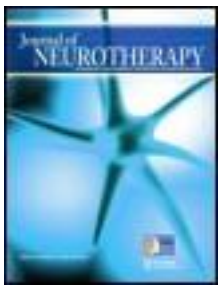

Journal of Neurotherapy: Investigations in Neuromodulation, Neurofeedback and Applied Neuroscience

News from Other Journals and Websites

David A. Kaiser PhD

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NEWS FROM OTHER JOURNALS AND WEBSITES

David A. Kaiser, PhD, Editor

Good news! Neurotherapy papers are appearing in more and more journals, increasing general awareness. Brain-computer interface papers using EEG training of various parameters are also on the rise, though only one is included below.

Authors are encouraged to submit recent preprints or reprints for this section and anyone can submit reviews or recommend websites. Contact David Kaiser at dakaiser@mail.rit.edu

RECENT MUST-READ PAPERS

Bearden, T. S., Cassisi, J. E., & Pineda, M. (2003). Neurofeedback training for a patient with thalamic and cortical infarctions. *Applied Psychophysiology & Biofeedback*, *28*, *241-253*.

Neurofeedback training to inhibit 4-8 Hz theta activity was conducted for 42 sessions from left hemisphere sites, producing significant reductions in theta amplitude at training sites and relative normalization of QEEG at the left posterior head region.

Egner, T., & Gruzelier, J. H. (2003). Ecological validity of neurofeedback: Modulation of slow wave EEG enhances musical performance. *Neuroreport*, *14*, 1221-1224.

The authors investigated pedagogic relevance of neurofeedback for enhancing normal function. Conservatoire students improved their musical performances after theta-up training.

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Fernández, T. (2003). EEG and behavioral changes following neurofeedback treatment in learning disabled children. *Clinical Electroencephalography*, *34*, 145-152.

Ten learning-disabled children (high on a theta/alpha ratio baseline) improved on the WISC and showed evidence of greater EEG maturation after training.

Fuchs, T., Birbaumer, N., & Lutzenberger, W. (2003). Neurofeedback treatment for attention-deficit/hyperactivity disorder in children: A comparison with methylphenidate. *Applied Psychophysiology & Biofeedback*, *28*, 1-12.

Twenty-two ADHD children underwent neurofeedback and 12 underwent stimulant therapy, according to parental preference. Variables of attention improved in both groups, as did behaviors related to the disorder.

Martens, W. H. J. (2003). Multisystemic therapy for antisocial juveniles: Suggestions for improvement. *Acta Psychiatrica Scandinavica*, *108*, 318.

The author recommends a combination of psychotherapeutic, neurologic and neurofeedback treatment be used for treating antisocial personality disorders in juveniles.

Vernon, D., Egner, T., Cooper, N., Compton, T., Neilands, C., Sheri, A., et al. (2003). The effect of training distinct neurofeedback protocols on aspects of cognitive performance. *International Journal of Psychophysiology*, *47*, 75-85.

In eight sessions of neurofeedback the SMR-group were able to selectively enhance their SMR activity whereas theta trainers failed to exhibit any changes in their EEG. The SMR-group also improved on cued recall performance and focused attentional processing.

Wang, X. (2003). Noninvasive laser-induced photoacoustic tomography for structural and functional in vivo imaging of the brain. *Nature Biotechnology*, *21*, 803-806.

The year 2002 saw the first report of fMRI neurofeedback; in 2003 an optical technique is added to the growing family of neurotherapeutic techniques.

ELECTROENCEPHALOGRAPHY

Bullock, T. H., McClune, M. C., & Enright, J. T. (2003). Are the electroencephalograms mainly rhythmic? Assessment of periodicity in wide-band time series. *Neuroscience*, *121*, 233-252.

Another new QEEG method to quantify periodicity–period specific average–indicates that EEGs are mostly aperiodic, generally and locally (peaks).

Herning, R. I., Better, W., Tate, K., & Cadet, J. L. (2003). EEG deficits in chronic marijuana abusers during monitored abstinence: Preliminary findings. *Annals of the New York Academy of Sciences*, *993*, 75-78.

During early abstinence, marijuana abusers show reduced theta and lower alpha rhythm activity during eyes closed compared to controls. These reductions persisted for the entire month of monitoring.

Kramarenko, A. V., & Tan, U. (2003). Effects of high-frequency electromagnetic fields on human EEG: A brain mapping study. *International Journal of Neuroscience*, *113*, 1007-1019.

Cellular phones may reversibly influence the human brain, inducing abnormal slow waves in EEG.

Laufs, H., Krakow, K., Sterzer, P., Eger, E., Beyerle, A., Salek-Haddadi, A., et al. (2003). Electroencephalographic signatures of attentional and cognitive default modes in spontaneous brain activity fluctuations at rest. *Proceedings of the National Academy of Sciences of the United States of America*, *100*, 11053-11058.

Alpha oscillations signal a neural baseline with "inattention" whereas beta rhythms index spontaneous cognitive operations during conscious rest.

Loo, S. K., Specter, E., Smolen, A., Hopfer, C., Teale, P. D., & Reite, M. L. (2003). Functional effects of the DAT1 polymorphism on EEG measures in ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry*, *42*, 986-993.

A specific genetic factor relevant to ADHD which mediates medication-related changes in cortical activity is associated with specific EEG profiles.

Mattia, D., Spanedda, F., Babiloni, F., Romigi, A., & Marciani, M. G. (2003). Quantitative EEG patterns following unilateral stroke: A study in chronic stage. *International Journal of Neuroscience*, *113*, 465-482.

Left and right hemisphere stroke patients could be differentiated in their EEG by comparing the changes from rest to challenge task conditions.

McFarland, D. J., & Wolpaw, J. R. (2003). EEG-based communication and control: Speed-accuracy relationships. *Applied Psychophysiology and Biofeedback*, *28*, 217-231.

Continual online gain adaptation could increase the speed and accuracy of EEG-based cursor control.

Seifert, J., Scheuerpflug, P., Zillessen, K. E., Fallgatter, A., & Warnke, A. (2003). Electrophysiological investigation of the effectiveness of methylphenidate in children with and without ADHD. *Journal of Neural Transmission*, *110*, 821-829.

P300a amplitudes are lower in non-medicated ADHD patients than in healthy children during a continuous performance task, but not for methylphenidate-treated hyperactive children.

Zampi, C., Fagioli, I., & Salzarulo, P. (2003). Time course of EEG background activity level before spontaneous awakening in the second semester of human life. *Neuroscience Letters*, *349*, 83-86.

Infant sleep EEGs reflect changes in activation levels prior to waking. If we could just condition them to suppress these changes, parents of newborns could get a good night's sleep.

MENTAL HEALTH

Aycicegi, A., Dinn, W. M., Harris, C. L., & Erkmen, H. (2003). Neuropsychological function in obsessive-compulsive disorder: Effects of comorbid conditions on task performance. *European Psychiatry*, *18*, 241-248.

OCD patients are impaired on delayed memory, response inhibition, impulsivity, and temporolimbic functions, but they are not impaired on executive function or verbal fluency.

Berlin, L., Bohlin, G., Nyberg, L., & Janols, L. O. (2003). Sustained performance and regulation of effort in clinical and non-clinical hyperactive children. *Child: Care, Health and Development*, *29*, 257-267.

Deficits in sustained performance problems among ADHD children appear when long interstimulus intervals are used, possibly reflecting poor regulation of effort.

Eigsti, I. M., & Shapiro, T. (2003). A systems neuroscience approach to autism: Biological, cognitive, and clinical perspectives. *Mental Retardation and Developmental Disabilities Research Reviews*, *9*, 205-215.

The authors conclude that autism is a heterogeneous disorder, likely to have multiple etiologies often associated with developmental growth and pruning of neural tissue. (Editor's note: A single mechanism could produce the multifarious effects we observe throughout the patient's lifespan as cognitive and sensorimotor development proceeds, say, without proper inhibitory action. Autism could be a relatively simple neuroendocrine disorder, one of too little estrogen and too much testosterone, perhaps.)

Eugene, F., Levesque, J., Mensour, B., Leroux, J. M., Beaudoin, G., Bourgouin, P., et al. (2003). The impact of individual differences on the neural circuitry underlying sadness. *Neuroimage*, *19*, 354-364.

Individual differences may be responsible for inconsistent research findings in affective neuroscience.

Goldstein, L. B. (2003). Neuropharmacology of TBI-induced plasticity. *Brain Injury*, *17*, 685-694.

Neuroleptics and other central dopamine receptor antagonists, benzodiazepines and some anti-convulsants may be detrimental to brain injury recovery.

Holtmann, M., Becker, K., Kentner-Figura, B., & Schmidt, M. H. (2003). Increased frequency of rolandic spikes in ADHD children. *Epilepsia*, *44*, 1241-1244.

The frequency of rolandic spikes in children with ADHD is significantly higher than expected from epidemiologic studies. How ADHD symptoms are related to this feature is unclear.

Kirschner, J., Moll, G. H., Fietzek, U. M., Heinrich, H., Mall, V., Berweck, S., et al. (2003). Methylphenidate enhances both intracortical inhibition and facilitation in healthy adults. *Pharmacopsychiatry*, *36*, 79-82.

MPH enhances both intracortical inhibition and facilitation, which suggests it acts on the motor cortex using a neurotransmitter in addition to dopamine.

Lisanby, S. H., Luber, B., Schlaepfer, T. E., & Sackeim, H. A. (2003). Safety and feasibility of magnetic seizure therapy (MST) in major depression: Randomized within-subject comparison with electroconvulsive therapy. *Neuropsychopharmacology*, *28*, 1852-1865.

Magnetic seizure therapy, a novel means of performing convulsive therapy using rapidly alternating strong magnetic fields, may be just as effective as ECT and reduce side effects.

Lisska, M. C., & Rivkees, S. A. (2003). Daily methylphenidate use slows the growth of children: A community based study. *Journal of Pediatric Endocrinology and Metabolism*, *16*, 711-718.

Height differences between treated children and sibling controls after two years of treatment across broad range of doses (10-80 mg per day) suggest grow-suppressive effects of methylphenidate are greater than suspected.

McGinty, D., & Szymusiak, R. (2003). Hypothalamic regulation of sleep and arousal. *Frontiers in Bioscience*, *8*, 1074-1083.

Mutually-inhibitory interactions between sleep-promoting and arousal-promoting systems, focused in and near the hypothalamus, are hypothesized to form a sleep-wake switch.

Michael, N., Gosling, M., Reutemann, M., Kersting, A., Heindel, W., Arolt, V., et al. (2003). Metabolic changes after repetitive transcranial magnetic stimulation (rTMS) of the left prefrontal cortex: A sham-controlled pro-

ton magnetic resonance spectroscopy (1H MRS) study of healthy brain. *European Journal of Neuroscience*, *17*, 2462-2468.

Rapid transcranial magnetic stimulation may act via stimulation of glutamatergic prefrontal neurons.

Miles, J. H., Takahashi, T. N., Haber, A., & Hadden, L. (2003). Autism families with a high incidence of alcoholism. *Journal of Autism and Developmental Disorders*, *33*, 403-415.

An association between maternal alcoholism and regressive onset autism (language loss, primarily) is reported.

Palomo, T., Archer, T., Beninger, R. J., & Kostrzewa, R. M. (2003). Neurodevelopmental liabilities of substance abuse. *Neurotoxicity Research*, *4*, 267-279.

Even elevated levels of estrogens and corticosteroids in the pregnant mother can act as neuroteratogens.

Woody, E. Z., & Szechtman, H. (2003). How can brain activity and hypnosis inform each other? *International Journal of Clinical and Experimental Hypnosis*, *51*, 232-255.

Authors argue for using hypnosis in the study of brain mechanisms of conscious experience.

NEUROIMAGING

Castellanos, F. X., Sharp, W. S., Gottesman, R. F., Greenstein, D. K., Giedd, J. N., & Rapoport, J. L. (2003). Anatomic brain abnormalities in monozygotic twins discordant for attention deficit hyperactivity disorder. *American Journal of Psychiatry*, *160*, 1693-1696.

MZ twins with ADHD have smaller caudate volumes than their unaffected co-twins, which provides further support for striatal models of ADHD pathophysiology.

Charney, D. S. (2003). Neuroanatomical circuits modulating fear and anxiety behaviors. *Acta Psychiatrica Scandinavica. Supplementum*, *417*, 38-50.

Anxiety disorders are heterogeneous, both symptomatically and neuroanatomically. The current descriptive nosologies are outdated and require genetic, neuroimaging and neurochemical approaches to refine anxiety disorder phenotypes and facilitate selection and development of anti-anxiety therapies.

Gross-Isseroff, R., Hermesh, H., Zohar, J., & Weizman, A. (2003). Neuroimaging communality between schizophrenia and obsessive compul-

sive disorder: A putative basis for schizo-obsessive disorder? *World Journal of Biological Psychiatry*, *4*, 129-134.

The caudate nucleus, orbitofrontal cortex, anterior cingulate gyrus and mediodorsal thalamic nucleus may be implicated in both schizophrenia and OCD, though the jury is still out on this.

Lacerda, A. L., Dalgalarrondo, P., Caetano, D., Camargo, E. E., Etchebehere, E. C., & Soares, J. C. (2003). Elevated thalamic and prefrontal regional cerebral blood flow in obsessive-compulsive disorder: A SPECT study. *Psychiatry Research*, *123*, 125-134.

OCD symptom severity correlated positively with inferior frontal lobe and right basal ganglia activity. Compulsive behavior was inversely correlated with right thalamus activity.

Palomo, T., Beninger, R. J., Kostrzewa, R. M., & Archer, T. (2003). Brain sites of movement disorder: Genetic and environmental agents in neurodevelopmental perturbations. *Neurotoxicity Research*, *5*, 1-26.

Neuroanatomy and neurochemistry of ADHD are reviewed with reference to metallic and organic environment and genes. The role of apoptosis and cellular plasticity are highlighted.