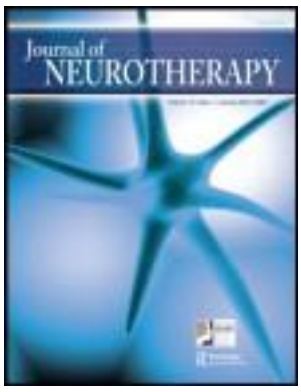


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## As Connectivity Training Comes of Age


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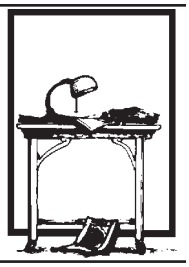
*EDITORIAL*



# As Connectivity Training Comes of Age

The history of biofeedback began with ear wiggling (Bair, 1901), but the history of SMR biofeedback emerged serendipitously from toxin-induced seizures in the context of investigating electroencephalographic correlates of behavioral inhibition (Sterman, Fairchild, & McRae, 1972; Sterman & Kovalesky, 1979; Clemente et al., 1963; Roth et al., 1967; Sterman et al., 1969). Preceded by alpha biofeedback (Kamiya, 1968), SMR biofeedback provided the best evidence that EEG training could alter neurophysiological function in a permanent fashion. SMR training acted as an anticonvulsant, reducing seizure frequency and motoric excitability as well as impacting sleep. In replicating and extending Sterman's epilepsy research, Lubar and Bahler (1976) noticed that after SMR enhancement and theta suppression training, a hyperactive epileptic showed, in addition to seizure reduction, a general decrease in his over-activity. This led to case studies in which hyperactivity was directly targeted by neurofeedback (e.g., Lubar & Shouse, 1976). More than 200 individuals has since been involved in controlled research for attention deficit hyperactivity disorder (cf. Monastra et al., 2005) and nearly 2,000 individuals in case studies and clinical trials (e.g., Alhambra et al., 1995; Lubar et al., 1995; Rossiter & LaVaque, 1995; Linden et al., 1996, Rossiter, 1998; Boyd et al. 1998; Thompson & Thompson, 1998; Kaiser & Othmer, 2000; Carmody et al., 2001; Monastra et al., 2002; Fuchs et al., 2003; Rossiter, 2004; Heinrich et al., 2004; Strehl et al., 2006). These studies found that neurofeedback training is often as effective or more effective than stimulant medication for ADHD and without notable side effects. Stimulant therapy, on the other hand, may stunt a child's growth, reducing the height growth rate by as much 20% (Charach et al., 2006; Swanson et al., 2006), and relieves the child of responsibility for governing his or her own behavior.

Identifying neurophysiological changes associated with EEG training will emerge as more clinicians gain access to neuroimaging technology. Beauregard and Levesque (2006; Levesque et al., 2006) reported brainstem and limbic activation in response to neurofeedback training compared to untreated ADHD children using fMRI technology. Other researchers have found rhythm-associated seizure resistance in rats, a creature not well known for placebo ef-

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fects (Miller et al., 1994). The anticonvulsant properties of SMR training suggests that this training increases the number, density, or efficiency of GABAergic receptors in the somatosensory pathway via long-term potentiation and synaptogenesis. A near-term goal of our field should be to identify associated neuroanatomical changes at the cellular level. Does SMR training leads to increased GABAergic receptor density or not? A couple studies and a few looks under the microscrope may resolve the issue once and for all.

In the mean time the field is transforming into more than simply motor-pathway inhibition training. Connectivity training is coming of age and in this issue of the Journal of Neurotherapy we see some of the initial forays into the effect of connectivity training on autism and other disorders.

*David A. Kaiser, PhD*
*Editor*

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