SOCIETY OF APPLIED NEUROSCIENCE (SAN) & NATIONAL INITIATIVE BRAIN & COGNITION (NIHC) MEETING 2014

UTRECHT, THE NETHERLANDS

CONFERENCE PROGRAM

AND

ABSTRACTS

SAN/NIHC 2014 MEETING

Utrecht 2014

Reliable & Rapid relief of treatment resistant depression by facilitated ketamine infusion.

Steven Richard Devore Best[1], Brian Griffin[2] and Ashley Kendall[2]

[1]*The Neuroscience Center, LLC*
[2]*Northwestern University*

Thalamocortical dysrhythmia (TCD) refers to abnormal oscillatory activity in a "circuit" between thalamus & cortex. The anterior cingulate gyrus is a region associated with neural representations of physical and social pain. Mental disorders associated with physical and social pain (e.g., depression, obsessive compulsive disorder, chronic "somatic" pain) may be characterized as TCD-related disorders. Research suggests that external neuromodulation consisting of transcranial low voltage electrical stimulation (tES) and repetitive transcranial magnetic stimulation (rTMS) can be used to ameliorate depression, a TCD-related disorder. More recently, considerable attention has been focused on the dissociative anesthetic drug ketamine, which is thought to alleviate symptoms of depression by inhibiting action of N-methyl-D-aspartate receptors. Ketamine provides temporary relief within several hours, and is a particularly effective intervention for acute suicidality. In contrast, external neuromodulation is associated with lasting decreases in depression symptomatology that take effect more slowly. We hypothesized that external neuromodulation would temporarily improve the anterior hypersynchronization so that more people would respond to infused ketamine. Accordingly, in the present study, 28 patients (13 men, 15 women; age range 18-67 years) suffering from TCD-related disorders were treated with combined ketamine/external neuromodulation for at least one month. Patients were assessed at pretreatment and at follow-up using Clinical Global Impression Improvement (CGI-I) ratings. Most patients showed significant improvement in functioning over the course of treatment, and the benefit lasted longer than usually seen with either method alone. This suggests that combined ketamine/external neuromodulation treatment may be a more effective treatment for TCD-related disorders than either treatment in isolation.

Zinc and depression. Modulation of serotonergic neurotransmission by zinc at a cellular level.

Zdzislaw Chilmonczyk[1], Jaroslaw Walory[1], Andrzej Bojarski[2], Ingebrigt Sylte[3], Gabriel Nowak[4] and Bernadeta Szewczyk[2]

[1]*National Medicines Institute*
[2]*Institute of Pharmacology PAS*
[3]*University of Tromso*
[4]*Department of Pharmacology, Jagiellonian University*

Growing body of evidence suggests that a deficiency (rather than an excess) of zinc leads to an increased risk for the development of neurological disorders. Zinc deficiency has been shown to affect neurogenesis and increase neuronal apoptosis, which can lead to learning and memory deficits. Altered zinc homeostasis is also suggested as a risk factor for depression, Alzheimer's disease (AD), aging, and other neurodegenerative disorders [1]. Recent preclinical data indicated the antidepressant-like activity of zinc in different tests and models of depression.There also are data indicating that the antidepressant-like activity of zinc observed in the forced swim test involves interaction with the serotonergic system [2].
On the receptor level zinc has been found to diminish agonists and antagonists binding to serotonin 1A receptors behaving as a negative allosteric modulator [3]. We found that zinc abolished the 5-HT1A receptor activation by its agonist 8-OHDPAT reversing (at micromolar level) the adenylate cyclase inhibition in CHO and HEK 293 cells with overexpressed 5HTRA1 gene. Combined, these data suggest that zinc might contribute to a subtype-selective pharmacological fine-tuning of serotonergic neurotransmission.

Aknowledgements: The work was partially supported by the Polish Norwegian Research Program grant Pol-Nor/198887/73/2013.

References:

1. Szewczyk B. Neurosci. 2013, 5, 33.
2. Szewczyk B, Poleszak E, et al. Prog. Neuropsychopharmacol. Biol. Psychiatry. 2009, 33, 323-329.
3. Barrondo S, Sallés J. Neuropharmacol. 2009, 56, 455-462.

The effectiveness of neurofeedback in adolescent anorexia nervosa

Nina Lackner[1,2], Human-Friedrich Unterrainer[1], Marguerite Dunitz-Scheer[3] , Peter Scheer[3], Sandra Wallner-Liebmann [4], and Christa Neuper[1]

[1] *Institute of Psychology, Karl-Franzens University Graz, Austria*
[2] *University Clinic of Psychiatry, Medical University Graz, Austria*
[3] *University Children's Hospital, Division of General Pediatrics, Medical University Graz, Austria*
[4] *Institute for Pathophysiology and Immunology, Medical University Graz, Austria*

Background: Strong research evidence exists for electrocortical dysfunctions in anorexia nervosa (AN). Deviations were found mainly within the alpha and the beta band indicating a "hyperarousal" in patients with AN. Neurofeedback as an operant-conditioning tool for self-regulation aims to train deficient brain frequency patterns.
Objective: The aim of the present study was to evaluate the clinical efficacy of alpha neurofeedback in a sample of adolescent AN. It was predicted that neurofeedback will demonstrate clinically significant improvements on eating-disordered pathology.
Methods: The study comprises 20 adolescent females diagnosed with AN according to ICD-10. All patients were treated at the University Children´s Hospital Graz and were randomly allocated in an experimental (EG, n=10) and a control group (CG, n=10). While both groups received usual maintenance treatment, the EG received ten sessions neurofeedback to enhance individual alpha frequency (IAF). Clinical data, psychological questionnaires (Three Factor Eating Questionnaire, Eating Disorder Cognition Questionnaire, Emotional Competence Questionnaire), and resting EEG (with eyes open and eyes closed) were collected before and after neurofeedback intervention.
Results: In the EG, an increase of relative theta band power in the eyes closed condition was observed ($p<.05$). No significant EEG changes were found in the CG. In addition, ANCOVAs demonstrate significant differences at post-test between EG and CG in eating disordered psychopathology and emotional competence.
Discussion: The present study supports the effectiveness of neurofeedback as additional treatment tool in the therapy of AN. Although the results are limited, this is the first study evaluating neurofeedback in a sample of AN patients.