

## Neurofeedback COPD Abstract

Submitted by Ed O'Malley PhD, FAASM

Abstract: EEG Biofeedback In EEG biofeedback (neurofeedback), a real-time display of the brain's electrical activity, fed back as visual or auditory information, enables the user to modify that brainwave activity. In a 2010 keynote address to the International Society for Neurofeedback and Research, Doidge<sup>23</sup> suggested that the misperception that the adult brain was fixed and unchangeable "led scientists to doubt the claims made by the pioneers of neurofeedback."<sup>23</sup> Only with the discovery of neuroplasticity did the work of neurofeedback investigators and clinicians begin to find acceptance among other researchers. Still, for some time, there existed limited evidence as to whether EEG biofeedback directly affects neuroplasticity, resulting in ongoing skepticism about its potential as a restorative therapeutic modality. However, several recent studies utilizing transcranial magnetic stimulation and functional magnetic resonance imaging (fMRI) have demonstrated objective, temporally direct changes in cortical activation and connectivity as a result of neurofeedback.<sup>24,25</sup>

In 2000, *Clinical EEG and Neuroscience* published a special issue on the topic of neurofeedback. The editor introduced the topic by writing,

" The literature, which lacks any negative study of substance, suggests that EBT (EEG biofeedback therapy) should play a major therapeutic role in many difficult areas. In my opinion, if any medication had demonstrated such a wide spectrum of efficacy, it would be universally accepted and widely used." <sup>26</sup>(pv)

Neurofeedback has since been shown to be an effective intervention for traumatic brain injury,<sup>27</sup> and there have also been clinical reports (though no formal studies) of neurofeedback providing symptom relief for patients with multiple sclerosis (S. Othmer, PhD, e-mail communication, November 12, 2012). These findings provide a rationale for studying neurofeedback as an intervention for PCCI because traumatic brain injury and multiple sclerosis share features with PCCI: that is, demyelination and an expanded extent of brain activation for short-term memory tasks, suggesting "compensatory recruitment of additional brain regions in order to perform the task successfully" (p. 27).<sup>28</sup>

Similar to trends in cognitive neuroscience, current neurofeedback strategies reflect 2 different but complementary directions: one driven by a focus on localization and the other by a focus on global brain function. The more common approach, with its roots in the localization school of neuroscience, could be characterized as a "diagnosis and treatment" approach, in which abnormalities in brainwave frequencies at particular locations are identified, ordinarily by means of a quantitative EEG. Researchers and clinicians have identified EEG patterns commonly associated with particular symptoms, and the neurofeedback equipment can be programmed to reward the brain for shifting its activity away from the symptom-associated patterns. For example, attention-deficit hyperactivity disorder (ADHD) in children frequently is associated with slow (theta) wave to fast (beta) wave ratios greater than 3:1 along the cingulate gyrus, located on the innermost surface of each hemisphere above the corpus callosum. A child with ADHD evidencing this pattern would be trained over a series of sessions to lower his/her theta wave amplitude.<sup>29</sup>

The present study used a newer approach to neurofeedback, rooted in the global view of brain function. The *NeuroOptimal* system, developed by the Zengar Institute ([www.Zengar.com](http://www.Zengar.com)) is designed to train the brain as a whole, without reference to particular locations or frequencies. Unlike classical neurofeedback approaches, in which the participant engages actively and/or consciously with the software and is rewarded for producing prescribed EEG patterns, the participant in the Zengar approach simply "lets go" and allows the brain to use the feedback—provided as brief interruptions to the music he or she is hearing—to enable its own innate capacity for self-organization.

The feedback delivered by the Zengar system is systemic—based on the whole brain’s dynamic activity over time, not its achievement of prescribed states in prescribed locations. The fundamental assumption is that lowering the amplitude of any specific frequency (eg, 8-12 Hz in the left prefrontal cortex) will, by necessity, affect other frequencies in other parts of the brain in the same way that strengthening a single muscle group will affect alignment in other parts of the body, and so it is more realistic to train the brain as a whole system rather than focus on a single location or set of frequencies. This approach recognizes that the brain has a natural tendency toward self-regulation and resilience, allowing flexible cognitive and behavioral responses to a challenging and changing environment.

We are aware that oncology clinicians will be curious about the biological mechanism by which this form of neurofeedback might ameliorate PCCI. This is particularly true, given that the studies verifying PCCI and attempting to identify its causes have used a different paradigm than the one that underlies this form of neurofeedback. However, the existing evidence that PCCI exhibits variable neuroimaging findings and affects multiple neuropsychological domains (particularly in complex frontal-subcortical networks) does suggest that PCCI is not a clearly localizable phenomenon, which points to the importance of developing management strategies that respect the brain as a complex and highly integrated system. Our suspicion is that the concepts of nonlinear dynamical systems theory (eg, self-organization of complex systems, sensitive dependence on initial conditions, basins of attraction, and the importance of feedback), best understood by theorists and researchers in the fields of complexity science and systems theory,<sup>30</sup> ultimately may be more productive in explaining both the changes in cognition seen during and after cancer treatment and also the mechanism underlying this form of neurofeedback.

Some work is already being done to bridge the fields of complexity science and neuroscience. In *Modeling Phase Transitions in the Brain*, Freeman asserts that “abrupt global reorganizations by phase transition in larger brain systems implement a wide variety of intellectual and intentional brain functions . . . including the switch from prodrome to epilepsy and from from sleep to wake or REM. . . . In each aggregate [of neurons] there are certain conditions that specify a critical point in the phase space at which the system is particularly susceptible to transit from one phase to another, as when the neurons in the sensory cortex transit from a disorganized state of expectation to an organized state of categorization, from noise to signal” (p. v, p. vii).<sup>31</sup> Freeman acknowledges that this view of brain function is as yet unproven, but it is currently a focus of study among computational neuroscientists,<sup>32</sup> and Freeman advocates the development of a detailed theory of nonlinear neurodynamics.

The Zengar system is rooted in this view of brain organization. Its software detects phase state changes, the precursors to phase transitions. Alerted by feedback that a phase transition is imminent, the brain is able either to reorganize to return to its prior phase (as when the mind refocuses on a task after wandering) or to transit to a new phase (as in the movement from wakefulness to sleep). Neither phase is preferred, or sought, or avoided by the software. Instead, feedback simply is given when the phase transition is about to occur. Because there is no diagnosis required for this form of neurofeedback, and no specific protocol is developed on the basis of that diagnosis, this approach is considered to be training the brain in flexibility and resilience rather than treating particular symptoms. As a result, the Zengar system is not a controlled medical device and, therefore, the developers have not sought FDA approval.

23. Doidge N. The brain that changes itself: the neuroplasticity

revolution and film clips of people undergoing plastic

change. Keynote address at: 18th Annual Conference of the

International Society for Neurofeedback and Research; September

27-October 3, 2010; Denver, CO.

24. Ros T, Munneke MA, Ruge D, Gruzellier JH, Rothwell

JC. Endogenous control of waking brain rhythms induces

neuroplasticity in humans. *Eur J Neurosci.* 2010;31:

770-778.

25. Ros T, Theberge J, Frewen PA, et al. Mind over chatter: plastic

upregulation of the fMRI salience network directly after

EEG neurofeedback. *Neuroimage.* 2013;65:324-335.

26. Duffy FH. The state of EEG biofeedback therapy (EEG operant

conditioning) in 2000: an editor's opinion. *Clin EEG*

*Neurosci.* 2000;31:v.

27. EEGInfo. Neurofeedback research: head injury. [http://](http://www.eeginfo.com/research/headinjury_main.html)

[www.eeginfo.com/research/headinjury\\_main.html](http://www.eeginfo.com/research/headinjury_main.html). Accessed

December 18, 2012.

28. McDonald BC, Saykin AJ, Ahles TA. Brain imaging investigation

of chemotherapy-induced neurocognitive changes. In:  
Meyers CA, Perry JR, eds. Cognition and Cancer. New York,  
NY: Cambridge University Press; 2008:19-32.

29. Demos JN. Getting Started With Neurofeedback. New York,  
NY: Norton; 2005.

30. Kelso JAS. Dynamic Patterns: The Self-organization of  
Brain and Behavior (Complex Adaptive Systems). Boston,  
MA: Massachusetts Institute of Technology Press; 1995.

31. Freeman WJ. Foreword. In: Steyn-Ross DA, Steyn-Ross M,  
eds. Modeling Phase Transitions in the Brain. New York,  
NY: Springer; 2010.