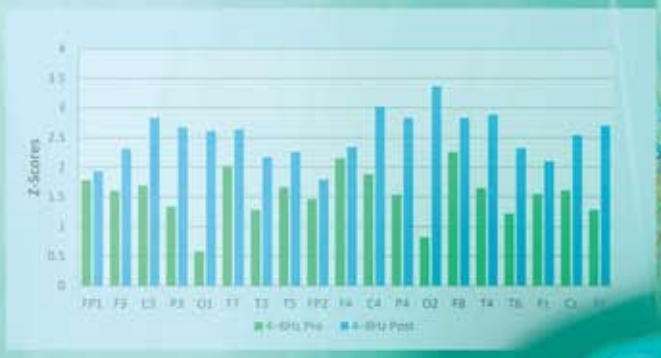
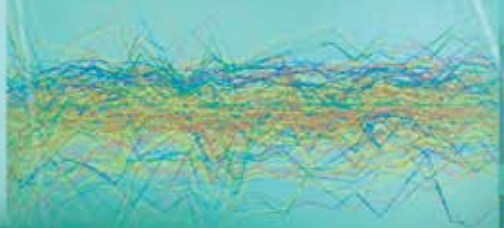
NEURO CONNECTIONS Newsletter

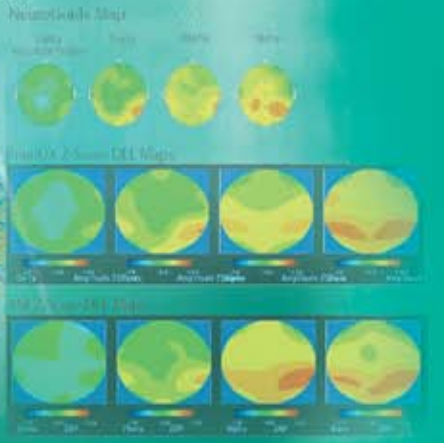
Spring 2014

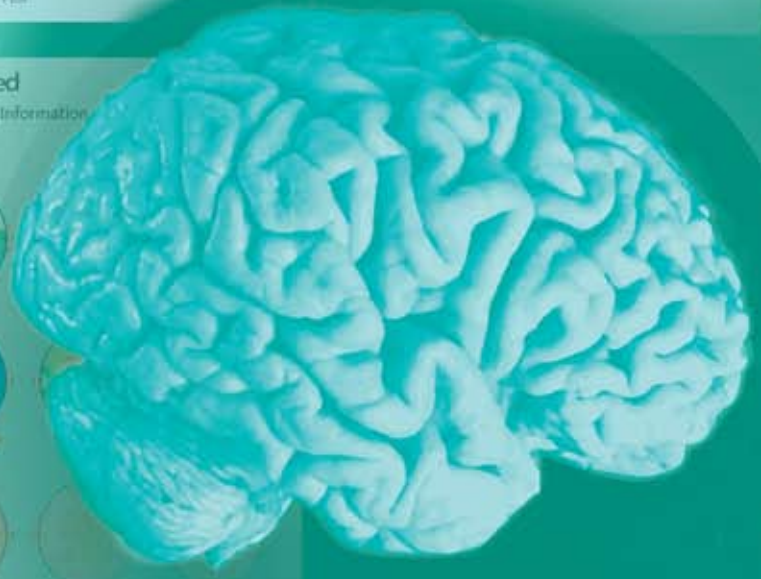
ISNR aapb

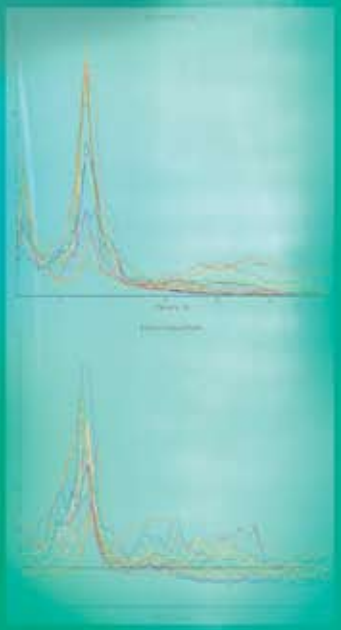
A joint newsletter from the *International Society for Neurofeedback & Research* and the *Association for Applied Psychophysiology & Biofeedback, Neurofeedback Section*

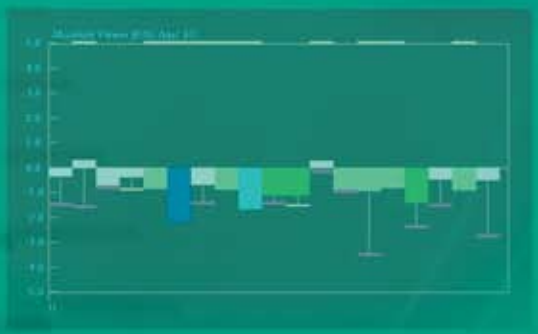












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NeuroConnections is published four times a year and will consider all materials pertaining to the practice and/or promotion of neurofeedback.

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ISSN 2151-6987 (print)
ISSN 2151-6995 (online)

NEURO CONNECTIONS

A joint newsletter from the ISNR & the aapb Neurofeedback Section

Contents

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

Ronald J Swatzyna, PhD, LCSW, BCN

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

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

Diagnosing ADHD

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

Prediction of ADHD Medication Response
via Neurobiomarker Profiling

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

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

Neurobiomarkers in ADHD

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

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

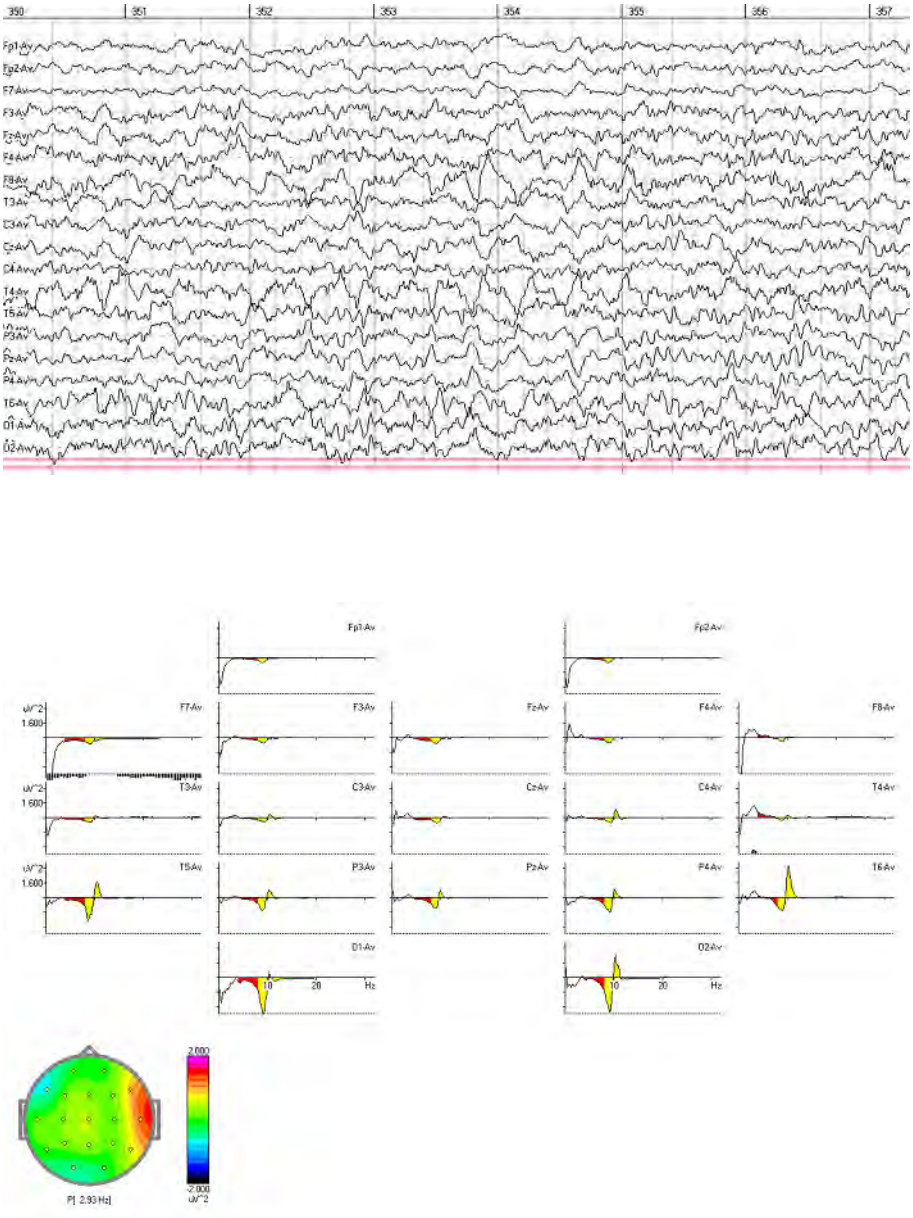

Figure 1: *Focal slowing in the EEG of a 12 y/o male, with diagnoses of Dx ADHD combined type & autism spectrum disorder.*

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

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

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

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

Neurobiomarkers in Refractory ADHD

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

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

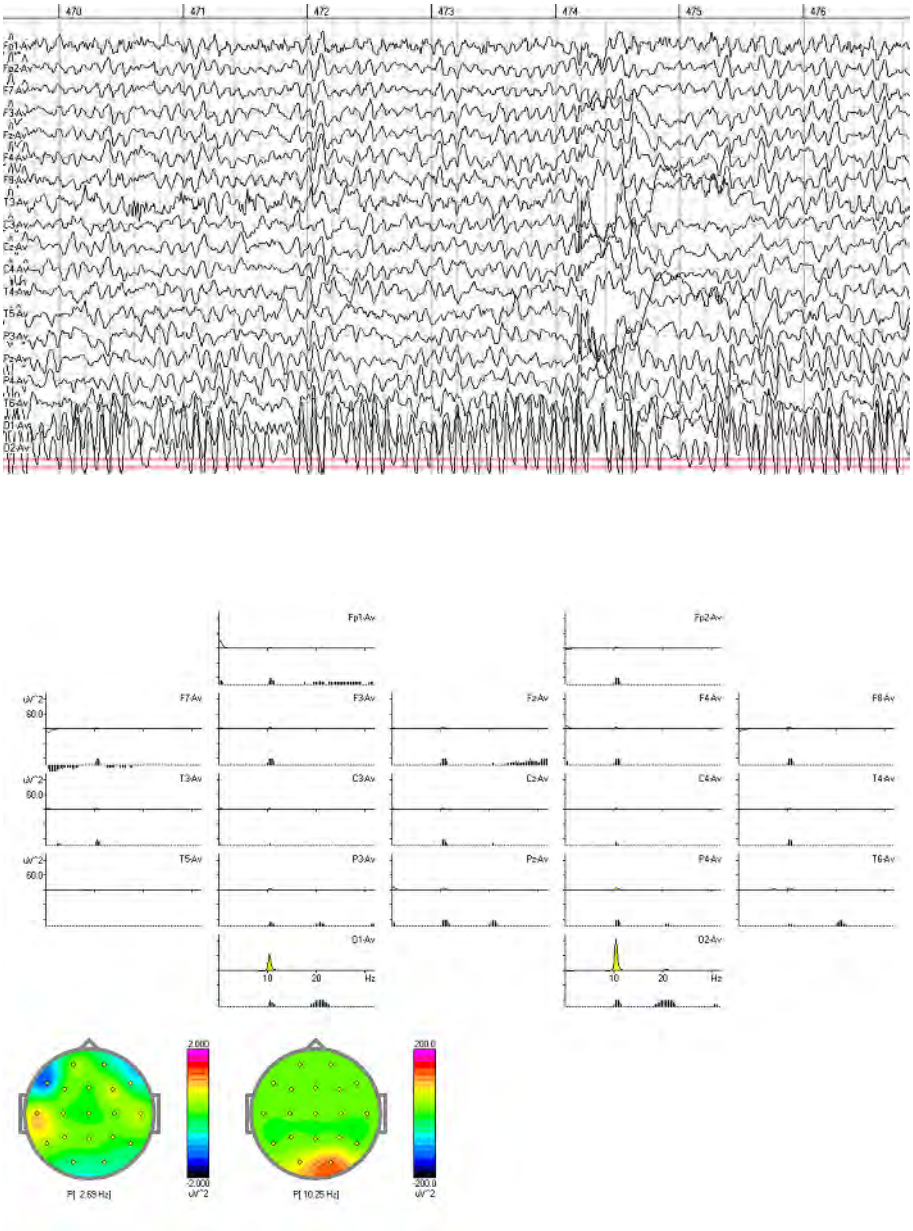

Figure 2: *Transient discharges recorded in the EEG of a 9 y/o female with history of ADHD & LD.*

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

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

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

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

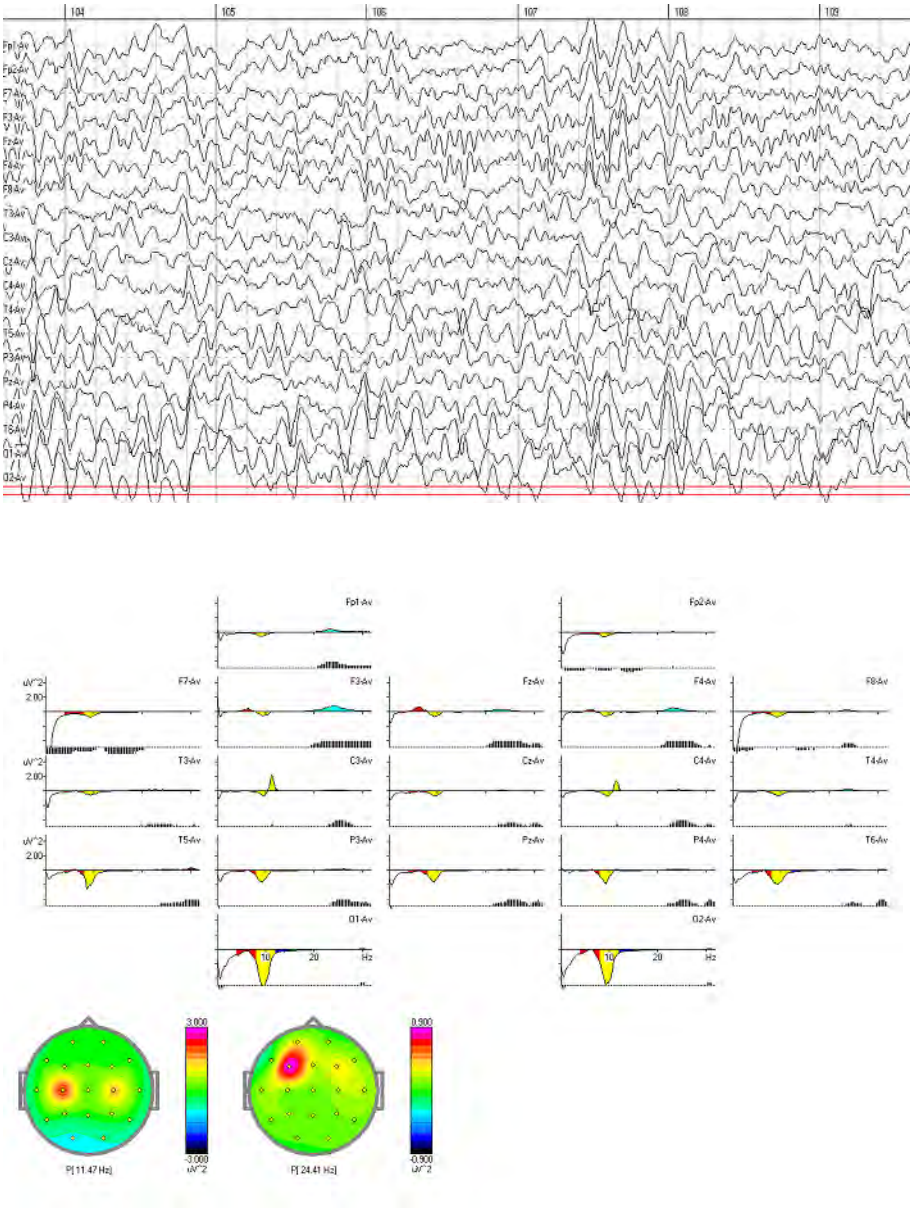

Figure 3: *Beta Spindles in the EEG of a 13 y/o male; Dx: ADHD combined type & generalized anxiety disorder.*

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

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

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

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

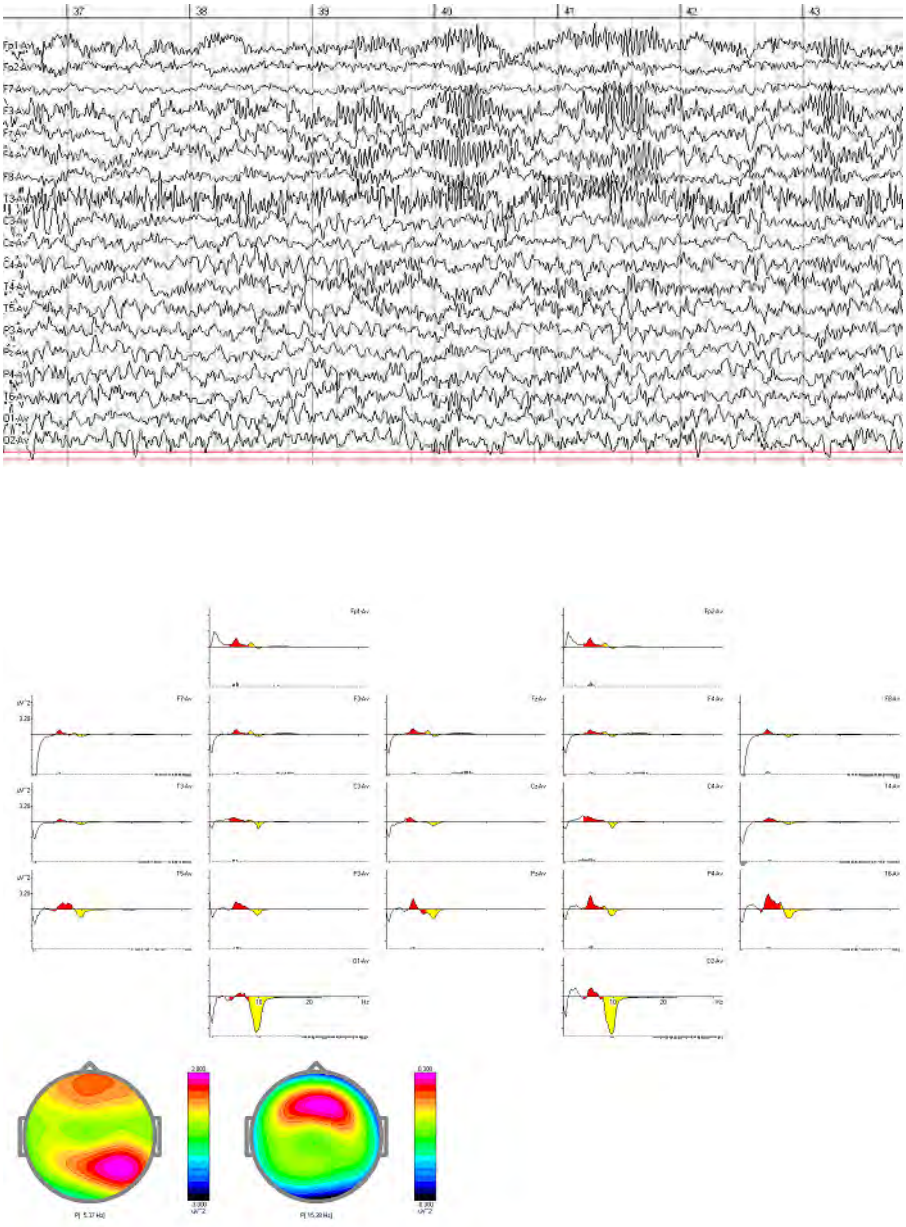

Figure 4: *8 y/o Female with suspected toxic encephalopathy. Dx: ADHD combined type and Mood Disorder, NOS.*

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

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

Summary

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

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

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

About the Author

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

Acknowledgement:

I appreciate the helpful comments from my colleagues Jay Gunkelman (www.brainsinternational.com), Drs. Gerald P. Kozlowski (www.gpkozlowski.com), Jay D. Tarnow (www.tarnowcenter.com), and Meyer Proler (www.brainsinternational.com).

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