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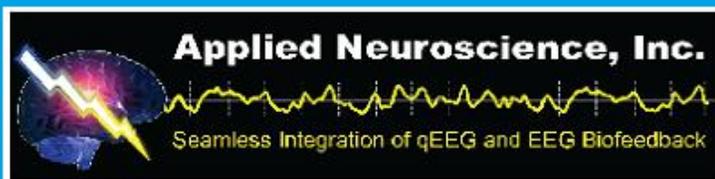
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PROCEEDINGS OF THE 2009 SABA CONFERENCE

Abstracts of Conference Presentations at the 2009 Society for the Advancement of Brain Analysis (SABA) 8th Annual Conference, Marineland, Saint Augustine, Florida

The 8th annual conference for the Society for the Advancement of Brain Analysis (SABA) was held in Marineland, Saint Augustine, Florida, May 1 to 5, 2009, and was sponsored by the Whitney Laboratory for Marine Bioscience, University of Florida. SABA is a daughter group of ISNR, a nonprofit membership organization of educators, researchers, and clinicians that is focused on psychological electroencephalography (EEG) with the purpose of integrating knowledge of brain structure and function in the practice of neurotherapy and psychological evaluation. SABA actively supports research in EEG technology, methodology, and interpretation in the context of EEG rhythm operant conditioning (neurofeedback) and neuromodulation, and this year's conference focused on both structural and functional neuroplasticity.

*David A. Kaiser, PhD
Editor*

Real-Time fMRI Feedback Training

Mario Beauregard, PhD

It has now become possible to image the functioning of the human brain in real time using functional MRI (rtfMRI) and thereby to have access simultaneously to both sides of the mind/brain, that is, subjective experience and objective quantitative measurements of brain activity. RtfMRI methods are reviewed, as well as recent rtfMRI studies showing learned control over localized brain activity involved in various functions (e.g., sensory and motor processing, pain and emotion regulation). The potential therapeutic

applications of this new neuroimaging approach are also discussed.

Can Models of Homeostatic Plasticity Explain Nervous System Functional Stability?

Dirk Bucher, PhD

A neuron's identity and function is determined by its morphology, the densities and spatial distribution of its specific types of receptors and ion channels, and its synaptic connections within the network. These features are all subject to dynamic regulation and must be matched to the functional

requirements in the face of changing environmental and behavioral demands, both during growth and development, and in adult life. Homeostatic mechanisms are needed to ensure that dynamic changes occur only within certain boundaries that keep neuron and network activity in a functional range. We are only beginning to understand how nervous systems strike a balance between altering individual neurons and synapses in the name of plasticity while maintaining long-term stability in neuronal system function. Our research focuses on the question of how stability of network function is achieved through regulation of neuronal properties, including morphology, synaptic, and intrinsic membrane properties.

What Can a Lazy Fish Teach Us About Synaptogenesis?

Kimberley Epley, PhD

Our goal has been to elucidate the basic principles of synapse function and development using zebrafish as a model system. We use mutant fish lines that show abnormal behavior. These fish have defects in the way neural excitation is translated into movement. Because zebrafish develop rapidly inside transparent eggs, we can analyze their neural function before they die. In addition, the transparency of the embryo itself enables optical studies, tracing individual proteins marked by genetic methods through development *in vivo*. We take advantage of these merits that the zebrafish system provides to pursue the following projects. Current projects in the lab center around two locomotory mutants we found to have defects in two key molecules of the neuromuscular synapse. One lacks acetylcholine receptors (AChR) in the muscle. As a result, the fish cannot mount a movement when the motor neuron releases ACh. The other mutant has a dysfunctional rapsyn. Rapsyn is a postsynaptic protein that brings AChRs together. In this fish, AChRs do not make clusters at the synapse and are diffusely distributed over the muscle cell surface. From the AChR-less mutant, we found that AChRs, which were thought to be passive players in synapse

formation, play an active role, directing rapsyn molecules to the synapse. In the rapsyn mutant fish, we found that not only do AChRs fail to form clusters at the synapse, but their functions are also altered. That is, when motor neurons fire at a high frequency, the amplitude of AChR current remains constant in wild type, whereas in rapsyn-mutant fish the response shows a marked attenuation with repeated firing of motor neurons.

Brain Laterality, Brain States and ADHD

Sigi Hale, PhD

The left and right cerebral hemispheres differ with respect to gross and functional anatomy (i.e., white-fiber systems and associated network properties) as well as microanatomy (i.e., cytoarchitecture). Moreover, it is well established that each hemisphere makes unique contributions to cognitive function: the left hemisphere (LH) linguistically encodes sensory information and the right hemisphere (RH) uses a more configural coding scheme. In other words we are endowed with a bimodal representation of our sensory world, and these representations compete for neural resources and lend themselves to distinct types of cognitive operations. Hence, adaptive functioning requires that we dynamically regulate their relative contributions during different operations.

We focus on the role of imbalanced utilization or integration of unihemispheric specialized information processing in ADHD. Several of our studies using converging measures of behavioral laterality, fMRI, and EEG have revealed mounting evidence that adults with ADHD demonstrate increased RH contribution during early stages of sensory information processing and that this is associated with compromised LH linguistic abilities and abnormal interhemispheric interaction. Moreover, we have found evidence that this is likely a brain-state associated phenomenon rather than reflecting inherent capacity. We suspect that increased RH relative to LH contribution during early stages of sensory processing is generally associated with ADHD symptoms and that what distinguishes ADHD-pathology from a more typical expression of such traits is a matter

of the frequency, duration, and/or adaptive expression of a RH biased mode of processing.

Toward this understanding we have recently found evidence of increased rightward alpha asymmetry in frontal and parietal regions of adults with ADHD during an eyes closed condition and during the Conner's Continuous Performance Task (CPT). Moreover, we have found robust rightward high beta asymmetry in parietal regions of adults with ADHD also during the CPT.

Instrumental Conditioning of Human Sensorimotor Rhythm (12–15 Hz) and Its Impact on Sleep as well as Declarative Memory Performance

*Kerstin Hoedlmoser, PhD,
Wolfgang Klimesch,
and Manuel Schabus*

The present study sought to clarify the effects of instrumental conditioning of sensorimotor rhythm (SMR; 12–15 Hz) in humans on sleep parameters during a 90-min midday nap as well as on declarative memory. Twenty-seven participants were randomly assigned to either 10 sessions SMR-conditioning or randomized-frequency-conditioning. Before and after this instrumental conditioning period, participants had to attend the sleep laboratory to take a 90-min nap and to perform a declarative memory task before and after sleep.

The three major findings are (a) the experimental design was successful in conditioning an increase in relative 12–15 Hz amplitude within 10 sessions ($d=0.7$), (b) the increased SMR activity is also expressed during subsequent sleep by eliciting positive changes in various sleep parameters (sleep spindle number [$d=0.6$], total sleep period [$d=0.7$], sleep onset latency [$d=0.7$]), and (c) this increased relative 12–15 Hz amplitude is associated with enhancement in declarative memory performance ($d=0.9$).

Results thus indicated that SMR frequency constantly increased over the 10 training sessions (in the SMR group only) and that this “shaping of one's own brain activity” also facilitated the expression of 12–15 Hz oscillations during subsequent sleep. Most interesting, these

electrophysiological changes were accompanied by profound positive sleep as well as memory performance.

The Periodicity Table: Introduction to Bimodulation and Entropy

David A. Kaiser, PhD

The Periodicity Table organizes spectral properties on number of signals, frequencies, and phase relationships (Kaiser, 2008). Of recent interest are the measures of bimodulation (Pearson product moment correlation of two frequencies at the same electrode site) and spectral entropy. Spectral entropy is a relative incidence measure from information sciences, which may be conceived as a measure of signal disorder or constituent variability. High spectral entropy occurs when activity is spread across most of the spectrum, and low spectral entropy is when activity is localized to a handful of frequencies, as seen in sleep and coma states. Nunes, Almeida, and Sleigh (2004) likened entropy to freedom of choice in that “conscious cortex is free to move among a huge number of available microstates” when entropy is high. Entropy indexes the number of possible microstate rearrangements, which can produce same macrostate. EEG rhythm training is discussed in terms of entropy training, increasing or reducing the possible accessible microstates as indicated by specific EEG rhythms. The value of these coefficients and related measures were discussed in terms of normative EEG assessment.

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Decision Making in Neurofeedback Protocol Selection

*David A. Kaiser, PhD,
and Penijean Rutter, MA*

Normative EEG analysis can provide dozens or even hundreds of statistical abnormalities for any individual given the large number of EEG sites, frequencies, and spectral coefficients examined by state-of-the-art techniques. General rules for prioritizing EEG findings are direly needed as we tailor our protocols to address specific behavioral or mental impairments. Two models for interpreting normative EEG are presented based on clinical practice and theoretical considerations. In the first model, the brain is conceived in terms of maturation, where functional and structural brain disorders reflect regression toward primitive brain behavior, that is, ontogenetic as well as phylogenetic immaturity. EEG indicators of immaturity include excessive delta, diminished connectivity, lack of functional differentiation in prefrontal cortex, reduced hemispheric specialization, and lack of coordination between anterior and posterior brain regions. In the second model, the brain is conceived as a reward-seeking machine in which all mental processes work to maximize reward within a limited resource system. Such resource allotment necessitates a hydraulic relationship between brain areas or systems. Whenever resources are allocation to one brain area or system they must be taken from another. With this in mind brain activity is organized along a number of dimensions including inhibition (output gating), meaning attribution (input gating), and recruitment. Disturbances in connectivity and evidence of hyper-recruitment or "resource hijacking" are viewed as likely candidates for EEG training.

Can Theories of Circuit Modification with Sensory Motor Rhythm Feedback Explain Our Remarkable Effects with Epilepsy?

*Denise Malkowicz, MD,
and Diana Martinez, MD*

Epilepsy is a disorder of recurrent seizures, paroxysmal abnormal electrical discharges arising from cortical neurons under the influence of thalamo-cortical circuits. Treatments with drugs and surgery have limited success, often with risk of serious side effects. Intensive SMR EEG feedback brain modification

training appears to promote neuroplasticity with desirable EEG changes, long-term potentiation (LTP), and reorganization of thalamo-cortical circuits, thereby altering neuronal networks and facilitating remediation from epilepsy. The present report reviews evidence for such an outcome in a patient with seizures secondary to brain injury.

After traumatic brain injury the individual had a 10-year history of severe refractory epilepsy with prolonged postictal states. He had failed multiple therapies. Initial QEEG showed diffuse delta and theta (+12 *z* score) and no 12–15 Hz sensorimotor cortex SMR. He underwent three spaced periods of intensive SMR EEG feedback training at C3–C4, with 5-month spans separating each of these periods. SMR EEG feedback resulted in seizure control within the first 3 weeks. He continued to improve in all areas of neurological function, including seizure control during the 5-month period between neurotherapy sessions. Posttraining QEEG showed normalization of delta and theta, increased SMR, and increased alpha and beta.

SMR feedback is associated with regulation of thalamo-cortical circuits and facilitates neuroplasticity through LTP, increasing neuronal protein synthesis, growth, and remodeling. This process continues to be robust, durable, and self-regenerating with clinical improvement seen between sessions. Previous studies by Serman et al. showed that SMR EEG feedback can reinforce and normalize thalamo-cortical circuits and result in LTP. In his animal studies, SMR training resulted in increased seizure thresholds when cats were exposed to epileptogenic compounds. Other studies in humans have shown improvement in seizure control with neurotherapy. Our participant is remarkable for the rapid and durable control of his refractory seizures with our intensive SMR EEG feedback protocol. It is concluded that intensive SMR EEG feedback training and consequent modification of thalamo-cortical and sensorimotor circuits through LTP remodeling seems to explain this remarkable effect on neuroplasticity and recovery from refractory epilepsy.

Genomic Bases of Neuronal Identity and Plasticity

Leonid Moroz, PhD

Our laboratory works to characterize basic mechanisms underlying the design of nervous systems and evolution of neuronal signaling mechanisms. The major questions are (a) why are individual neurons so different from each other, (b) how do they maintain such precise connections between each other, (c) how does this fixed wiring result in such enormous neuronal plasticity, and (d) how does this contribute to learning and memory mechanisms? By taking advantage of relatively simpler nervous systems of invertebrate animals as models, we combine neuroscience, genomics, bioinformatics, evolutionary theory, zoology, molecular biology, microanalytical chemistry, and nanoscience to understand how neurons operate, remember, and learn. As part of the NIH Center of Excellence in Genomic Sciences, our first project investigates the genomic basis of neuronal identity and plasticity. Because of the tremendous difficulties in mapping single cells and processes in the mammalian brain, we study the giant neurons of the sea slug *Aplysia californica*, a well-established model organism for cellular neuroscience. Our objective is to investigate nearly all messenger RNA (mRNA) involved in simple feeding and defensive networks.

A Nonpharmacological Alternative for the Treatment of Insomnia: Instrumental Conditioning of Brain Oscillations

*Manuel Schabus, PhD,
Kerstin Hoedlmoser,
and Wolfgang Klimesch*

Electroencephalographic recordings over the sensorimotor cortex show a very distinctive oscillatory pattern in a frequency range between 12–15 Hz termed sensorimotor rhythm (SMR). SMR appears to be dominant during quiet but alert wakefulness and synchronizes by the inhibition of motor behavior. This frequency range is also known to be abundant during light nonrapid eye movement sleep and is overlapping with the sleep spindle band. Given our recent findings

in a healthy population (Hoedlmoser et al., 2008) where we showed improved sleep quality and cognitive performance, we were encouraged to extend this approach to humans suffering from primary insomnia by again comparing instrumental conditioning (IC) of the SMR frequency band with a placebo control.

Twelve participants with primary insomnia (11 women; $M = 29.3$, $SD = 10.6$) attended the sleep laboratory 19 times (four nights, $10 \times$ SMR-IC, $5 \times$ placebo-IC). A counterbalanced, within-subjects design was used. Results confirmed the increase of 12–15 Hz activity over the course of the 10 SMR-IC training sessions ($p < .03$) but not over the course of the placebo-IC training sessions. Of interest, the increased SMR activity was associated with the enhancement of subjective sleep quality measured by the Pittsburgh Sleep Quality Index ($p < .01$). Furthermore, sleep onset latency was reduced after SMR-IC ($p = .056$) but not after placebo-IC. Therefore, we could show that people suffering from primary insomnia could benefit from SMR-conditioning as indicated by improved measures of subjective and objective sleep quality.

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The SMR Story

M. Barry Serman, PhD

This overview provides an updated review and integration of the neurophysiological rationale, implications for synaptic plasticity and learning, and basic and clinical methods and findings pertaining to brain modification training (BMT), formerly known as neurofeedback. It is based on documented findings, rational theory, and the research and clinical experience of the author. While considering general issues of physiology, learning principles, and methodology, it

focuses on the uniqueness of the sensorimotor rhythm (SMR) training, arguably the best established clinical application of EEG operant conditioning (BMT). The basic research literature provides ample data to support a very detailed model of the neural generation of SMR as well as the most likely candidate mechanism underlying its efficacy in clinical treatment. Further, although more controlled clinical trials would be desirable, a respectable literature supports the clinical utility of this alternative treatment for epilepsy. However, the skilled practice of clinical neurofeedback requires a solid understanding of the neurophysiology underlying EEG oscillation, operant learning principles, and mechanisms, as well as an in-depth appreciation of the ins and outs of the various hardware/software equipment options open to the practitioner. It is suggested that the best clinical practice includes the systematic mapping of quantitative multielectrode EEG measures against a normative database before and after treatment to guide the choice of treatment strategy and document progress toward EEG normalization. We conclude that the research literature reviewed in this article justifies the assertion that neurofeedback treatment of epilepsy/seizure disorders constitutes a well-founded and viable alternative to anticonvulsant pharmacotherapy.

Mapping Brain Networks with Mutual Information Analysis

Jason R. Soss, MD

Current emerging theories of brain function envision a model of cognitive function defined by coherent interactions of anatomically distant neuronal populations. This model is supported by many studies showing that brain activity, including consciousness and epilepsy, are correlated with firing rate synchronization across multiple neuronal assemblies. This neuronal activity is reflected in the EEG signal as the frequency component. Through measurements of the EEG signal at different regions, the functional network can be elucidated by determining the

degree of synchronous firing patterns between regions. Using mathematical techniques of mutual information analysis, we can propose the strength of interactions between regions and track these changes during tasks or events to determine what brain regions are responsible. Application of this technique has been used to determine the epileptic network for surgical treatment, evaluating participants' awareness for performance needs (i.e., airline pilots, truck drivers), studying the necessary brain functions for consciousness, and for brain/machine interfacing.

Biofeedback and Neurofeedback: Science or Fiction?

Lynda Thompson, PhD

This talk shares information that was presented at a National Institutes of Health (NIH) symposium on Mind–Body Medicine on May 7, 2009. The three areas discussed are Meditation, Biofeedback, and Tai Chi, and the goal is to bring the researchers at NIH up to speed with respect to biofeedback. The presentation underscores the fact that the practice of biofeedback developed from research findings and continues to be based on the careful application of learning procedures, especially operant conditioning techniques. There is a brief overview of the history of the field, dating back to the 1969 founding of the Biofeedback Research Society (forerunner of the AAPB). Clinical applications of biofeedback for various conditions are mentioned, with discussion of level of efficacy that has been established through research published in peer-reviewed journals. With respect to heart rate variability training, for example, there is established efficacy concerning asthma and hypertension with two or more large sample size, randomized, controlled trials for both of those conditions. The main emphasis is on neurofeedback training with particular mention of applications to seizure disorders and ADHD, which are the best validated applications. (The latest research from Europe—a German randomized controlled study establishing neurofeedback efficacy in ADHD and a Dutch meta-analysis of ADHD

studies—should help them realize that lack of American funding for biofeedback research has meant waiting for Europeans to validate a made-in-the-USA intervention.) In line with the symposium’s emphasis on health promotion through mind–body medicine, the data presentation concludes with a case study that highlights the combined approach of biofeedback and neurofeedback for effective stress management.