to be engaged by the visual and musical feedback for longer periods of time. Every handful of sessions, he would decide he was done training 5-10 minutes into it and stand up and pull the electrodes off of his head, or he would not tolerate being hooked up at all, and we would have to reschedule, but most

Continued on page 30

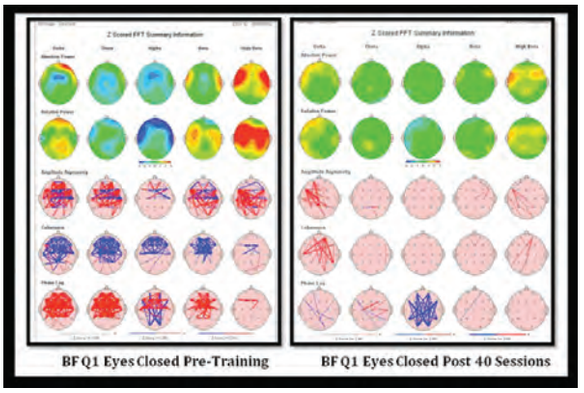


FIGURE 1

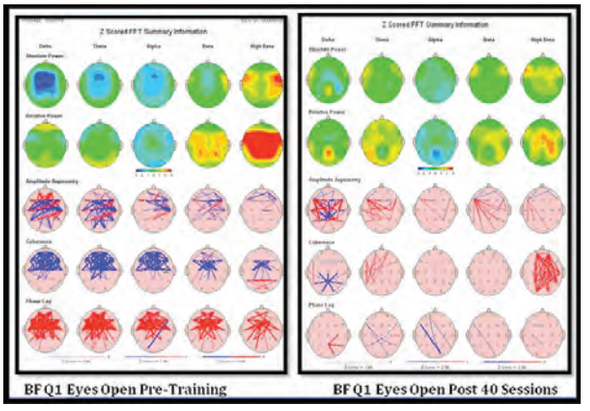


FIGURE 2

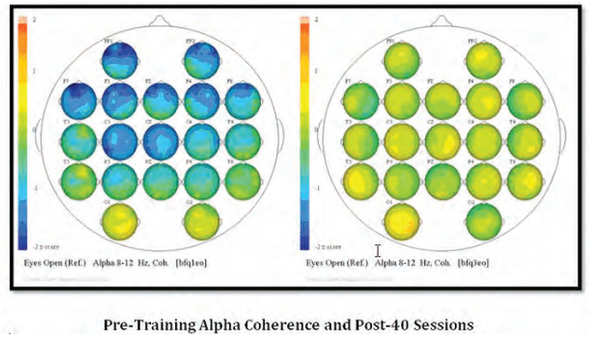


FIGURE 3

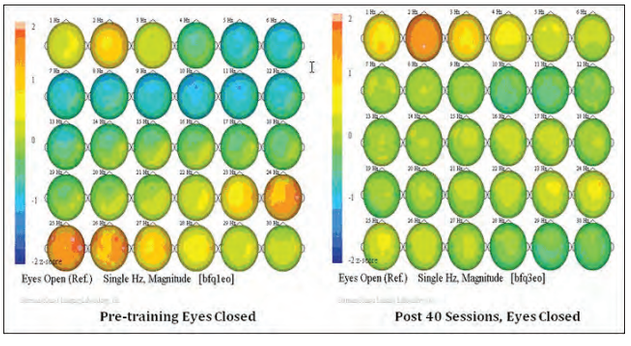


FIGURE 4

Z-Score Training
continued from page 29

of the sessions we were able to coax him or distract him long enough that we kept him in the chair at least 20-30 minutes.

Billy's mother reported less aggression and bed-wetting during the first 10 sessions but also an increase in his restlessness at night saying that he was sleeping less and seemed more activated. Between 15-20 sessions he began to sleep through the night completely several times a week, something he had not done in several years, and his restlessness during the other nights of the week decreased markedly. His ticcing also lessened significantly, and his mother reported increasingly fewer upper body spasms.

As we progressed through training, Billy became more verbal. When we hit 20-30 sessions, he was making eye contact and addressing peers voluntarily without prompting. His was also more easily engaged by external stimuli such as television shows, school work and video games, demonstrating longer periods of focus and attention. There were some days that I couldn't get Billy hooked up and in the chair, and other days where he trained for 40 minutes and wanted to go longer.

His behavioral problems at school and in social interaction improved over the course of training. His mother reported that the most notable difference she observed was his ability to process verbal or social cues and respond much more quickly and easily than he had before. Previous to treatment, Billy would take a long time to respond to instructions or questions, often becoming agitated or angry when he had difficulty formulating a response. As his brain maps improved, so did his speed and timing in verbal and social interactions.

After forty sessions of training, we observed a decrease in his hypocoherence (See Figures 1-3) across both recording conditions, a decrease in the high beta amplitudes seen in his pre-training maps, and what appeared to be a shift toward global regulation in all EEG variables that were analyzed. During this same time period, Billy's mother reported improvements in symptoms and behaviors that could possibly correlate with the changes in the EEG.

It has been our experience that the children or adults that we work with that exhibit autistic spectrum disorder symptoms often have EEG maps that show either an excess or a deficit in connectivity measures between brain areas when compared to a normative database. We can postulate, using a functional connectivity model, that problematic connectivity measures between particular areas of the brain can correspond to behavioral excesses or deficits that the client is demonstrating.

In Billy's case, his difficulties with social interaction, verbal processing, and self-regulation appeared to lessen as his EEG normalized in both activity and connectivity. Feedback from his family members proved consistent with our observations.

The fact that Billy was able to effect such a significant change in his own EEG (Egner 2001) without a cognitive understanding of the goal he was trying to accomplish indicates some interesting possibilities for future application of brain based biofeedback training with significantly impaired populations.

Banaschewski, T., & Brandeis, D. (in press). Annotation:What EEG/ERP tell us about brain function that other techniques cannot tell us. Journal of Child Psychology and Psychiatry.

References

Baving, L., Laucht, M., & Schmidt, M.H. (2002). Frontal brain activation in anxious school children. Journal of Child Psychology and Psychiatry, 43, 265–274.

Budzynski, T. H. (1996). Brain brightening: Can neurofeedback improve cognitive process? *Biofeedback, 24* (2), 14-17.

Collura T.F., Jacobs E.C., Braun DS, & Burgess, RC (1993). EView–a workstation-based viewer for intensive clinical electroencephalography. *IEEE Transactions*

on Biomedical Engineering, 49, 736-744.

Duffy, F., Hughes, J. R., Miranda, F., Bernad, P. & Cook, P. (1994). Status of quantitative EEG (QEEG) in clinical practice. *Clinical. Electroencephalography, 25* (4), VI - XXII.

Egner, T., & Gruzelier, J.H. (2001). Learned selfregulation of EEG frequency components affects attention and event-related brain potentials in humans. Neuroreport, 12, 4155–4159.

Fehmi, L.G. (1978). EEG biofeedback, multi-channel synchrony training, and attention. Sugerman, A., &Tarter, R.E. (Eds.). Expanding Dimensions of Consciousness. New York: Springer Publishing Company, pp. 155-182.

Fehmi, L.G. & Collura, T.F., (2007) Effects of Electrode Placement Upon EEG Biofeedback Training: The Monopolar-Bipolar Controversy Journal of Neurotherapy, Vol. 11(2)

Fernandez, T., Herrera, W., Harmony, T., Diaz-Comas,L., Santiago, E., Sanchez, L., Bosch, J., Fernandez-Bouzas, A., Otero, G., Ricardo-Garcell, J., Barraza,C., Aubert, E., Galan, L., & Valdes, R. (2003). EEG and behavioral changes following neurofeedback

treatment in learning disabled children. Clinical Electroencephalography, 34, 145–152.

Hammond, D. C. (2005a). Neurofeedback with anxiety and affective disorders. *Child & Adolescent Psychiatric Clinics of North America, 14*, 105-123.

Nunes RR, Almeida MP, & Sleigh JW (2004). Spectral entropy: a new method for anesthetic adequacy. Revista brasileira de anestesiologia, 54, 404-22.

Sichel, A.G., Fehmi, L.G., & Goldstein, D.M. (1995) Positive outcome with neurofeedback treatment in a case of mild autism. Journal of Neurotherapy, 1 (1), 60-64.

Thatcher, R.W., Walker, R.A., Biver, C., North, D., Curtin, R., Quantitative EEG Normative databases: Validation and Clinical Correlation, J. Neurotherapy, 7 (No. ¾): 87 - 122, 2003.

Thatcher, R.W. (1998) EEG normative databases and EEG biofeedback. Journal of Neurotherapy, 2(4): 8-39.

Thatcher, R.W., North, D., and Biver, C. EEG and Intelligence: Univariate and Multivariate Comparisons Between EEG Coherence, EEG Phase Delay and Power. Clinical Neurophysiology, 2005, 116(9):2129-2141.

Washing a Patient Off Their Medication
continued from page 27

3. DuVal, G. Ethics in psychiatric research: Study design issues. Canada Journal of Psychiatry. 2004;49:55–59.
4. Preskorn, SH, Lacey, RL. Polypharmacy: When is it rational? Journal of Psychiatric Practice. 2000;13(2):97-105.
5. Suppes T, Baldessarini RJ, Motohashi N, Tondo L, Viguera AA. Special treatment issues: Maintaining and discontinuing psychotropic medications. *Rush AJ (ed): Mood Disorders. Systematic Medication Management. Mod Probl Pharmacopsychiatry. Basel, Karger, 1997, vol 25, pp 235-254 (DOI: 10.1159/000061671)*
6. Hoffman, DA. "First, Do No Harm – A Basic Tenet in Jeopardy?" Journal of Neurotherapy. 2006: 10(4):53-61.
7. Emory WH, Schiller M, Suffin SC. (June, 2004). Referenced–EEG in the treatment of eating disorders. Poster No. 221 for 44th Annual Meeting of New Clinical Drug Evaluation Unit (NCDEU). Phoenix, Arizona.
8. Schiller M, Emory, H. Background, methodology and support of referenced-EEG. In:

Physician's Guide to Referenced-EEG (rEEG). CNS Response; 2005;1-5.

9. Schiller MJ, Emory WH, Shaffer J, Hamilton JT, Hoffman DA, Davis A, Suffin SS. (May, 2005). EEG guidance of psychopharmacologic treatment: multi-site experience. Poster No. 10 for 158th Annual Meeting of American Psychiatric Association. Atlanta, GA.
10. Schiller, M, et. al., Referenced-EEG in the Treating of Eating Disorders, presented at NIMH's 44th NCDEU in June 2004.
11. Schiller, MJ, Emory WH, Shaffer J, Hamilton JT, Hoffman DA, Davis A, Suffin SS. (May, 2005). Cigna Atlanta Demonstration Project: July 2000 – July 2002. In Poster No.10 for 158th Annual Meeting of American Psychiatric Association, Atlanta, GA.
12. Shaffer JH, Milner JE, Schiller MJ. (May, 2005). rEEG-Guided Pharmacotherapy for

Severely Ill. Dually Diagnosed Patients. Poster No. 684 for 158th Annual Meeting of

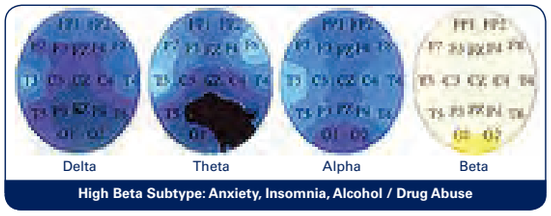
American Psychiatric Association. Atlanta, GA.

13. Suffin SC, Emory WH, Gutierrez N, et. al., A QEEG Method for Predicting Pharmacotherapeutic Outcome in Refractory Major Depressive Disorder, J of Physicians and Surgeons. 2007:12(4):104-108.
14. Suffin SC, Emory WH. Neurometric subgroups in attentional and affective disorders and their association with pharmacotherapeutic outcome. Clin EEG Neurosci 1995; 26:76-83.
15. Zajecka J, Fawcett J, Amsterdam J, Quitkin F, Reimherr F, Rosenbaum J, Michelson D, Beasley C. (1998). Safety of abrupt discontinuation of fluoxetine: a randomized, placebo-controlled study. *J Clin Psychopharmacol., 18(3):193-7.*
16. Wernicke, Joachim F. MD, PhD *; Adler, Lenard MD +; Spencer, Thomas MD ++; West, Scott A. MD [S]; Allen, Albert J. MD, PhD *; Heiligenstein, John MD *; Milton, Denai MS *; Ruff, Dustin PhD *; Brown, W. Jeffrey MS *; Kelsey, Douglas MD, PhD *; Michelson, David MD *, Changes in Symptoms and Adverse Events After Discontinuation of Atomoxetine in Children and Adults With Attention Deficit/Hyperactivity Disorder: A Prospective, Placebo-Controlled Assessment. Journal of Clinical Psychopharmacology. 24(1):30-35, February 2004

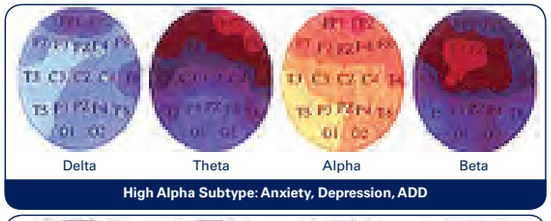
SCOTTSDALE NEUROFEEDBACK INSTITUTE

QEEG mapping service

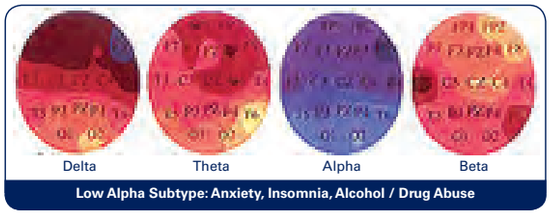
QEEG / TOPOGRAPHIC BRAIN MAPS:
Generalized Anxiety Disorder Subtypes



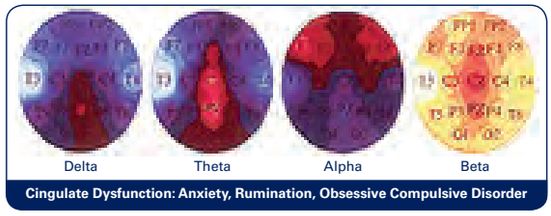
High Beta Subtype: Anxiety, Insomnia, Alcohol / Drug Abuse



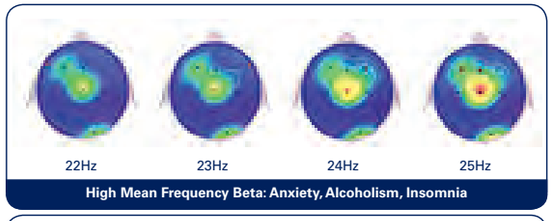
High Alpha Subtype: Anxiety, Depression, ADD



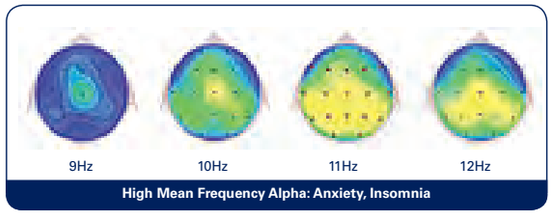
Low Alpha Subtype: Anxiety, Insomnia, Alcohol / Drug Abuse



Cingulate Dysfunction: Anxiety, Rumination, Obsessive Compulsive Disorder



High Mean Frequency Beta: Anxiety, Alcoholism, Insomnia



High Mean Frequency Alpha: Anxiety, Insomnia

AVAILABLE SERVICES

Full Package: #'s 1-7: minimum recommended for Neurotherapy *Includes electronic copy. Priority mail is $20 extra.*	**$225.00**
Full Package: #'s 1-6: Without report (1-5 only) *Includes electronic copy. Priority mail is $20 extra. If one database used the minimum is $75.00*	**$195.00**
01) NX Link - NYU/E. Roy John Normative Database (Eyes Closed)	$70.00
A) NX Link Discriminant Analyses: ADD, LD, Depression, Memory/Dementia, Substance Abuse, Head Injury, Schizophrenia/Thought Disorders	
02) EureKa3! - NovaTech EEG LORETA Analysis System and Adult Normative *Database - Eyes Closed*	$70.00
03) Neuroguide - R. Thatcher Normative Database	$70.00/each
A) Eyes Closed Linked Ears Z-scores // Eyes Closed LaPlacian Z-Scores	
B) Eyes Open Linked Ears Z-Scores // Eyes Open LaPlacian Z-Scores	
04) Neurorep - W. Hudspeth QEEG Analysis System	$70.00/each
A) Eyes Closed - Weighted Average, Z-scores, Magnitude, % Power, LaPlacian, Average Spectrum, coherence, connectivity	
B) Eyes Open - Weighted Average, Z-scores, Magnitude, % Power, LaPlacian, Average Spectrum, coherence, connectivity	
05) Thatcher TBI Discriminant Analysis and Severity Index	$70.00
06) Thatcher Learning Disabilities Discriminant Analysis and Severity Index	$70.00
07) Clinical Correlations and Neurotherapy Recommendations by Bob Gurnee	$70.00

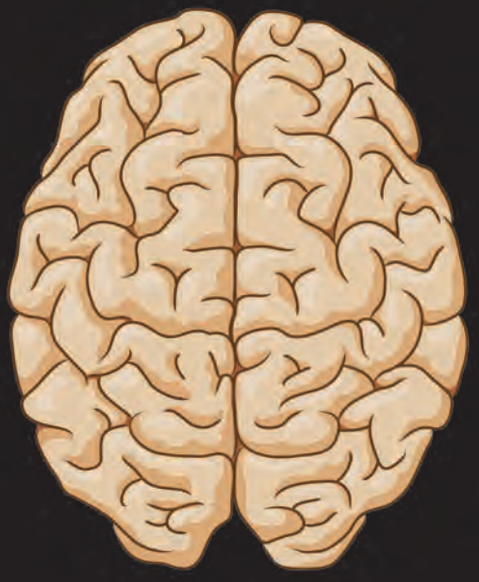
total value: $630

08) Conventional Medical EEG - Read by Neurologist	**$125.00**
09) EureKa3! – NovaTech EEG LORETA Analysis - Eyes Open-Non Database	**$70.00**
10) Neurorep - W. Hudspeth QEEG Analysis System: Task *Weighted Average, Z-scores, Magnitude, % Power, LaPlacian, Average Spectrum*	**$70.00**
11) Supervision and Training Hourly Rate	**$100.00**
12) Extra set of Printed Maps sent priority mail	**$35.00**
13) Electronic (sent via FTP or E-mail) and Paper Copies of Maps sent priority mail with package purchase *(Standard package rates only include electronic or paper copies of maps, not both)*	**$20.00**
14) Overnight Shipping & Handling (Price varies with carrier, destination, & package weight)	**$Varies**

ROBERT L. GURNEE
MSW, BCIA:EEG, QEEG Diplomate, Director

SCOTTSDALE NEUROFEEDBACK INSTITUTE / ADD CLINIC
8114 East Cactus Road #200, Scottsdale, AZ 85260
Tel: (480) 424 7200 Fax: (480) 424 7800
Web: www.add-clinic.com Email: add@add-clinic.com
Established 1982

HELPING THE



HELP US.

The ISNR Research Foundation hopes you'll consider being part of the solution. The problem is that for neurofeedback to be better recognized by other medical and psychological professionals, and for better insurance coverage, we need to perform studies proving effectiveness.

You know it worked for you.

The ISNR Research Foundation hopes to accomplish the necessary research to demonstrate that neurofeedback is or is not effective in particular disorders.

You can HELP THE BRAIN HELP US by making a donation to the Foundation

ISNR
INTERNATIONAL SOCIETY FOR NEUROFEEDBACK & RESEARCH

NEUROFEEDBACK AND SELF REGULATION

WWW.ISNR.ORG

Resolving the International "Blind-Spot": ISNR RF Information Officers

We recently published a meta-analysis on Neurofeedback and ADHD where we were finally able to conclude that: *"…Neurofeedback treatment for ADHD can de considered "Efficacious and Specific" (Level 5)…"* (Arns, de Ridder, Strehl, Breteler & Coenen; 2009). This conclusion could be mainly drawn based on some excellent and recent studies from Germany, of which 2 were published in German. This made me realize that since the ISNR meetings are always held in the US and that for most English speaking people it is very hard to be able to keep up-to-date with publications in non-English journals, that there was a scientific 'blind-spot' towards studies published in other languages.

After discussing this with the ISNR Research Foundation it was decided to identify a group of international Information Officers who would report back to the ISNR Research Foundation (ISNRRF) on international scientific publications, articles but also on legal developments and grants/funding in the field of Applied Neuroscience (e.g. Neurofeedback, QEEG, Neuromodulation). We hope that this initiative will help to resolve the international 'Blind-Spot' so we are better aware of relevant research performed across the globe. Furthermore, we also hope that this information will enable ISNR to better understand and learn from international developments such as regulatory issues and grants obtained for Applied Neuroscience research and foster further collaborations with internationally like-minded organizations.

We currently have received a positive response from the people below for the geographical areas indicated (Carlos, Nerida, Tato, Alexei, Mitzi, Jo and Pavel thanks for your help!). We are still seeking for other Information Officers for the areas that have not been covered yet. If you are interested, please contact me!

Mexico / S-America	Carlos Novo
Australia	Nerida Saunders
Russia	Tato Sokhadze
Russia	Alexei Berd
South Africa	Mitzi Claassen
Japan	Jo Sato
Slovakia	Pavel Krivulka

We hope to be able to provide a first update on international developments during the ISNR meeting in Indianapolis next September, so hopefully see you there!

Kind regards,

Martijn Arns

On behalf of the ISNR Research Foundation martijn@brainclinics.com

Arns, M., de Ridder, S., Strehl, U., Breteler, M & Coenen, A. (2009) Efficacy of Neurofeedback Treatment in ADHD: the Effects on Inattention, Impulsivity and Hyperactivity: a Meta-Analysis. *EEG and Clinical Neuroscience*, 40(3): 180-189.

Research Foundation Donations since Last Issue Total = $1,510

Delta Level:

Gail Awaya
Ann Richman
Seaburn Fisher
Thomas Fink
Kristen Sokol
Shanti Fry

SMR level

Dee Edmonson
Howard Gray Atkins
Bob Gurnee (SNI)
Lynda Kirk
Barbara Cohen
John Fisher

Beta Level

Alberto Texidor
Deborah Stokes
John Davis

Gammalevel

Martijn Arns
(Brain Clinics Diagnostics)
Jon Frederick

Brodmann booklet sales:

18 booklets=$360

Neuropathological Theories and EEG Gamma Oscillation Abnormalities in Autism

Estate (Tato) Sokhadze, Ph.D., Joshua Baruth, M.S., and Manuel Casanova, M.D., University of Louisville School of Medicine



Informed clinical consensus defines autism as a behavioral syndrome characterized by pervasive impairment in several areas of development: social interaction, communication skills, and repertoire of interests and activities. Thus far there have been no neuropathological findings nor laboratory/performance based measures providing construct validity to the diagnosis. It is not surprising that given the complexity of the clinical symptoms researchers have claimed abnormalities in widely divergent areas of the brain in autism. In the absence of pathognomonic abnormalities clinical research in autism has been guided by a variety of ideologies and epistemological assumptions each contributing to the development of explanatory models or "theories": executive function (Ozonoff et al., 1997), "weak central coherence" (Frith and Happe, 1994), complex information processing (Minshew et al., 1997), "theory-of-mind" (Baron-Cohen et al., 1985), and empathy (Baron-Cohen, 2004). By themselves these theories are incapable of accounting for all of the developmental, social, cognitive and affective variables which define autistic psychopathology. Genetic factors in autism should definitely be considered as well. Autism is thought to be influenced by multiple genes as well as environmental factors thus providing for multifactorial inheritance (Muhle et al, 2004). Accordingly, there is no singular causal pathway to autism.

Therefore, an integrative framework capable of relating available theoretical constructs appears justified. Recent attempts at deriving such an overarching meta-theory have focused on a basic abnormality of neural connectivity (Belmonte et al., 2004). This model is empirically based on lack of coordinated brain activity and abnormal "binding" in the brains of autistic patients that can be detected with EEG methodology (Brock et al., 2002; Brown, 2005). According to Baron-Cohen & Belmonte (2005), the combination of local sensory hyperarousal and low-level over-processing of incoming sensory stimuli concurrent with abnormalities of attention selectivity and focus, may be a consequence of the over-connected low-level processing neural networks in autism spectrum disorders. In such over-wired networks signal is insufficiently differentiated from noise or task-irrelevant information, and as a result information capacity is drastically reduced (Belmonte et al., 2004; Casanova, 2006; Rubinstein and Merzenich, 2003). Higher-than-normal noise in cortical processes also affects normal development of differentiated representations, because cortical response selectivity in space and time is a product of balanced inhibitory and excitatory processes. Such over-representation by non-differentiated systems could plausibly account, for example, for the strong aversive reactions to auditory, tactile, and visual stimuli that are commonly recorded in autistic individuals. The abnormal long-range neural connectivity model is suggested to explain deficits in high-level complex information processing functions where rapid and integrated operation of many separate neural systems is required (Minshew et al., 1997; Welchew et al., 2005). In the autistic brain, high local connectivity may develop along with deficient long-range connectivity.

In recent years, neuropathological studies of autism have revealed abnormalities in several brain regions. Changes in brain size with widespread increases in both gray and white matter volumes suggest that the underlying pathology in autism consists of widely distributed histological abnormalities. The available neuropathological and structural imaging data suggest that autism is the result of a developmental lesion capable of affecting normal brain growth. One possible explanation for this is the recent finding of minicolumnar abnormalities in autism, in particular demonstration of minicolumns of reduced size and increased number in the autistic brain (Casanova et al., 2002ab, 2006ab). The increased number of minicolumns reported in autism suggests a possible disruption during the earlier stages of neurodevelopment in the brain of an autistic patient. Furthermore, a minicolumnar abnormality may translate difficulties in the integration of information into a delay in language acquisition. In all, minicolumnar abnormalities may incapacitate a patient as a social being by distorting elements of the child's biopsychological experience.

The modular arrangement of the cortex is based on the cell minicolumn: a self-contained ecosystem of neurons and their afferent, efferent, and interneuronal connections (Mountcastle, 2003). Our preliminary studies indicate that minicolumns in the brains of autistic patients are narrower, with an altered internal organization (Casanova, 2006). More specifically, their minicolumns reveal less space for inhibitory local circuit projections. A defect in these GABAergic fibers may correlate with the increased prevalence of seizures among autistic patients. Based on the descriptions given thus far, it is possible to propose a disruption of the normal balance between excitation and inhibition in the columnar organization of autistic patients. In this regard, a series of noteworthy studies report that both children and adults with autism were superior to a control group in their ability to discriminate novel, highly similar stimuli (Plaisted et al., 2003). Autistic children also have a superior ability in discriminating display items in visual search tasks; such enhanced discrimination in autism results from low-level perceptual processing of incoming stimuli, and this is called the bottom-up approach (O'Riordan et al., 2000).

It is well known that networks of inhibitory interneurons acting as GABA gated

pacemakers are critically involved in gamma EEG (30-80 Hz) oscillations (Grothe and Klump, 2000). Therefore, analysis of high frequency EEG oscillations in patients with autism may provide additional information about potential neural deficits in autism. Abnormalities in these mechanisms have been associated with binding problems (the co-activation of neural assemblies), which may be present in both autism and schizophrenia (Brock et al., 2002; Grice et al., 2001). Oscillatory activity in the gamma-band of the EEG has been related to Gestalt perception and to cognitive functions such as attention, learning, and memory (Kaiser, 2003). Electrophysiological studies show strong evidence that synchronized cortical activity in the gamma frequency range could be a correlate of feature binding to form a single coherent percept. Binding of widely distributed cell assemblies by synchronization of their gamma frequency activity is thought to underlie cohesive stimulus representation in the human brain (Kahana, 2006). According to this assumption, changes in gamma EEG activity have been considered indicators of processing of Gestalt-like patterns (Herrmann and Mecklinger, 2000, 2004; von Stein et al. 1999).

It has been proposed that "weak central coherence" (Frith and Happe, 1994) in autism could result from a reduction in the integration of specialized local networks in the brain caused by a deficit in temporal binding (Brock et al., 2002). Visual and auditory perception anomalies associated with weak central coherence may be attributed to a reduction in synchronization of gamma activity between networks processing local features; this may explain some of the features of language deficits, executive dysfunctions, and other impairments in social communication associated with autism. Brown (2005) tested adolescents with autism in an experiment which presented either shapes with visual illusions (Kanizsa figures, Kanizsa, 1976) or random pictures. When perceiving the shape, the group with autism differed from the controls in three ways: (1) gamma power was higher than in the control group; (2) the group with autism showed a very early burst of gamma activity between 80 and 120 ms; and (3) the later gamma (around 300 ms) component in the autistic patients was more powerful and started much earlier. The inability to reduce gamma activity according to Brown (2005) would lead to the inability to decide which event requires attention when there are multiple choices. Excessive gamma can therefore be linked to a reduction in the ability to focus attention. In autism, uninhibited gamma activity suggests that none of the circuits in the brain can come to dominance because too many of them are active simultaneously (Brown et al., 2005). The "temporal binding deficit" hypothesis of autism (Brock et al., 2002) suggests that many features of autism, such as superiority in processing detail (local processing) and disadvantages in global processing, can be explained by a failure of binding between cortical areas.

An increased and earlier burst of gamma activity in autism might be linked to increased ability to perform tasks in which ignoring contextual information is an advantage. The disadvantage of such an overactive system however lies in an inflexible re-focusing of the brain system onto events rapidly occurring in sequence. Abnormalities of gamma oscillatory synchrony in autism can therefore result in significant perceptual and cognitive deficits. In our own study on 12 children with autism and 12 age-matched controls we tested Brock's "temporal binding deficit in autism" hypothesis (Brock et al., 2002; Rippon et al., 2007) using a visual attention task with Kanizsa illusory figures as stimuli. In this task subjects have to respond with a button-press to rare (25% probability) Kanizsa squares (targets) among Kanizsa triangles (rare non-target distracters, 25% probability) and non-Kanizsa figures (standards, 50% probability).

Dependent measures in EEG gamma band were recorded continuously with an Electrical Geodesics Inc. 128-electrode array net, referenced to vertex. Extraction of induced gamma band power in single trials (0-800 ms post-stimulus) was performed with Morlet wavelet analysis using MATLAB. We selected several channels from the frontal, central, parietal and occipital areas to cover the whole head, in order to examine EEG gamma oscillations in various brain regions. We found that the power of gamma oscillations in response to non-target Kanizsa and non-Kanizsa standard stimuli was higher in the autism group at the left frontal (F1, F7), left and right parietal (P1, P2, P7, P8), and occipital (O1, O2) EEG channels. Figure 2 shows differences between induced early and late induced gamma activity in response to non-target and target Kanizsa figures.

Group (control, autism) differences in gamma oscillation power to non-target and target Kanizsa stimuli were better expressed over the lateral frontal (F7, F8) and parietal (P7, P8) EEG sites. At all recording sites the power of gamma to non-targets in the autism group was higher compared to controls. We found also that at the lateral frontal and parietal sites the difference between target and non-target stimuli was more negative in the autism group at the right hemisphere. There were certain anterior *vs.* posterior differences; most notably the power of gamma was similar in the control and autism groups at parietal sites, but higher in the autism group at the frontal sites. The most reproducible finding was that gamma induced by non-target stimuli was globally higher in autistic subjects compared to controls at all sites. Analysis of gamma coherence at 4 sites of EEG (F7, F8, P7, and P8) using BESA Coherence

Continued on page 36

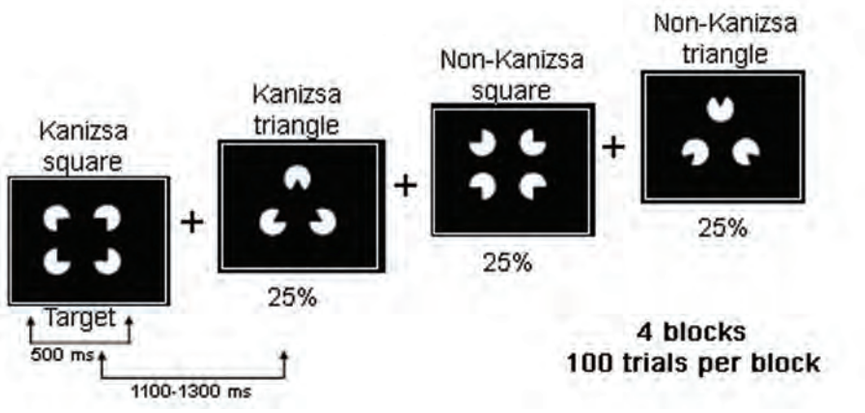


FIGURE 1. *Kanizsa and non-Kanizsa figures were used as stimulus material in this experiment. In particular, the stimulus types used in the experiment are Kanizsa square (target), Kanizsa triangle, non-Kanizsa square, and non-Kanizsa triangle. Non-target Kanizsa triangle was introduced for differentiation of processing Kanizsa figures and targets. The stimuli consisted of either three or four inducer disks which are considered the shape feature and either do or not constitute an illusory figure (square, triangle). This test with Kanizsa illusory figures is readily inducing gamma response during perceptual processing of stimulus (Brown, 2005; Herrmann and Mecklinger, 2000, 2004)*

Abnormalities in Autism continued from page 35

Tools revealed significantly lower coherence in the autism group. A *Hemisphere* (left, right) X *Topography* (anterior, posterior) X *Group* (autism, control) interaction indicated that the autism group had reduced coherence coefficient values at anterior (i.e., frontal) sites of the right hemisphere.

Our findings of excessive gamma oscillations and low inter- and intra-hemispheric coherence in response to non-target items is in agreement with other studies noting that neural systems in the brains of autistic patients are often inappropriately activated (Belmonte and Yurgelin-Todd, 2003a), and may be due to a disruption in the ratio between cortical excitation and inhibition (Casanova et al., 2002ab; Casanova, 2006; Rubenstein and Merzenich, 2003). In autism, increased gamma activity indicates that activity induced by perceptual processes starts earlier and continues longer, because the neural networks subserving cognitive processes involved in combining information processing are not functioning normally. A reduction in the ability to decrease gamma oscillations may reflect inhibitory deficits as well, resulting in difficulties directing attention. Earlier onset and enhanced gamma activity can be used by some individuals to improve their atypical perceptual abilities and demonstrate well-known islets of superior local processing of detail at the expense of reduced global processing abilities.

Enhanced, prolonged and weakly differentiated gamma responses to both target and non-target stimuli in sensory specific cortical areas (e.g., visual cortex at occipital EEG sites) and/or delayed gamma activation at the frontal integrative cortical areas suggests disrupted neural signaling (low functional connectivity), and supports the hypothesis of abnormal regional activation patterns (local over-processing vs. global under-processing). In addition to these failures of modulation of induced high frequency EEG oscillations in response to stimuli, the brain of autistic patients is over-activated and over-loaded with incoming sensory information. In general, perceptual filtering in autism seems to occur in an all-or-none manner, with little specificity for the location of the stimulus, the behavioral relevance of the stimulus, or the sensory modality. The attention of autistic patients seems founded more on the coarse control of general arousal than on selective activation of specific perceptual systems.

Currently our laboratory is looking into the therapeutic effects of low-frequency (i.e., inhibitory) repetitive transcranial magnetic stimulation (TMS) and neurofeedback on the excessive high-frequency EEG activity characteristic of individuals with autism. Our preliminary results using TMS were already published recently in the Journal of Autism and Developmental Disorders (Sokhadze, et al. 2009). Our study underway is open for enrollment and we continue research in this promising direction of neurotherapy based on TMS; we plan to add neurofeedback in the future development of our treatment protocol.

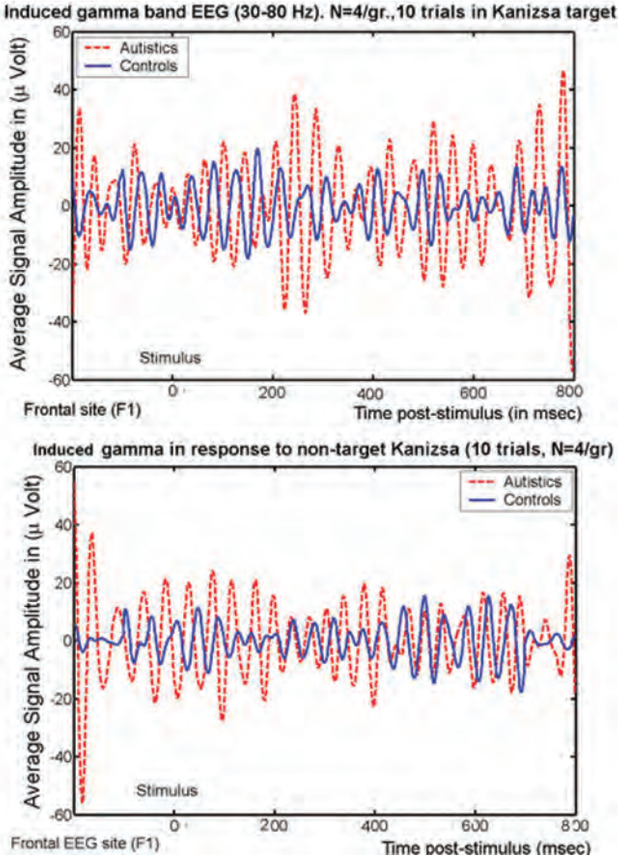


FIGURE 2. *Induced gamma frequency oscillations in response to Kanizsa target and non-target stimuli. Children with autism show higher power of the late (240-500 ms post stimulus) gamma oscillations in response to target Kanizsa stimulus, and higher power of early (40-180 ms post-stimulus) gamma oscillations in response to non-target Kanizsa stimulus.*

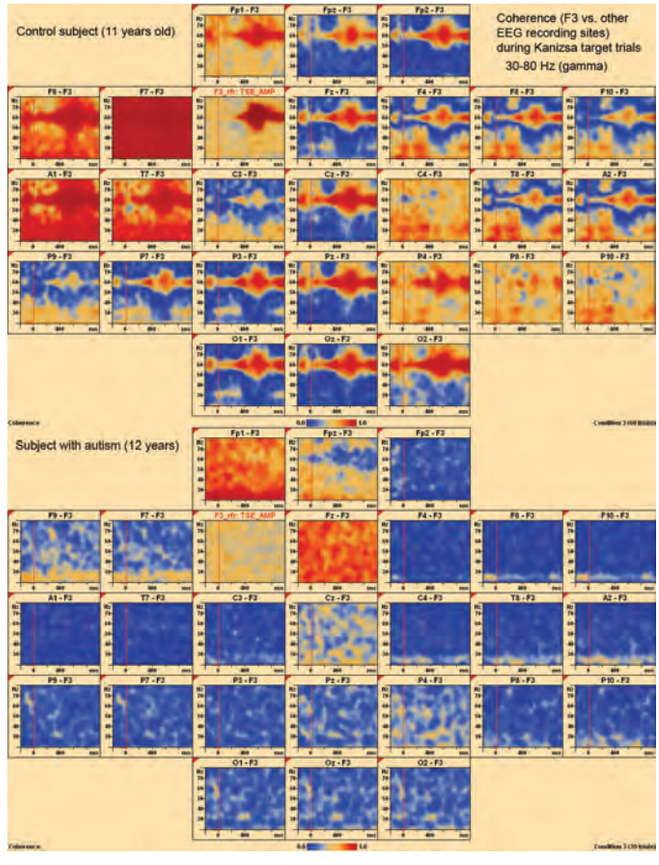


FIGURE 3. *Coherence of scalp EEG gamma oscillations in response to target Kanizsa stimulus in a typical child and in a patient with autism. Coherence is analyzed using the Coherence Module of BESA (Brain Electric Source Analysis) software (MEGIS Software, GmbH, Germany). Left frontal site (F3) shows high hemispheric phase coherence in control subject, but not in patient with autism.*

REFERENCES:

Baron-Cohen, S. (2004) The cognitive neuroscience of autism. J Neurology Neurosurgery Psychiatry, 75, 945-948.

Baron-Cohen, S., and Belmonte, M.K. (2005) Autism: a window onto the development of the social and the analytic brain. Annu Rev Neurosci. , 28, 109-126.

Baron-Cohen, S., Leslie, A.M., and Frith, U. (1985) Does the autistic child have a "theory of mind"? Cognition, 21, 37-46.

Belmonte, M.K., and Yurgelun-Todd, D.A. (2003) Functional anatomy of impaired selective attention and compensatory processing in autism. Cognitive Brain Research, 17, 651-664.

Belmonte, M.K., Allen, G., Beckel-Mitchener, A., Boulanger, L., Carper, R. and Webb, S.J. (2004) Autism and abnormal development of brain connectivity. Journal of Neuroscience, 24, 9228-9231

Brock, J., Brown, C.C., Boucher, J., and Rippon,G. (2002) The temporal binding deficit hypothesis of autism. Dev Psychopathol., 14, 209-224.

Brown, C. (2005) EEG in autism: is there just too much going on in there? Manuel F. Casanova (Ed.), Nova Science Publishers, New York, pp 109-126.

Brown, C., Gruber, T., Boucher, J., Rippon, G., and Brock, J. (2005) Gamma abnormalities during perception of illusory figures in autism. Cortex, 41, 364-376.

Casanova, M.F. (2006). Neuropathological and genetic findings in autism: the significance of a putative minicolumnopathy. Neuroscientist, 12(5), 435-441.

Casanova, M.F., Buxhoeveden, D.P., Switala, A.E.,and Roy E. (2002a) Minicolumnar pathology in autism. Neurology, 58, 428–32.

Casanova, M.F., Buxhoeveden, D.P., Switala ,A.E.,and Roy E. (2002b) Neuronal density and architecture (gray level index) in the brains of autistic patients. J Child Neurol, 17, 515–521.

Casanova, M.F., van Kooten, I., van Engeland, H., Heinsen, H., Steinbursch, H.W.M., Hof, P.R., Trippe, J., Stone, J., and Schmitz, C. (2006a) Minicolumnar abnormalities in autism II. Neuronal size and number. Acta Neuropathologica, 112(3), 287-303

Casanova, M.F., van Kooten, I., Switala, A.E., van England, H., Heinsen, H., Steinbuch, H.W.M., Hof, P.R., and Schmitz, C. (2006b) Abnormalities of cortical minicolumnar organization in the prefrontal lobes of autistic patients. Clinical Neuroscience Research, 6(3-4), 127-133

Frith, U., and Happé, F. (1994) Autism: Beyond theory of mind, Cognition, 50, 115–132.

Grice, S.J., Spratling, M.W., Karmiloff-Smith, A., Halit, H., Csibra, G., de Haan, M., and Johnson, M.H. (2001) Disordered visual processing and oscillatory brain activity in autism and Williams Syndrome. Neuroreport, 12, 2697-2700.

Grothe, B., and Klump, G.M. (2000) Temporal processing in sensory systems. Curr Opin Neurobiol. 10, 467-473.

Herrmann, C.S., and Mecklinger, A. (2000) Magnetoencephalographic responses to illusory figures: early evoked gamma is affected by processing of stimulus features. International J Psychophysiology, 38, 265-281.

Herrmann, C.S., Munck, M.H.J., and Engel, A.K. (2004) Cognitive functions of gamma-band activity: memory match and utilisation, Trends in Cognitive Sciences , 88, 347–355

Kahana, M.J. (2006) The cognitive correlates of human brain oscillations. The Journal of Neuroscience, 26, 1669-1672.

Kaiser, J. (2003) Induced gamma-band activity and human brain function. The Neuroscientist, 9, 475-484.

Kaiser, J., Hertrich, I., Ackermann, H., Mathiak, K., and Lutzenberger, W. (2005) Hearing lips: Gamma-band activity during audiovisual speech perception. Cerebral Cortex, 15, 646-653.

Kanizsa, G. (1976) Subjective contours, Sci. American, 235, 48-52.

Minshew, N.J., Goldstein, G., and Siegel, D.J. (1997) Neuropsychologic functioning in autism: profile of a complex information processing disorder. J Int Neuropsychol Soc. 4, 303-316.

Mountcastle, V.B. (2003) Introduction. Computation in cortical columns. Cereb Cortex, 13, 2-4.

Muhle, R., Trentacoste, S.V., and Rapin, I. (2004) The genetics of autism. Pediatrics. 13, 472-486

O'Riordan M. (2000) Superior modulation of activation levels of stimulus representations does not underlie superior discrimination in autism. Cognition. 77(2):81-96.

Ozonoff, S. (1997) Casual mechanisms of autism: unifying perspectives from an information-processing framework. In: D.J. Cohen and F.R. Volkmar, Editors, Handbook of autism and pervasive developmental disorders, John Wiley, New York , pp. 868–879.

Plaisted, K., Saksida, L., Alcántara, J., and Weisblatt, E. (2003) Towards an understanding of the mechanisms of weak central coherence effects: Experiments in visual configural learning and auditory perception, Philosophical Transactions of the Royal Society of London B , 358, 375–386.

Rippon, G., Brock, J., Brown, C., and Boucher, J. (2007) Disordered connectivity in the autistic brain: Challenges for the 'new psychophysiology', International Journal of Psychophysiology, 63, 164-172

Rubenstein, J.L., and Merzenich, M.M. (2003). Model of autism: increased ratio of excitation/inhibition in key neural systems. Genes Brain Behav 2, 255–67.

Sokhadze, E., Singh, S., El-Baz, A., Baruth, J., Mathai, G., Sears, L., and Casanova M. (2009) Effect of a low-frequency repetitive transcranial magnetic stimulation (rTMS) on induced gamma frequency oscillations and event-related potentials during processing of illusory figures in autism spectrum disorders. Journal of Autism and Developmental Disorders, 39, 619-634.

Von Stein, A., Rappelsberger, P., Sarnthein, J., and Petsche, H. (1999) Synchronization between temporal and parietal cortex during multimodal object processing in man. Cereb. Cortex, 9, 137-150.

Welchew, D.E., Ashwin, C., Berkouk, K., Salvador, R., Suckling, J., Baron-Cohen, S. and Bullmore, E. (2005) Functional disconnectivity of the medial temporal lobe in Asperger's syndrome, Biol. Psychiatry, 57, 991– 998.

TECH TALK

Interview with Dr. Robert Thatcher on the Evolution of his 19-channel Live Z-Score and LORETA Training System

Michael Gismondi, LMHC

MG: What constitutes the underlying brain organization that permits current 4-channel z-score neurofeedback (ZNF) to be clinically meaningful and efficacious? When a neurofeedback clinician uses ZNF, there is the assumption your normative database, and its real time mathematical transforms, will allow us to observe the many relationships *and* required balances *and* flag the key deviations between power variables along with measures of connectivity. How does your mathematical and statistical modeling of brain normalcy, and its deviations, find its neurological validation?

RWT: That's an important question, one that I have been pursuing, in one form or the other, for several decades. Recently, there has been a series of studies using diffusion MRI and fMRI, especially the work of Drs. Yong He and Hagmann and others that attempts to integrate what we know about the anatomical structure and function of the brain as we characterize its *network* or connectivity-based properties. This research speaks of 5 to 6 high-density "functional modules" with high local connectivity also referred to as "hubs" that are driven and coordinated by pacemakers, primarily the thalamus. The thalamus is of course at the center of cortical activity detectable via EEG. The hippocampus, septal regions, reticular formations, the ventral tegmental areas are all examples of additional pacemaker areas that drive or contribute to the different bands of EEG. But all the EEG frequencies we can detect and relate back to anatomical sources are "nested" and functionally interrelated; they are not separate or random. Walter Freeman and others have shown that when you plot the EEG spectrum on a log-log scale it is a one over "F" distribution; it's a straight line above two hertz.…below that we are capturing mostly glia cell activity, but from 2 to 100 hz it's a straight line. The low frequencies, from 2 to 9hz, are the most "global" or far-reaching from a spatial and temporal view; their effects ripple through the entire spectrum. Higher frequencies are progressively more "local" and spatially constrained. These various "functional modules" engage in both local and global processing of neural information, and they are always talking to each other.

MG: Could you explain the meaning and significance of Freeman's "straight line" distribution of EEG values from 2 to 100 hz?

RWT: The straight line in the 1/f distribution is a scaling function and depending on the slope, provides a measure

Continued on page 38

Tech Talk
continued from page 37

of nonlinear dynamics in that if one knows something about a short segment of the line at a given frequency then one knows the entire spectrum. It also means that the higher frequencies are nested within the low frequencies with a spatial scaling in that the lower frequencies are global excitability oscillations that bind some of the neurons in all of the modules in the same time frame and nested time frames.

MG: Some of the modules address auditory and somatosensory information processing, some address visual information processing, some address attention control, and still others look at the deeper limbic system structures, and then there is the concept of the "default hub." Connectivity hubs and modules with their own unique neural structure and function would seem to give us another way to approach the placement of electrodes for ZNF.

RWT: That's right, but when we take surface EEG recordings, we are actually gathering information on how these modules interrelate and function jointly; we gather information on how well the brain as a whole is maintaining homeostasis. Z-scores reduce the complexity we are seeing by bringing together all the various metrics of activation and connectivity and integrating them into one common framework. In pathology, these hubs or modules become disregulated; compensations can then be observed in the EEG. Various systems in the brain are now out of balance. We can combine the various measures and normalize them, adjusting for the various intercorrelations. We now have a way to determine when the brain is moving towards greater balance and regulation. But at the same time, we must be mindful of what the client's key symptoms are, and what parts of the brain are implicated via the observed loss of function. A symptom IS a loss of function; Luria and others taught us that. When there is a loss of function, there is an effort on the part of the brain to compensate, and you want to be able to separate the two out, and not train them or treat them in the same manner. In other words, you want to be able to distinguish between the weakened system variables that need to be addressed and the compensatory activities that are still needed and probably need to be left alone. The reasoning is as you increase the connectivity and/or the appropriate activation levels of the weakened functional circuit, the compensatory activities found in the EEG which also appears to be out of range or even symptomatic now normalize on their own.

Another way of saying that is ZNF is compromised if it isn't guided by a careful assessment and a tight linking of complaint with lost function to specific anatomical areas and their expectable EEG values in health and pathology…PLUS some idea of what the likely compensatory activity looks like in the EEG so you don't train it and pull the rug out from under the client's functioning, or at the very least, have an inefficient mode of treatment.

MG: You've stressed that hypercoherence was a major way in which we might observe, on the EEG level, the brain compensating for a loss of function

RWT: That is one sign I am aware of, extremes of variability is perhaps another, and I think of these EEG patterns as a loss of efficiency…a functional regression of sorts. A region of the brain has lost its ability to process information, locally and/or globally. The brain tries to "work harder" in the ways that it can. Neurofeedback, properly rendered, attempts to open up and re-connect the parts of the brain that the neuroscientific literature tells us are most responsible for the loss of function in the first place.

MG: I would imaging this approach takes on even greater importance when you are dealing with clients with disorders like autism or specific learning disabilities, where there is a complex interplay and juxtaposition of "islands of functioning" amidst a "sea of dysfunction".

RWT: Yes, with the additional possibility of compensatory skills and talents and behaviors. Our lab's most recent paper is a study on autism from the viewpoint of EEG phase reset, a new metric that goes deeper than coherence and phase in characterizing the actual workings of the brain in both pathology and in peak performance states.

MG: Where do your new NeuroGuide neurofeedback training module extensions fit into all this?

RTW: Everything I have developed and plan on releasing this year is designed towards one end; to help further constrain the interpretive complexity as we help the clinician get better at precisely localizing and characterizing the areas of lost or compromised function. For treatment to proceed as efficiently as possible, we need rapid, *seamless* access to whole brain QEEG data recorded simultaneously rather than piecemeal so that we can, at the very least, retain phase and coherence information across the entire brain system. We need to avoid the fundamental disconnects we see currently between QEEG assessment and training, where the QEEG is forced to be little more than a quickly outdated snapshot of a rich and dynamic motion picture. We need a common measurement system if we are to monitor training and measure progress. We need to be able to train with as many or as few electrode sites as the problem at hand requires. We need to make it fast and easy for the clinician to do a full re-mapping whenever it is needed and to then resume training in the same session. The new training system will be an add-on module to NeuroGuide Deluxe, and as a result, the seamless integration between 19-channel QEEG mapping and training will be assured.

MG: It's my understanding that you will at some point include a computerized symptom questionnaire that will link symptoms and client complaints to both spatial localizations of the presumed areas of lost function, based on the findings of the neuroscience research literature, not unlike Tom Brownback's CNC 1020 system.

RWT: We have developed our own symptom check list that is based on the fMRI, PET and EEG/MEG literature about hubs and modules as well as classical neuropsychological information about functional specialization. I have not had a chance to examine Tom's system yet, but I know that it is based on the scientific literature and that is what is important. We start with a symptom check list and predict likely brain modules and sub-modules and predict EEG scalp locations and then QEEG Z scores are compared to the predicted locations to produce a protocol for neurofeedback. An upgrade adds LORETA source localization Z scores to the statistics that generates a protocol where there is maximal overlap.

MG: In addition to what you've mentioned, two very important features of your new training system, originally inspired by some of Freeman and Buzaki's work, and now coming out of Walter Klimesch's lab, namely, phase reset training and cross frequency phase training. Could you describe this approach to the modification of cognitive performance?

RWT: Phase reset is made up of two components, phase lock and phase shift. Phase lock is positively correlated with present measures of coherence, which is a measure of stability in phase differences over time. Phase shift, the disengagement from a phase lock between sites, is negatively correlated to coherence. Phase shift is related to the functional circuit's ability to recruit momentary neural resources and processing pathways. Phase lock is the binding together of the recruited resources for a brief period as a commitment to a particular information processing task OR compensation, which is just another way to speak about function. Using our normative database of over 650 subjects, we have calculated norms for phase lock and shift. I view phase reset as more fundamental or an underlying measure of connectivity, especially in studies of cognitive performance and its rehabilitation. This is what is being found in the work of Paul Sauseng and Walter Klimesch, and Walter Freemen, and I am trying to put these findings to work, so to speak, in the context of a more powerful form of neurotherapy. Phase reset provides us with more detailed and precise information on how the functional modules talk to and work with each other, and in turn the modules give us a more precise way to characterize how symptoms are linked to the brain. In all of our studies at my lab relating EEG measures to types of pathology and their neuropsychological testing correlates, phase reset measures are consistently at or near the top of our predictors for not only pathology, but intellectual ability as well, which is a very exciting finding. Cross-frequency phase lock refers to the locking of two different frequency bands, for example, theta waves to beta and/or gamma waves in the course of cognitive function. We have a way to track these relationships and train them as needed.

MG: It's exciting that both Buzaki and Paul Sauseng will be presenting at ISNR in September, and in fact Dr. Sauseng is presenting right before your talk on phase reset and cross frequency phase locking. I know you have been referring to phase reset as "the mother of coherence." I have used the phase of "the machine code of cognition" to describe phase reset and the more fundamental information it provides.

RWT: I am looking forward to the many discussions that come out of that and to learn new things and receive feedback on our 19 channel Z score neurofeedback methods. The new training system, which we are presently calling "NeuroGuide Feedback" or simply "NF," will be released in three phases and will also be presented at the ISNR meeting.

NF-1 is a 19 channel surface EEG recording using real-time Z scores and seamless integration of QEEG and neurofeedback and includes the computerized symptom checklist with corresponding anatomical and QEEG values to develop a protocol that as a guide to what fits the patient's symptoms and complaints. We hope to release this module around the end of June, 2009 with updates over the next several years.

NF-2 adds the LORETA Z score assessment to further constrain and localize the determination of the areas of functional loss that need to be addressed.

NF-L adds the LORETA 3-dimensional neurofeedback capability so that we can more directly modify the functional modules via Brodmann areas and Laplacian feedback. This is also called Neuroimaging Neurofeedback in which one can modify specific components of hubs and modules with higher spatial accuracy than can be done with just surface EEG biofeedback. This is similar to fMRI Neuroimaging therapy but at a fraction of the cost and an order of magnitude higher temporal resolution. We hope to have a prototype of LORETA Z score neurofeedback around the time of the ISNR meeting in Indianapolis.

MG: So, with all that constraining and specifying of the lost function areas, it sounds like a lot of the targeting will be gradually automated for the neurotherapist.

RWT: Yes, but with the provision for clinician override and re-direction at all times. In the present context, we look for overlaps in what the symptoms tell us about areas of lost function, as illuminated by normative QEEG and LORETA. These areas of overlap help us begin to differentiate loss of function or areas of weakened function and probable compensatory activities. We are striving to provide the neurotherapist with better and better hypotheses, but his or her clinical reasoning remains in control..

Tower of London

continued from page 25

Cummings, J.L. (1993). Frontal-subcortical circuits and human behavior. *Archives of Neurology, 50*, 873-880.

Dennis, M. (1991). Frontal lobe function in childhood and adolescence: A heuristic for assessing attention regulation, executive control, and the intentional states important for social discourse. *Developmental Neuropsychology, 7*, 327-358.

Forbes, G. B. (1998). Clinical utility of the Test of Variables of Attention (T. O. V. A.) in the diagnosis of attention-deficit/hyperactivity disorder. *Journal of Clinical Psychology, 54*, 461-467.

Grattan, L.M., & Eslinger, P.J. (1991). Frontal lobe damage in children and adults: A comparative review. *Developmental Neuropsychology, 7*, 283-326.

Green, V. A., Pituch, K. A., Itchon, J., Choi, A., O'Reilly, M., & Sigafoos, J. (2006). Internet survey of treatments used by parents of children with autism. *Research in Developmental Disabilities, 27*, 70-84.

Hughes, C., Russell, J., & Robbins, T.W. (1994). Evidence for executive dysfunction in autism. *Neuropsychologia, 32*, 477-492.

Jarusiewicz, E. (2002). Efficacy of neurofeedback for children in the autistic spectrum: A pilot study. *Journal of Neurotherapy, 6*(4), 39-49.

Kaiser, D. A. (2006, September). *Functional connectivity of ADHD and Asperger Syndrome Children.* Poster session presented at the annual meeting of the International Society of Neuronal Regulation, Denver, Colorado.

Kleinhans, N., Akshoomoff, N., & Delis, D. C. (2005). Executive Functions in Autism and Asperger's Disorder: Flexibility, Fluency, and Inhibition. *Developmental Neuropsychology, 27*(3), 379-401.

Klin, A., McPartland, J., & Volkmar, F. R. (2005). Asperger syndrome. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (3rd ed.). *Handbook of autism and pervasive developmental disorders, Vol. 1: Diagnosis, development, neurobiology, and behaviour* (pp. 88-125). Hoboken, NJ, US: John Wiley & Sons Inc.

Lawson, J., Baron-Cohen, S., & Wheelwright, S. (2004). Empathizing and systemizing in adults with and without Asperger syndrome. *Journal of Autism & Developmental Disorders, 34*, 301-310.

Lezak, M.D. (1995). *Neuropsychological assessment* (3rd ed.). New York: Oxford University.

McPartland, J., & Klin, A. (2006). Asperger's syndrome. *Adolescent Medicine Clinics, 17*, 771.

Owen, A.M. (1997). Cognitive planning in humans: Neuropsychological, neuroanatomical and neuropharmacological perspectives. *Progress in Neurobiology, 53*, 431-450.

Reid, A. (2005). Autistic Spectrum Disorders: Assessment and Intervention Results after Neurofeedback in 146 Cases. Student Award Presentation, International Society for Neuronal Regulation annual meeting. Denver, Colorado, September, 2005.

Ross, E. D. (1981). The Aprosodias: Functional-Anatomic Organization of the Affective Components of Language in the Right Hemisphere. *Archives of Neurology, 38*, 561-569.

Shallice, T., & Burgess, P.W. (1991). Deficits in strategy application following frontal lobe damage in man. *Brain, 114*, 727-741.

Solnick, B. (2005). Effects of electroencephalogram biofeedback with Asperger's syndrome. *International Journal of Rehabilitation Research, 28*(2), 159-163.

Thompson L. & Thompson M. (1995). Exceptional Results with Exceptional Children. *Proceedings of the Society for the Study of Neuronal Regulation.* Annual Meeting: Scottsdale, Arizona.

Thompson, M. & Thompson, L. (2003). *The Neurofeedback Book: An Introduction to Basic Concepts in Applied Psychophysiology.* Association for Applied Psychophysiology, Wheat Ridge, Colorado.

Thompson, M., & Thompson, L. (2005). Improving Attention in Adults and Children: Differing Electroencephalography Profiles and Implications for Training. *Biofeedback, 34* (3), 99-105.

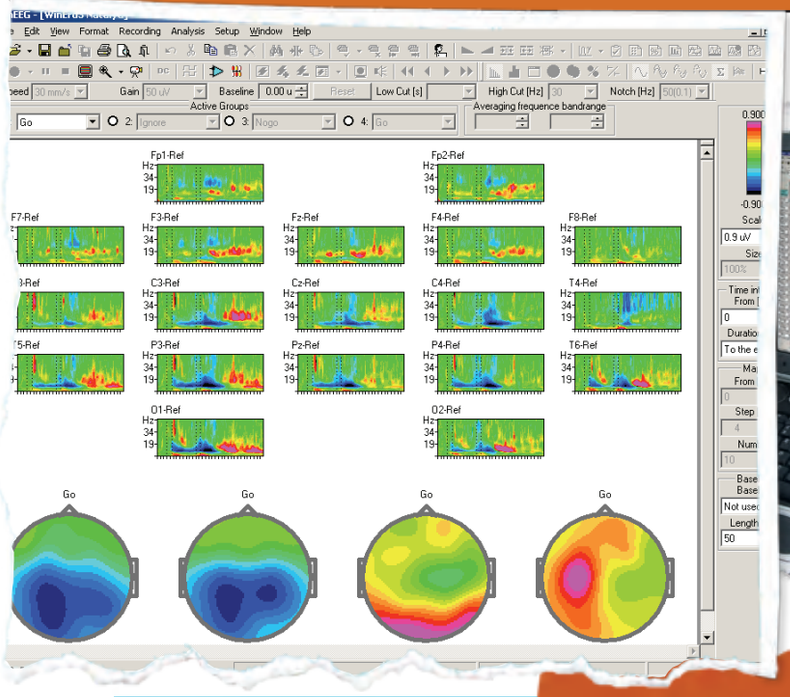
Thompson, L. & Thompson, M. (2007). Autistic Spectrum Disorders including Asperger's syndrome: EEG and QEEG findings, results, and neurophysiological rationale for success using neurofeedback training. Presented at the 11th Annual Meeting of the Biofeedback Foundation of Europe, Berlin, Germany. Abstract reprinted in *Applied Psychophysiology and Biofeedback, 32*(3-4), 213-214.

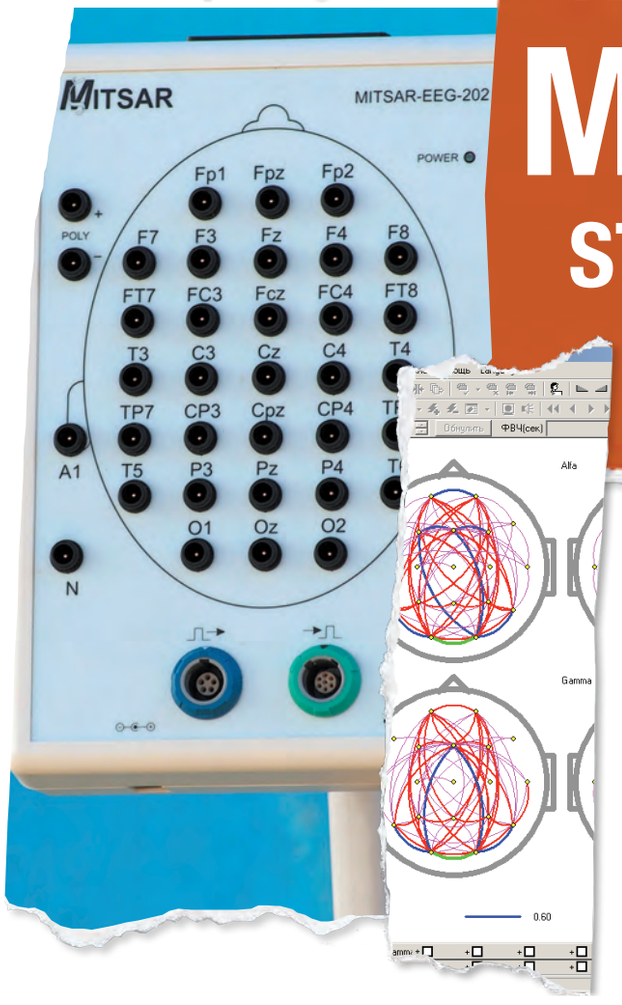
Thompson, M., Thompson, L., & Reid, A. (in press). *Neurofeedback Outcomes in Clients with Asperger's Syndrome.* Journal of Applied Psychophysiology and Biofeedback.

Wing, L. & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders, 9*, 11-29.

NOVA TECH EEG

WWW.NOVATECHEEG.COM 480.280.6938





MITSAR

STARTING AT $6500

MITSAR - EEG

21 AND 32 CHANNEL EEG AMPLIFIERS (AC & DC VARIANTS)

WINEEG SOFTWARE

SOFTWARE FOR QEEG/ERP AND LORETA ANALYSIS

HBI NORMATIVE DATABASE

NORMATIVE DATABASE FROM 6-80 YEARS OLD FOR QEEG AND ERP