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KEYNOTE PRESENTATIONS

QEEG and Neurofeedback in the Assessment and Treatment of Psychological Disorders

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Neurofeedback therapy works by harnessing the natural plasticity of the brain—its ability to learn and adapt to the world, from childhood through to older adult age. There are many brain networks critical to neuroplasticity, working beneath the mind to shape the content of conscious awareness. Dysregulation of these networks can result from a myriad of factors—such as genetic predisposition, negative life experience, and hindered brain development. Such dysregulation will detrimentally affect how the brain processes our experiences, in ways the conscious mind cannot control.

This talk will discuss the functional architecture and electrophysiological underpinnings of neurofeedback therapy, together with a review of assessment and treatment methods. There will be (a) an examination of quantitative EEG as a parametric tool for assessing the regulation of brain systems function, (b) the role of neuropsychological assessment, and (c) coverage of theoretical viewpoints, methods, and applications of neurofeedback therapy, including its relationship to operant learning theory. The presentation will also discuss the relationship between neurotherapy and psychotherapy, emphasizing their complementary, respective roles in redressing dysregulation in the physical world of neurons and networks (neurotherapy) and the dysfunctional thoughts and

feelings that can emerge as a result (psychotherapy). Examples of the application of neurofeedback therapy will be presented with an emphasis on Attention-deficit/Hyperactivity Disorder (ADHD).

Neurobiology of Attention-deficit/Hyperactivity Disorder

Mark Bellgrove, PhD

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Attention-deficit/Hyperactivity Disorder (ADHD) is a prevalent mental health condition of childhood that frequently persists into adulthood. In this presentation I will overview our current knowledge regarding the neurobiology of ADHD, surveying the genetic, pharmacological, brain imaging, and neuropsychological literatures. I will argue that despite significant advances in our understanding of the pathophysiological mechanisms of ADHD, research studies persist in studying ADHD as though it is a unitary construct. Advances in our understanding of the disorder and strategies for the appropriate targeting of medications to individuals with specific phenotypes must tackle the issue of clinical and underlying neurobiological heterogeneity. Some potential research methods to gain traction on this issue will be discussed.

QEEG and ERPs in ADHD Assessment, Diagnosis and Treatment

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Modern neuroscience demonstrates that there are many reasons why people experience behavioral symptoms of Attention-deficit/Hyperactivity Disorder (ADHD), including existence of a focus near the Rolandic fissure, maturation lag, disruption of the basal ganglia thalamo-cortical circuits, etc. Resting-state spontaneous EEG and event-related potentials (ERPs) in different behavioral paradigms are among functional neuromarkers in neuroscience. It has been consistently shown that the ERP waves such as CNV and P300 fit the criteria for biomarkers: 1) have high test-retest reliability; 2) consistently reflect experimental manipulations in sensory and cognitive domains; and 3) discriminate ADHD from healthy population with quite large effect sizes. It's also a common view that ERP waves are the sum of activities from widely distributed cortical areas and must be decomposed into separate latent components with distinct localizations and different functional meanings.

This lecture presents 10 years of the author's experience of applying ERPs in clinical practice of ADHD. The experience includes studies on: 1) test-retest reliability of ERP latent components; 2) ERP neuromarkers of ADHD; 3) ERP indexes of neuropsychological domains such as energization, monitoring, task switching, etc.; 4) predicting effects and side-effects of Ritalin in ADHD population; 5) creating neurofeedback protocols for ADHD on the basis of ERP assessment; 6) creating tDCS protocols for ADHD on the basis of ERP assessment; 7) monitoring the effects of treatments by ERPs.

EEG Anomalies in ADHD: Linking EEG Activity with Mechanisms and Behavior

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Attention-deficit/Hyperactivity Disorder (ADHD) is one of the most common psychiatric disorders of childhood, affecting approximately 5% of primary school children. Almost all models of the disorder accept that the behavioral cluster which is ADHD

results from an underlying central nervous system (CNS) dysfunction. However, the exact nature of this dysfunction is poorly understood. Several electrophysiological-based models of ADHD have been proposed and recent research has suggested that most are too simplistic in nature, and the underlying CNS dysfunctions are inaccurately labelled. Part of the problem results from the use of multiple bands in the analysis of the EEG, as this approach does not allow an understanding of the role of any discrete band on functioning. In a different approach, our group has been decomposing the EEG into single bands and relating anomalies in these bands to specific brain states (such as arousal) and to behavior. Results from a number of studies, and their implications for understanding the link between brain and behavior, will be discussed.

Neurofeedback, ADHD and Sleep (Part I) The NIMH-funded ADHD Research: 'The Definitive Trial' into the Efficacy of Neurofeedback in ADHD? (Part II)

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Recent insights suggest an etiological contribution of sleep disorders in sub-groups of ADHD patients, specifically sleep-onset insomnia (Arns & Kenemans, 2012). Chronobiological treatments, such as melatonin and morning bright light, have demonstrated clinical effects in ADHD and we recently demonstrated an association between the worldwide prevalence of ADHD and solar intensity (Arns, van der Heijden, Arnold, & Kenemans, 2013), as a further indication for the role of circadian dysregulation and sleep in the etiology of ADHD. In relation to neurofeedback, it has been demonstrated that Sensori-Motor Rhythm (SMR) neurofeedback impacts on the sleep spindle circuitry (SSC) resulting in increased sleep spindle density (see Arns & Kenemans, 2012, for review). Overlap between the reticulo-thalamo-cortical SSC and the circadian network has been reported, suggesting overlap between neurofeedback and chronobiological treatments. The treatment effects on ADHD symptoms such as inattention, hyperactivity, and impulsivity thus arise as a result of normalized sleep, as will be demonstrated based on a recent study, where only for SMR neurofeedback it was found that improvement in sleep-onset latency mediated the improvements on inattention. For Theta/Beta

neurofeedback this was not found, suggesting specificity of SMR and Theta/Beta neurofeedback protocols in the treatment of ADHD.

In the second part of this presentation the current evidence level for various neurofeedback protocols in the treatment of ADHD and different efficacy designs will be reviewed (Arns, Heinrich, & Strehl, 2014), as well as the need, rationale, and strategy of the NIMH funded double-blind placebo controlled iCAN study (international Collaborative ADHD Neurofeedback study) that is currently recruiting 140 children with ADHD at Ohio State University and the University of North Carolina (The Collaborative Neurofeedback Group, 2013).

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