Biofeedback
Volume 36, Issue 4, pp. 142–147

SPECIAL ISSUE

A Three-Stage Neuropsychological Model of Neurofeedback: Historical Perspectives

Jack Johnstone, PhD

Q-Metrx, Inc., Burbank, CA, and Department of Psychology, University of California, Los Angeles

Keywords: neurofeedback, neuromodulation, brain electrical activity, arousal, connectivity

Neuromodulation by means of electroencephalography biofeedback is increasingly used clinically to promote self-regulation. This general method has been applied in a variety of neurobehavioral disorders, some of which do not have any other type of reliable medical treatment, such as autism, dyslexia, and mild head trauma. The concept of arousal is central to neurofeedback, and the relation between behavioral and neurophysiological arousal is considered. The arousal approach is differentiated from similar concepts such as regional brain activation. Recent studies emphasizing connectivity training also are discussed. These three approaches have evolved in the course of clinical practice and now provide a unique set of noninvasive clinical tools.

Introduction

Electroencephalography (EEG) biofeedback (or neurofeedback) has been developed and used as a tool to address a number of neurobehavioral disorders, some of which do not have any other type of reliable medical treatment, such as autism, dyslexia, and mild head trauma. Indeed, neurofeedback is increasingly used in peak performance training, to optimize cognitive and affective status in the absence of clinical diagnosis. In this article, we review three models guiding procedures used in the practice of neurofeedback and point out the parallel between the hierarchy of these models, the developing sophistication of neurofeedback technology, and the need to address a wide variety of clinical symptoms and presentations. We have previously proposed a set of EEG phenotypes (Johnstone, Gunkelman, & Lunt, 2005) and suggest here a staged neurofeedback method for modulating these types of patterns, focusing on issues in cortical arousal, regional activation, and connectivity.

The Arousal and General Regulatory Model

A model suggesting mechanisms for the action of neurofeedback initially emphasized a construct called "arousal" (Sterman, 1982). In studies of attentional disorder, Othmer, Othmer, and Kaiser (1999) demonstrated efficacy of neurofeedback training in 342 children, primarily using electrodes placed at C3 and C4, that is, over the left and right central regions (e.g., the motor strip). Improvement in the Test of Variables of Attention was highly significant following approximately 20 training sessions on the central motor strip, as shown in Figure 1. Note that data were sorted by initial score and documented improvement in the large majority of cases, not explained by practice effect.

Arousal implies a certain primacy in neuropsychological function, and an assessment of arousal is often extremely useful in characterizing clinical case presentations. The arousal model is typically taught as the primary concern in client assessment, and protocols for neurofeedback often have been employed exclusively based on this model. It is clear that this approach has both clinical utility and clear limitations. An important concern is using the construct arousal as though it were a well-defined, unified concept. Our thesis is that arousal must be differentiated from related concepts, such as regional activation, and must be understood as a complex multidimensional construct to be used more effectively in neurofeedback.

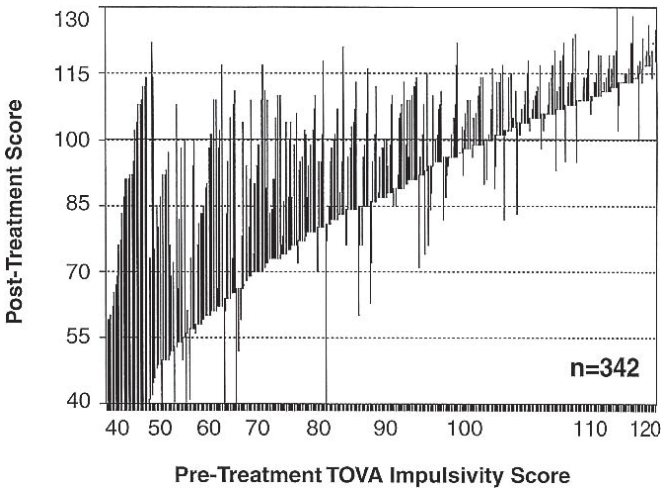

Figure 1. Effect of electroencephalography biofeedback on Test of Variables of Attention scores (from Othmer et al., 1999, p. 288).

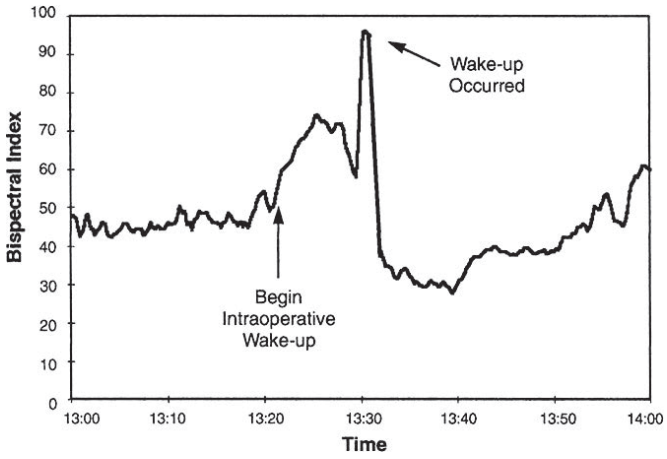

Figure 2. Bispectral index and cortical arousal (courtesy of Aspect Medical Systems, Inc.).

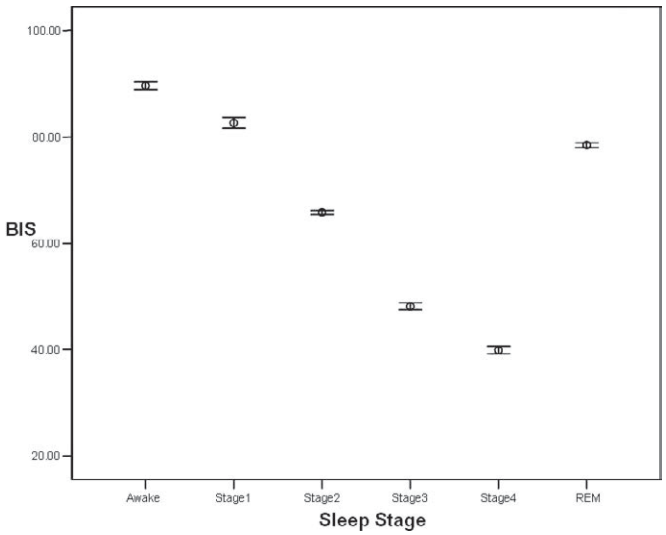

Figure 3. Sleep stage and BIS index (from Johnstone et al., 2008).

In neurophysiological terms, arousal may be considered as a point on the sleep-wake cycle. In humans, the cortex is aroused globally by the reticular activating system. In contrast, other species such as the dolphin have separate sleep-wake cycles for the two hemispheres. This differentiates the notion of arousal from that of localized activation of a region of the cerebral cortex. A good example of using information about EEG in assessment of arousal is available in anesthesia monitoring using bispectral analysis such as the BIS™ index developed and commercialized by Aspect Medical Systems (see Johnstone, 2002). The BIS™ index is used to track depth of anesthesia and calculates a scale with a range from 0 to 100, where higher values represent increasing wakefulness. This method is routinely used in surgical applications to adjust anesthesia so a patient does not receive too little or too much anesthetic agent. Figure 2 shows the monitor used during a wake-up test during anesthesia.

Recent studies have shown that the BIS™ index, developed to track depth of anesthesia, is also effective in assessing the depth of natural sleep. Johnstone, Hongmei, Smith, and Greenwald (2008) used the BIS™ index concurrently with standard polysomnographic analyses and showed excellent correspondence between values obtained automatically and continuously from the BIS monitor as compared with the gold standard manually scored sleep record (see Figure 3). Note that REM sleep shows a more activated pattern. When electromyography activity is also considered, REM can easily be separated from Stage I sleep.

It is also the case that EEG changes related to arousal (e.g., falling asleep) are most evident on the motor strip. The primary EEG characteristic of Stage II sleep is the appearance of vertex waves, so named because they are seen maximally over the central strip in the midline (e.g., the vertex). Figure 4 shows a full-head EEG with rapid progression from wakefulness (e.g., well-developed posterior alpha) to Stage II sleep with prominent vertex waves. This rapid transition to Stage II occurred repeatedly in this case and was considered clinically abnormal. Interestingly, attentional difficulty was a significant presenting complaint.

The presence of vertex waves is used in scoring of sleep stages to define Stage II. Note the repetitive high-amplitude activity in the frontocentral midline. These figures document the presence of sleep activities maximally in regions also used for neurofeedback training related to arousal.

Furthermore, the concept of arousal is multidimensional. Neurophysiological arousal is not identical to behavioral and mental arousal. Autonomic arousal is not identical to cortical arousal (Togo, Cherniack, & Natelson, 2007). Figure 5 shows a scheme for considering arousal in two dimensions. The x-axis shows dominant EEG activity, ranging from diffusely slow to fast (desynchronized). The y-axis shows behavioral arousal ranging from hypoaroused (unresponsive) to hyperaroused (panic/anxious). One of the important features of this model is the notion of mismatches (e.g., high behavioral arousal with slow EEG, such as is frequently seen clinically with autistic spectrum disorders).

An individual may show hyperactivity behaviorally, such as repetitive stimming behaviors, but may show an EEG pattern dominated by delta and theta frequency activity. It is well known that physical hyperactivity is frequently associated with excessive theta activity in the frontal cortex and is often successfully treated with psychostimulant medication. The individual x, y coordinates are helpful in developing neurofeedback interventions where it may not be appropriate to calm the nervous system down but rather to "calm it up."

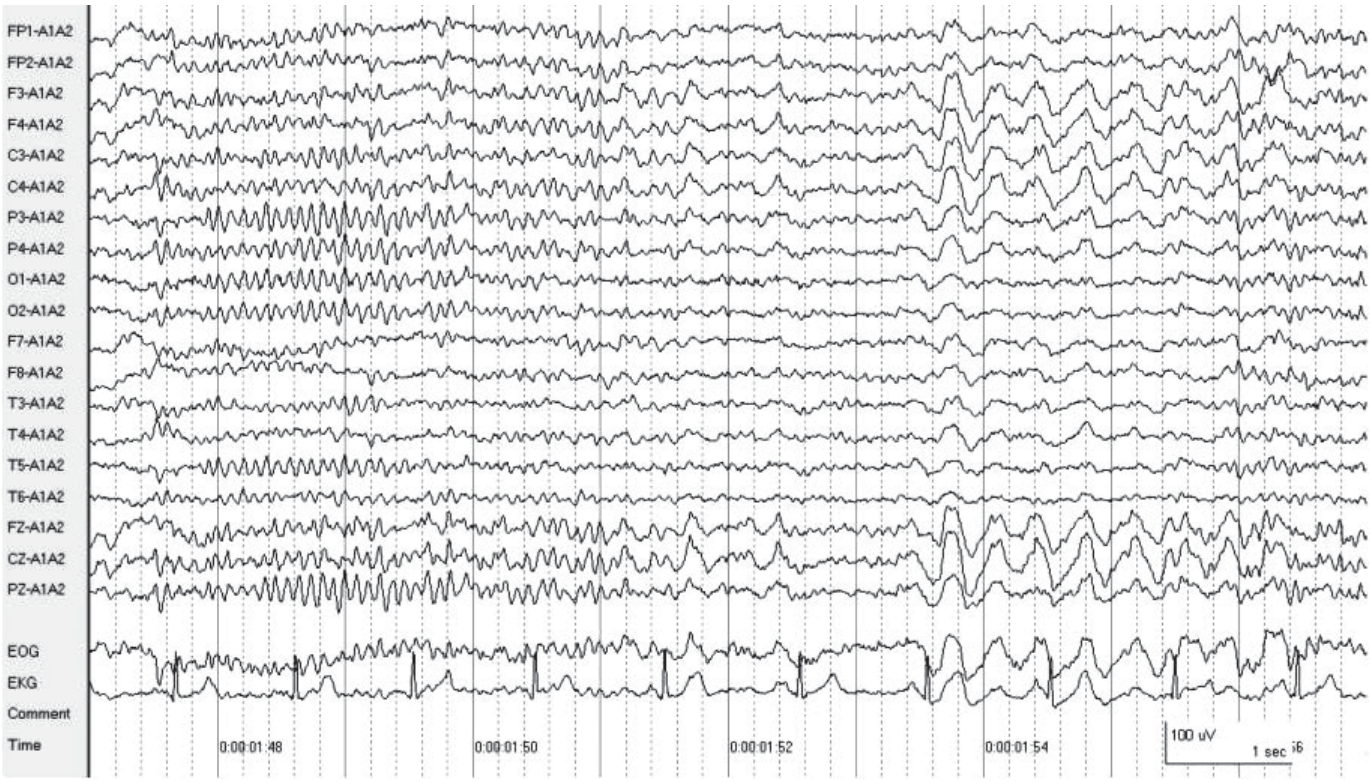

Figure 4. Rapid descent into Stage II sleep (courtesy of Q-Metrx, Inc.).

Other techniques to affect general regulatory processes as well as localized function are also now being explored clinically, including ultra-low-frequency neurofeedback training (Othmer, 2006) and slow cortical potential training (Strehl et al., 2006), as well as low-energy neurofeedback systems (see Hammond, 2007).

Regional Training Guided by Quantitative EEG

When quantitative EEG (QEEG) is available as part of client assessment, it is possible to measure neurophysiological activity directly. There are patterns that can be identified that implicate dysfunctional localized regional processing, and these patterns can be used to guide neurofeedback intervention. This approach is not a replacement for intervention based on arousal and general regulatory issues but rather an extension. Protocols targeting arousal issues are often followed by, or are trained in association with, protocols targeting regional activation as identified and validated in EEG recordings and QEEG analyses.

As an example, protocols have been developed targeting the left frontal regions in depression based on QEEG findings. Davidson has provided convergent evidence that left frontal hypoactivation is a primary feature in major depression (see Henriques & Davidson, 1991; for review, see Hugdahl & Davidson, 2002). This type of information is frequently used in a neurofeedback protocol targeting the left frontal region and frontal asymmetries (see Baehr, Rosenfeld, Baehr, & Earnest, 1999). Figure 6 shows a comparison of a 55-year-old woman with major depression to the Neurometric Database (John et al., 1987). Note the left greater than right asymmetries, primarily in the theta and alpha frequency range.

Recent results using neuromodulation techniques such as repetitive transcranial magnetic stimulation also appear to help relieve depression by stimulation of localized regions in the left frontal cortex (O'Reardon et al., 2007). These authors tested whether transcranial stimulation over the left dorsolateral cortex was safe and effective in acute treatment of major depression. This technique produced improvements in depression with minimal side effects. In our practice, we frequently recommend neurofeedback training to increase activation in left frontal regions when such asymmetries are apparent.

Difficulty with language processing is frequently due to localized dysfunction. Problems with verbal fluency and memory are typical with left frontotemporal lobe damage. In such cases, QEEG results can be useful in specifying location and extent of damage and in characterizing the EEG frequency composition of the damaged region that requires modulation.

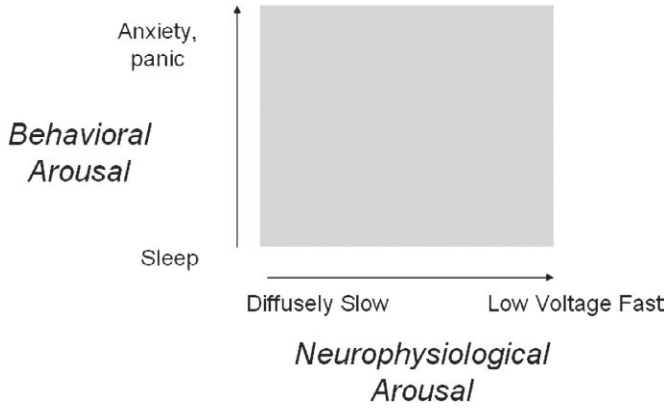

Figure 5. Behavioral arousal and neurophysiological arousal, the x-y axis.

Disorders of sensory integration and perceptual continuity are often associated with localized slowing and asymmetries over the parietal cortex. Difficulty with sensory integration can be considered a disorder on its own but is also frequently a component of other disorders, including autism, learning disability, and attention-deficit disorder. In these disorders, there is often no problem with sensory acuity but rather difficulty associating environmental stimuli and integrating them into the perceptual stream. Parietal regions can be targeted for neurofeedback training based on specific QEEG findings and client symptoms.

Training Relationships Among Regions: Connectivity

One of the fundamental principles in neuroscience is that brain regions are connected neuroanatomically by bundles of nerve fibers, typified by the commissures that connect the cerebral hemisphere and the arcuate fasiculus connecting posterior and anterior cortical regions. We can measure the intensity of activity in a given region with EEG and QEEG. We can also measure the functional connectivity by means of the correlation or coherence of activity between and among regions using QEEG methods. Neurofeedback can be used to increase or decrease functional connectivity among brain regions, using a variety of metrics including synchrony, comodulation, phase, instantaneous coherence, and difference training. These measures are all used to promote similarity of activity in one or more regions or to promote differences among regions. Early use of such an approach to promote synchrony is found in the work of Fehmi (1978), who has also pursed this model as a relaxation and stress reduction technique (Fehmi & Robbins, 2007).

A recent study using QEEG to guide neurofeedback protocol development has been reported by Coben and Padolsky (2007). These authors studied 37 children diagnosed with autistic spectrum disorder and a wait-list control group. They used QEEG methods to detect regions with excessive coherence and then applied neurofeedback training to reduce similarity between specific regions in certain EEG frequencies. After 20 training sessions, improvements were found in parent rating scales, neuropsychological test scores, and QEEG connectivity measures. Figure 7 shows the results of the multivariate connectivity analysis method used by Coben and Padolsky (2007). In this case, there is hypercoherence in the theta and alpha frequencies in the left anterior regions and hypocoherence involving the right posterior cortex.

The use of coherence in modulating specific localized regions is also of interest. It is often the case that neurofeedback training can be used to directly modulate specific cortical regions as described above. Decreased coherence of a given region with multiple adjacent regions implies dysfunction of the given region, and it can be targeted for amplitude training directly. There are other cases in which modulation of a region may be best affected by input from neighboring regions, a sort of reafferentation process via coherence training.

Clinical Use of the Progressive Three-Stage Model

Neurofeedback practitioners now have tools to address arousal issues, regional dysfunction, and interaction among regions. These tools are often used in sequence (e.g., stabilize arousal, remediate specific regional dysfunction, and increase or decrease interaction among regions). In the example of the autistic child who presents initially with difficulty sitting for a neurofeedback session, initial protocols may help to modulate arousal (e.g., SMR on

Interhemispheric Asymmetry Z-Scores: Sequential (Bipolar) and Multivariate

	Fp1-F7: Fp2-F8	F3-F7: F4-F8	Fp1-F3: Fp2-F4	F3-Fz: F4-Fz	C3-Cz: C4-Cz	T3-T5: T4-T6	P3-O1: P4-O2	F7-T3: F8-T4	Total	Posterior	Anterior
Delta	1.05	1.60	-1.58	-1.60	-0.46	-1.52	-1.22	-0.50	0.49	0.40	0.69
Theta	3.03	2.49	-0.05	-0.52	1.01	-1.00	-1.53	0.92	0.68	0.87	0.40
Alpha	2.69	2.43	-0.14	0.17	1.07	0.70	-2.48	0.86	1.25	1.40	0.11
Beta	0.26	2.24	1.03	1.29	1.28	0.76	-2.47	0.23	1.21	1.59	-0.16

Figure 6. Database comparison showing left greater than right frontal asymmetry (courtesy of Q-Metrx, Inc.).

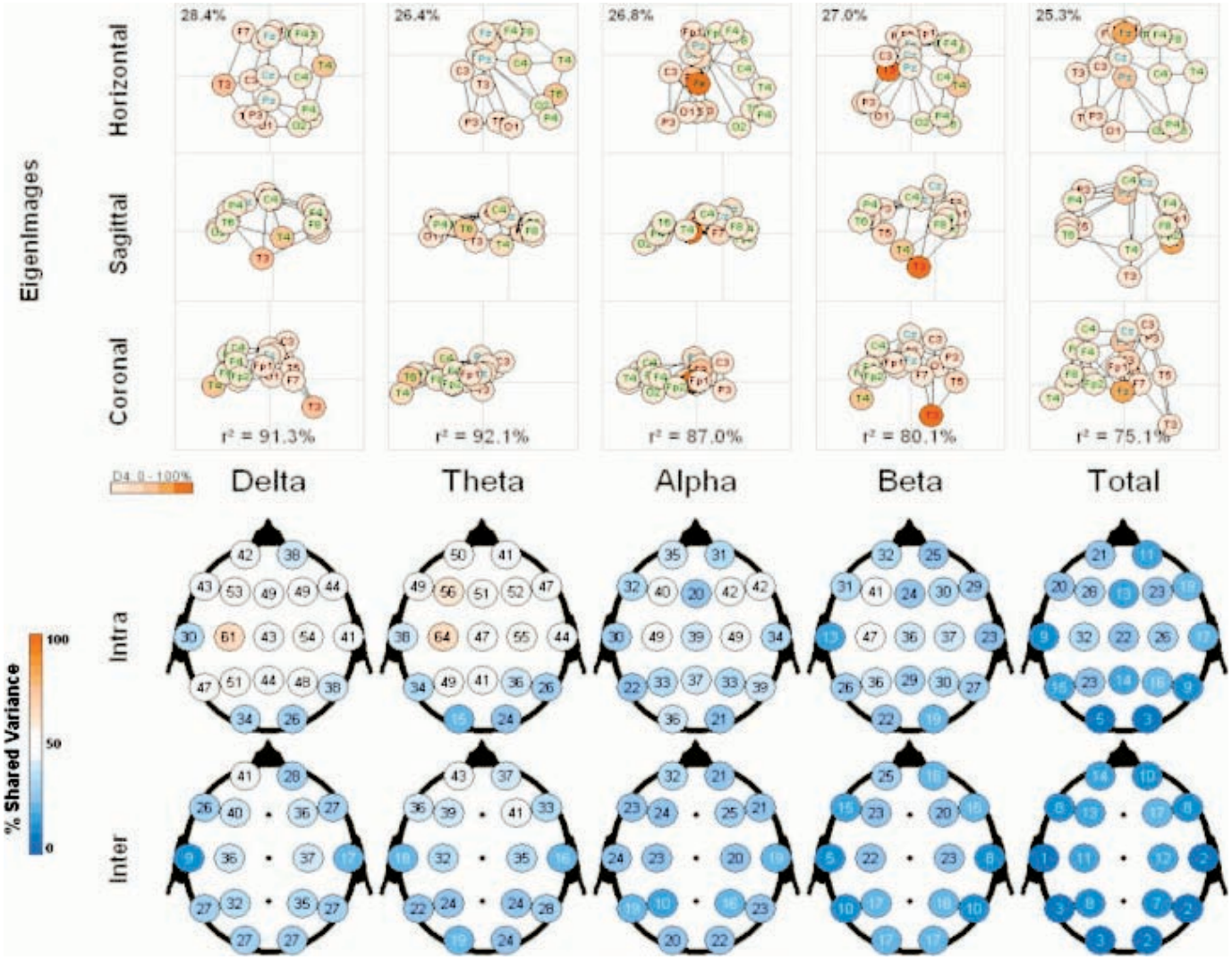

Figure 7. Multivariate connectivity analysis (courtesy of Q-Metrx, Inc.). Sponsorship for color figures provided by BrainMaster Technologies, Inc.

the motor strip). Subsequent protocols might be used to remediate sensory integration or language function, typically with interhemispheric parietal amplitude or synchrony training and regional training over the left frontotemporal cortex. Additional training to promote or reduce connectivity may be useful in conjunction with regional training concerns (e.g., interhemispheric frontal training). Depending on the severity of the clinical presentation, it may not be necessary to include all three stages. It may also be useful to combine these protocols within the same training sessions.

In conclusion, we suggest the utility of a hierarchical model to address neurofeedback protocol development. The primacy of the construct arousal is acknowledged, but more precision is needed to separate this from related concepts such as activation and attention. Furthermore, the distinction between behavioral arousal and cortical and autonomic arousal needs to be made more clear because these distinctions are important in developing neurofeedback protocols. The use of QEEG in identifying specific regions to train also is reviewed, particularly with respect to depression, language disability, and sensory integration disorders. Finally, new techniques can be used to address relationships among regions by rewarding and inhibiting measurements of synchrony, phase, and coherence. The progressive use of these methods allows for application of neurofeedback technology in a wide variety of clinical neurobehavioral disorders.

References

Baehr, E., Rosenfeld, J. P., Baehr, R., & Earnest, C. (1999). Clinical use of an alpha asymmetry neurofeedback protocol in the treatment of mood disorders. In J. E. Evans and A. Abarbanel (Eds.), *Quantitative EEG and neurofeedback* (pp. 181–201). New York: Academic Press.

Coben, R., & Padolsky, I. (2007). Assessment-guided neurofeedback for autistic spectrum disorders. *Journal of Neurotherapy, 11*(1), 3–23.

Fehmi, L. G. (1978). EEG biofeedback multi-channel synchrony training and attention. In A. Sugerman (Ed.), *Expanding dimensions of consciousness* (pp. 155–182). New York: Springer Press.

Fehmi, L. G., & Robbins, J. (2007). *The open-focus brain—Harnessing the power of attention to heal mind and body*. Boston: Trumpeter Books.

Hammond, C. (2007). *LENS: The low energy neurofeedback system*. New York: Routledge.

Henriques, J. B., & Davidson, R. J. (1991). Left frontal hypoactivation in depression. *Journal of Abnormal Psychology, 100,* 535–545.

Hugdahl, K., & Davidson, R. J. (2002). *The asymmetrical brain*. Cambridge, MA: MIT Press.

John, E. R., Prichep, L., & Easton, P. (1987). Basic concepts, methods and results of norm constructions. In A. S. Gevins & A. Rémond (Eds.), *Methods of analyses of brain electrical and magnetic signals. EEG Handbook* (pp. 449–495). Amsterdam: Elsevier Science Publishers B.V.

Johnstone, J. (2002). Bispectral analysis of the EEG: A brief technical note. *Journal of Neurotherapy, 6*(3), 77–81.

Johnstone, J., Gunkelman, J., & Lunt, J. (2005). Clinical database development: Characterization of EEG phenotypes. *Electroencephalography and Clinical Neuroscience, 36*(2), 99–107.

Johnstone, J., Hongmei, C., Smith, C., & Greenwald, S. (2008). *Monitoring depth of normal sleep using the EEG Bispectral Index (BIS™)*. Manuscript submitted for publication.

O'Reardon, J. P., Solvason, H. B., Janicak, P. G., Sampson, S., Isenberg, K. E., Nahas, Z., et al. (2007). Efficacy and safety of transcranial magnetic stimulation in the acute treatment of major depression: A multisite randomized controlled trial. *Biological Psychiatry, 62,* 1208–1216.

Othmer, S. (2006). *Educatief materiaal voor download*. Applied Neuroscience Foundation. Retrieved September 11, 2008, from http://www.appliedneuroscience.nl/file.php?fId=138

Othmer, S., Othmer, S. F., & Kaiser, D. A. (1999). EEG biofeedback: An emerging model for its global efficacy. In J. R. Evans & A. Abarbanel (Eds.), *Introduction to quantitative EEG and neurofeedback* (pp. 244–310). San Diego, CA: Academic Press.

Sterman, M. B. (1982). EEG biofeedback in the treatment of epilepsy: An overview circa 1980. In L. White & B. Tursky (Eds.), *Clinical biofeedback: Efficacy and mechanisms* (pp. 311–330). New York: Guilford Press.

Strehl, U., Leins, U., Goth, G., Klinger, C., Hinterberger, T., & Birbaumer, N. (2006). Self-regulation of slow cortical potentials: A new treatment for children with attention-deficit/hyperactivity disorder. *Pediatrics, 118,* 1530–1540.

Togo, F., Cherniack, N. S., & Natelson, B. H. (2006). Electroencephalogram characteristics of autonomic arousals during sleep in healthy men. *Clinical Neurophysiology, 117,* 2597–2603.

Jack Johnstone

Correspondence: Jack Johnstone, PhD, Q-Metrx, Inc., 1612 West Olive Avenue, Suite 301, Burbank, CA 91506, email: jack@q-metrx.com.