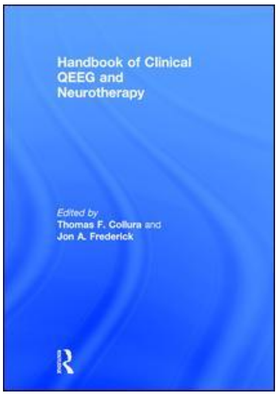


Handbook of Clinical QEEG and Neurotherapy

Thomas F. Collura, Jon A. Frederick

Perspective and Method for a QEEG Based Two Channel Bi-Hemispheric Compensatory Model of Neurofeedback Training

Publication details
https://www.routledgehandbooks.com/doi/10.4324/9781315754093.ch19
Richard Soutar
Published online on: 17 Nov 2016

PART VI

QEEG and Brain Dynamical Approaches

19

PERSPECTIVE AND METHOD FOR A QEEG BASED TWO CHANNEL BI-HEMISPHERIC COMPENSATORY MODEL OF NEUROFEEDBACK TRAINING

Richard Soutar

Abstract

Neurofeedback has recently evolved rapidly into a wide variety of technical methods seeking to enhance efficacy and reduce treatment time. However, more conservative traditional methods employing more sophisticated perspectives, such as the two channel approach, may have been overlooked with respect to their clinical potential. This chapter reviews an exploration of a basic two channel bi-hemispheric methodology, based on QEEG, and grounded in a traditional arousal model of neurofeedback enhanced by recent findings in neuroimaging. This method also calls for a bio-psycho-social approach to clinical neurofeedback as well as the recognition and management of metabolic and psychosocial limitations that can confound training outcomes.

Introduction

Over the last decade neurofeedback has evolved into a wide variety of effective methods which have developed increasingly complex techniques and rationales. Some methods only bear a faint resemblance to the original single channel approach which is presently still the primary method being investigated at the research level for efficacy. The more complex methods demand a high level of sophistication and technical background in math and science that many clinicians entering the field find alien to their clinical training as well as intimidating. Consequently a large number of practitioners apparently continue to use a one channel training approach. On the one hand, the research literature in the field of neurofeedback has established the efficacy of this paradigm (Arns, de Ridder, Strehl, Breteler, & Coenen, 2009; Monastra, 2005; Rossiter, 2004) and defined the specific protocols that can be used effectively by a conservative practitioner. On the other hand, the newer methods are an effort to enhance the efficacy of neurofeedback through more complex and technologically sophisticated means to reduce the number of sessions required to achieve significant results. Whether this has been accomplished remains to be proven through equivalent research but clinical reports are promising.

The shift in the more traditional approach from single to multichannel protocols happened very rapidly, when compared to the reign of the one channel paradigm. A cursory review of journal articles and conference workshops show there was only a limited exploration of an emerging two channel method such as proposed by Baehr, Rosenfeld, Baehr, and Earnest (1999), and Valdeen Brown (personal communication, January 15, 1998), or the coherence methods of training such as represented by Horvat (2009). At the same time, QEEG guided neurofeedback in several different forms also expanded assessment methods and protocol derivation rationales and an ISNR position paper recommended it strongly as a future direction of the field (Hammond et al., 2004). Having trained other professionals in neurofeedback methods for over 15 years, I have observed that the complexity of QEEG has been daunting to many practitioners and remains so to this day. As the field leaped forward into complex multichannel methods, as well as adding a host of other neuromodulation technologies, it seemed important to our team of practitioners to fill in the gap between the simple and rapidly emerging complex approaches through a more thorough exploration of the dynamics of two channel bi-hemispheric training. We were further inspired by the emerging findings in neuroimaging literature that were highly pertinent to this approach, especially the work of Alstott, Breakspear, Hagmann, Cammoun, and Sporns (2009), Davidson (1995), Heller, Nitschke, Etienne, and Miller (1997), Pascual-Leon (2005), and others, which appeared to be supportive of this direction. At the same time, the utilization of QEEG to improve outcomes through more effective strategies of sensor placements seemed an important enhancement. The new emerging findings about network theory and an explicit systems approach to explain local and regional activity were also supportive of this perspective (Buzsaki, 2006; Freeman, Ahlfors, & Menon, 2009; Honey, Kotter, Breakspear, & Sporns, 2007; Meehan & Bressler, 2012). However, providing a simplified or streamlined approach to QEEG analysis and protocol derivation would be necessary to increase the accessibility of the technology to clinicians wishing to enter the field.

As commercial QEEG databases became available in the mid to late 1990s and pre/post training maps became more common among advanced practitioners, certain puzzling aspects of the technology became apparent. One such feature was the tendency for clients to report and demonstrate very significant changes in symptomology, through measures such as the Test of Variables of Attention (TOVA) and the Beck Inventories, yet display only modest changes in a normative direction in their maps. We had five clinics across the country at the time and recorded hundreds of such cases. Our discussions with other clinicians who were utilizing QEEG at that period confirmed our observations. At that juncture we also employed every reported method of neurofeedback we could find as well as peripheral biofeedback, photic stimulation, HEG, Alpha Stim, and adjunctive clinical methods such as EMDR. Our conclusion was that since QEEG is an enduring signature (John, Prichep, Fridman, & Easton, 1988), any significant change in the QEEG was likely to be positive if it was associated with positive changes in symptoms. Some pre/post maps moved significantly toward a normative pattern and others moved in more complex ways, often with areas moving away from the norm. It is traditional among practitioners to show their best maps and cases when explaining the technology because the displayed changes are tacitly didactic. However, it is not always the norm. Once again, emerging research in the neuroimaging field has begun to explain this conundrum through the theory of compensatory response (Pascual-Leon, 2005). Compensation and plasticity are the two terms that ground our entire methodology. We felt it was important to measure the percent change in a map to more effectively define significant change in brain function with respect to QEEG measures.

Another conundrum also emerged over time as our clinical cases increased into the thousands. Some individuals responded robustly to neurofeedback and others did not. Clinicians typically look to their equipment or protocols as the point of failure and yet this ignores other more obvious confounds. Judith Lubar (Lubar & Lubar, 1999) found that events in the family system could profoundly alter the efficacy of neurofeedback. To make this kind of observation requires appropriate clinical skills that many practitioners who are not trained in clinical psychology or counseling, including family systems theory, do not have. In fact, without this training many professionals are likely to be blind to this effect. We determined it was critical to consider these outside confounds and manage them to produce a higher efficacy level of neurofeedback rather than focus on technology alone.

We found adding this dimension of intervention to our practice enhanced our efficacy far more than just changes in equipment or protocols. To ignore this aspect of training is to disregard decades of research in the behavioral sciences. A cursory reading of Alan Schore's (1994) work regarding the impact of trauma on the emotional development of the brain and the behavioral consequences that ensue should be sufficient to convince the average clinician.

An additional confound we encountered with respect to outcomes was in the biological realm. In reviewing the trend screens from session to session we frequently find that although significant changes are made during the session, sometimes there is considerable backsliding with respect to progress. When no psychosocial confounds are uncovered we are typically inspired to investigate physiological or metabolic confounds. Since the recruitment of metabolic resources is critical to neuro-metabolic processes, any deficit in this area is likely to limit the ability of the brain to respond to challenges. The brain uses a large amount of the body's resources (Raichle & Gusnard, 2002; Tomasia, Wang, & Volkowa, 2013) and is sensitive to changes in these resources (Kilner, Mattout, Henson, & Friston, 2005). It is reasonable to assume, based on the present research, that every brain has metabolic limitations based on its resources, especially glycogen, glutamate, and lactate, as well as sodium and potassium (Be'langer, Allaman, & Magistretti, 2011). The effect, for instance, of hypothyroid function on the brain is well documented (Niedermeyer & Lopes da Silva, 2005) and levels of Thyroid T3 have been shown to be linked with critical neuronal function including enzyme production, the production of extracellular matrix proteins, glutamate regulation, growth factors controlling neuronal growth, and neuritogenesis (Gilbert & Zoeller, 2010; Trentin, 2006). Hypothyroid clearly manifests as slowed alpha while increasing its power. Attempting to downtrain this metabolic limitation is generally futile, but hormone supplementation can alter a pre/post map rapidly. Unfortunately many clinicians are not trained in this area and do not recognize such limitations, again turning to better equipment technology for a solution. We found that treating these metabolic deficiencies greatly enhances clinical outcomes.

As an outcome of these historical findings in our clinical records, we developed an agenda to review every case from a bio-psycho-social perspective. This approach assumes a Diathesis Stress model (Zubin & Spring, 1977) that was introduced into psychology to explain the emerging research findings on schizophrenia, such as the concordance studies being conducted in that period. The assumption is that stressors in any one of these three dimensions can result in the elicitation of genetic weaknesses and related symptomology. It is parallel to Mark Schwartz's model of "Window of Vulnerability" as outlined in *Biofeedback: A Practitioners Guide* (Schwartz & Andrasik, 2003). We have developed our own integrated assessment system to process and integrate measures and reports but this approach can be done with any good variety of measures commercially available. It could easily be done with standard inventories and available databases. We prefer a higher level of integration and have developed our own and will be using that for didactic purposes in the case presentation that follows. We have discovered that making these measures easily accessible means they are more likely to be employed by our clinicians for more effective outcomes. In addition we have chosen a difficult case that only partially resolves at the QEEG level but significantly resolves at the symptom level to more effectively demonstrate our points of interest. From our 20 years of clinical experience with neurofeedback we believe that a more comprehensive approach such as follows is a more productive direction to pursue than purely enhancing neurofeedback modalities.

Rationale for the Two Channel Method

Arousal Theory, Cognition, and the Vertical Axis

In the field of psychology, as well as psychiatry, the arousal model has been well established for decades. Sterman (1996), drawing on his own pioneering research, recruited this perspective specifically with respect to neurofeedback (NFB) and Abarbanel and Evans (1999) explicated it in more extensive detail at a later date. Sterman constructed his perspective on neurofeedback based on the experimental observation that repeated changes induced in the sensory motor rhythm (SMR) activity correlate with important permanent changes in the striatum (Sterman & Egner, 2006). In his model, Sterman (1996) proposed that three systems of brain activity influence thalamic generation of EEG at the scalp. The vigilance system involves the reciprocal relationship between specific centers in the brain stem and ascending inputs to thalamic, limbic, and cortical regions. The sensorimotor system involves ascending proprioceptive inputs to the thalamus and sensorimotor cortex and related cortical afferents. The cognitive integration system involves neural centers that process and integrate sensory and motor activity.

Abarbanel and Evans (1999) note that Sterman's theory did not directly include the role played by limbic oscillatory activity in the manifestation of cortical EEG. He does, however, lay out how limbic activity may influence attentional mechanisms. Fortunately, Kirk and MacKay (2003) do outline mechanisms by which low frequency theta activity related to emotional processing is shifted through arousal mechanisms involving the mammillary pathways into higher frequency theta that results in memory processing. The investigation of Morillas-Romero, Tortella-Feliu, Bornas, and Aguayo-Siquier (2013), showing the relationship between attention and emotion, further documents this relationship nicely. Emotion works hand in hand with cognition at the neurophysiological level (Demasio, 1994, 1999) and this critical finding has considerable implications for psychology and neurofeedback.

The idea that specific networks become readily available at each unique level of arousal is indirectly well established in the psychology literature. Performance is arousal dependent according to the Yerkes-Dodson Law (Diamond, Campbell, Park, Halonen, & Zoladz, 2007). Sterman's arousal theory resonates well with this perspective. Various tasks call forth different levels of arousal and recruit different networks. Simple or well learned tasks require less arousal while more complex or new tasks require more arousal. This has recently been verified in the developing research around the Default Mode Network (Buckner, Andrews-Hanna, & Schacter, 2008). Long-Term Potentiation (LTP), the primarily mechanism involved with memory consolidation, is most efficient at moderate glucocorticoid levels and suggests learning consolidation is arousal dependent (Diamond, Campbell, Park, Halonen, & Zoladz, 2007). A more challenging task recruits more networks and increases metabolic load on the cortical system reducing alpha idling of networks and increasing both cell column activity (beta) and hemodynamic response (Kilner et al., 2005). The hemodynamic response is merely an effort of the astrocytes to provide resources, if they are available, to the neurons as they become progressively active to process the task at hand. If the resources are not available then metabolic limitations will clearly hamper the learning process. The shifting of network resources due to the orienting response has been traditionally observed under the term desynchronization and is a well established principle of electrophysiology (Niedermeyer & Lopes da Silva, 2005). A high value stimulus triggers complex or automatic responses that in turn increase arousal and recruit networks associated with higher arousal levels. Task expression becomes state dependent, calling forth different networks from various regions of the brain based on arousal (Fan et al., 2012). Consideration of these findings invites an emergent network systems perspective with respect to the expression and integration of cognitive functions that transcends simple anatomically defined distinctions regarding function such as Brodmann areas (Breakspear, Jirsa, & Deco, 2010; Nakagawa, Jirsa, Spieglar, McIntosh, & Deco, 2013). This in turn suggests that strategic intervention from a systems perspective (Othmer, Othmer, & Kaiser, 1999) is most likely to be effective when implementing neurofeedback. Specificity of training may not be as important as accuracy in selecting a general network to train with upstream and downstream influences considered. Based on our present limited understanding of networks, we may be most effective at attempting to establish optimal attractor states in meta-networks involving Rich Club Hubs using norms as an approximate or fuzzy reference. These Rich Club Hubs have a dominant influence on network activity because they have the most connections with the shortest pathways between network nodes in what is now recognized as a Small World scale free network design that appears to define brain networks in general (Meehan & Bressler, 2012). In reviewing Laird et al. (2012), it can readily be seen that a large number of these hubs, by a strange twist of fate, can already be found to closely correlate with the present 10–20 system.

It is well understood at this point in psychology that memory is frequently state dependent. Buzsaki's (2006) work on hippocampal theta has led to a general recognition that theta and gamma are linked in function. Meehan and Bressler (2012), in reviewing the literature, note that gamma in turn triggers beta activity and consequently all three are tied together. Hence the underlying reasons for the value of training beta to theta ratios or enhancing beta frequencies while inhibiting theta frequencies or the relationship between fast waves and slow waves. One group of researchers proposed that this be considered a measure of inhibitory control of the cortex over subcortical domains. Their preferred term for this mechanism was "Brain Rate" (Pop-Jordanova & Pop-Jordanov, 2005). As arousal increases and decreases, sympathetic tone shifts with corresponding shifts in activation between hemispheres as well as shifts in frequency ratios between hemispheres along the Horizontal Axis. The spectral shift from slow wave to more fast wave dominance and beta arousal is tied to the Horizontal Axis (interhemispheric) through its classical reciprocal relationship with alpha.

Arousal Modification, Affect Regulation, and the Horizontal Axis

The extremes of approach and avoidant behavior and their correlates of sympathetic tone require more refined ongoing adjustment and modification to implement social behavior than that which the basic ascending reticular arousal mechanisms might offer. This modification of arousal appears to be mediated by the ongoing dynamic relationship between the left and right hemispheres. Davidson's (1995) research explored the electrophysiological aspects of this consideration and found that higher levels of alpha amplitude in the right hemisphere with respect to the left hemisphere resulted in a propensity towards a reduction in approach type of social behaviors and a tendency for depression. Since that time Choi et al. (2011) has demonstrated the efficacy of asymmetry neurofeedback to reduce features of depression using a random trials design. In addition, Davidson found that this alpha asymmetry correlated with lower arousal levels in the left hemisphere with respect to the right hemisphere. Heller, Nitschke, Etienne, and Miller (1997) reported that beta asymmetry, with higher beta amplitudes in the right hemisphere with respect to the left hemisphere, correlated with anxiety of various kinds. Avram, Baltes, Miclea, and Miu (2010) had similar findings. The research of Heller et al. shows that enhanced right hemisphere beta results in increased sympathetic tone and often corresponding anxiety. It appears that a traumatized network system may become stuck in an overaroused hypercoupled state involving excessive cell column activity and corresponding beta asymmetry (elevated right hemisphere beta). Further research has revealed this can include excessive frontal or posterior beta activity, depending on the level of chronic exposure and whether the response is worry, panic, or rumination (Engels et al., 2007). This increased tone enhances norepinephrine activity and adrenal response as well as upregulating the ACTH system. Over time the body inevitably begins to suffer the consequences of chronic hyperarousal (Sapolsky, 1999) including neuronal death and telomere shortening (Epel et al., 2004). Hans Selye noted that this trend leads to exhaustion of the organism and death. An alternative response available to the organism is withdrawal from stereotypical activities that lead to frustrating situations that may expose it to trauma (Beck, 1979). Since this is correlated in humans with social withdrawal activity (Davidson, 1995), it may be concluded that alpha asymmetry and depression constitute a protective response. It not only includes a reduction in dopamine but reductions in serotonin. In this unique state, the organism is alert to danger and functional but hesitant to engage. This would buy an organism time to incubate novel alternative behaviors and recover some measure of resources. From this perspective depression is a protective moratorium from overarousal levels that can lead to severe excitotoxicity. In viewing depression and anxiety from this perspective they provide a unique balancing act at an elevated level of arousal. We define this left/right dimension of training as the Horizontal Axis.

Baehr et al. (1999) introduced an asymmetry protocol developed initially by Peter Rosenfeld based on the research by Richard Davidson, as mentioned above. This protocol involved training homologous sites, F3-F4, using two channels of EEG (one for each side). Variations of this protocol circulated in the neurofeedback community at the time. The goal of this type of protocol was to increase alpha activity in the right hemisphere and decrease it in the left in order to enhance left hemisphere activation. The Horizontal Axis, however, has perhaps a more complex dynamic than appreciated at the time. One recognized aspect of this dynamic is found in Pascual-Leone's (2005) research on compensatory mechanisms of the brain. TBI in one hemisphere can lead to reductions in transcallosal inhibitory mechanisms that in turn result in one hemisphere co-opting function in the contralateral hemisphere to maintain an allostatic state while the injured region goes offline for repairs. Alstott et al.'s (2009) modeling of lesions predicts this outcome as well. The implication is that training a region of diminished function should take into consideration contralateral inhibitory mechanisms. A two channel protocol to manage the effect of training a specific dysregulated site on a contralateral site and its reciprocal response allows for the monitoring and management of compensatory mechanisms. The reason for opting for homologous sites is based on the reported bilateral nature of major functional networks (Laird et al., 2012). It also suggests that fine tuning of a protocol should consider innate hemispheric norms and dynamics as well as compensatory mechanisms between frequency domains as well as functional domains. Teipal et al. (2009) notes that interhemispheric coherence very efficiently and accurately reflects the integrity of intracortical and subcortical fiber systems. He also argues that interhemispheric coherence is also a proxy measure of intrahemispheric integrity. Consequently a high level of leverage over cortical function should be expected from training in this manner and utilizing amplitude to shift coherence is the most conservative approach. Careful adjustment on the fly in the reinforcement rates of the amplitude enhancement and inhibits of the various component bands based on their interhemispheric response allows for an optimal adjustment of reinforcement in harmony with the unfolding interhemispheric dynamics of the training session. Monitoring a trend or review screen in real time as the training progresses, while using a two channel monopolar montage, allows the clinician to observe emerging trends in symmetry between hemispheres as well as the layering or distribution of component bands that indicate arousal levels. Additionally, coherence measures can be monitored to discern the level of compensatory activity in response to changes in reinforcement rates. Increasing and decreasing coherence rates can be observed to follow amplitude patterns as homologous sites increase and decrease communication in an effort to move in a relatively normative direction. This activity in amplitude and coherence, however, often involves long periods of movement far outside normative ranges of activity and can be easily observed as the brain seeks an optimal autocorrelative solution. By the avoidance of limiting the compensatory activity with arbitrary ranges of norms, the maximum adaptive responses can be encouraged. These homologous sites appear to provide maximum leverage in the system.

Theoretical Conundrums of QEEG Assessment and Application

Given the existence of a bewildering number of interacting specific systems in the brain, challenging a system to increase regulation through operant conditioning of brainwaves would seem problematic. Through QEEG we can identify regions of the brain that are dysregulated but it is often a very complex picture with many levels of possibility with respect to training protocols to employ. Meehan and Bressler (2012) and others in the neuroimaging community note that network systems in the brain display a pattern of effective connectivity that is Small World with a Rich Club Hub system that dominates information exchange. Honey et al. (2007), McIntosh and Korostil (2008), and many other key researchers comment that networks are typically situated with upstream modulation and downstream feedback loops that can potentially instigate or maintain dysregulation. Identifying the exact source(s) of dysregulation transcends our technical abilities at present. With respect to QEEG analysis and protocol development, this results in a difficult and unclear decision making conundrum.

Early NFB practitioners utilized protocols that implicitly employed a systems perspective. One electrode location was strategically selected to generate change in the entire system. In fact, Sterman's experiments had implicitly proven this to be the case. A careful reading of his research showed that he was able to influence temporal lobe instability by training over the motor strip with a monopolar placement. In fact he reported that training over the seizure location was not as effective as training over the motor strip (Sterman, 2000). Over time, however, clinical experience clearly demonstrated that there were distinct advantages and disadvantages in training in different locations. Unfortunately, the rules were not always clear. Both Demos (2005) and Soutar (1999; Soutar & Longo, 2011) proposed guidelines for what could be done in each quadrant of the brain in terms of positive and negative outcomes; however, the underlying mechanisms that contributed to them were unclear as the research community was still struggling with network theory as it is today. As practitioners moved from an implicit systems theory approach to a more QEEG or location based approach, it appears to us that they began to lose sight of the implicit systems perspective. The focus on location in some instances bordered, and perhaps still does, upon electronic phrenology. The idea of always localizing a functional problem to a small ROI is problematic when considering the level of connectivity of the brain and the diffuse nature of EEG.

The Value of Location

To date, the majority of NFB practitioners have developed location based training from decades of clinical experience demonstrating that clients respond differentially to training, both different frequencies and different locations. This is consistent with the above observations in the neuroimaging literature. A unitary vision of timing and timing remediation that could alter the entire system is too simplistic and ignores local systems dynamics and their influence and contribution to an entire complex system such as the brain. Within any system such as the brain, it makes more sense to assume multiple timing events that are autocorrelative (Bassette, Meyer-Lindenberg, Archard, Duke, & Bullmore, 2006; Buzsaki, 2006). Efficient remediation is frequently likely to be local and strategic, often requiring the tuning of several networks. In addition, there is the decision to be made regarding training modality, i.e. amplitude versus coherence. Given the compensatory nature of phase and coherence (Alstott et al., 2009; Pascual-Leon, 2005), the established dangers of undermining these mechanisms, and the documented negative clinical consequences associated with training these dimensions (Horvat, 2009), it seems reasonable to conclude that amplitude training is a very reliable and conservative approach. Training amplitude by definition alters the other dimensions of measurement but at the brain's discretion. Although these measurement domains are mathematically discrete, they are nonetheless interdependent at the neuronal level with respect to the physics of brainwave production (Nunez & Srinivasan, 2006). Any clinician can easily confirm this by reviewing coherence, phase, symmetry, and dominant frequency while amplitude training. It is simple with today's technology to train any one of these domains and observe the global as well as specific response. Given the interdependence of these domains and the efficacy, reliability, and safety of training amplitude, it makes sense to continue to develop the one channel amplitude perspective into a multichannel amplitude perspective beginning with a thorough understanding of two channel training dynamics. The work of Baehr, Rosenfeld, and Baeher (1997) and Baehr et al., (1999) originally moved in this direction. Yet to our knowledge, little research or clinical reporting followed.

Applying the Standard Bi-Hemispheric Protocol

As mentioned above, the work of Kilner et al. (2005) and others demonstrated that a strong bidirectional link exists between electrophysiology and hemodynamics and consequently between NFB and hemodynamics. Training down slow waves and increasing fast waves typically increases demand on astrocytes to increase lactate production for neuronal metabolism, which in turn places demand on local capillaries to provide hemoglobin and glycogen. In view of this relationship it actually makes more sense to increase beta in the LH and reduce slow wave activity (delta, theta, or even alpha) in order to enhance perfusion and generate greater activation than to just train alpha down. The contrary should hold for the right hemisphere. Training alpha or theta up and beta down should more robustly exert pressure to reduce perfusion and metabolic activity. There are neurotransmitter correlates to this as well. Activating the left hemisphere exerts pressure on the neural system to increase dopamine output while reducing activation in the right hemisphere exerts pressure to decrease norepinephrine output (Davidson, 1995). From this perspective, hemispheric balance of activity provides a general improvement in system function but can be focused and modified by shifting to different meta-networks to address regional dysregulation related to more specific functions. Training different homologous sites allows the practitioner to address more specifically symptoms associated with these locations while moving the system globally in a normative direction. This complex dynamic integrates emotional spectrum of variation with cognitive spectrum of variation. Training left–right asymmetry indirectly trains arousal.

Typically, then, we enhance beta in the LH while reducing slow wave activity based on the map findings. If we are reducing delta or theta then we are emphasizing what I have termed the Vertical Axis of arousal, and increasing inhibitory control and general signal to noise ratio in the brain. If we are reducing alpha, then we are emphasizing what I have termed the Horizontal Axis of arousal, which further modifies the effect of arousal and integrates limbic and cortical activity as well as cognitive attention and emotional functions. By the same token, in the RH, we are reducing delta or theta to reduce slow waves on the Vertical Axis or we enhance alpha to train the Horizontal Axis. We may also inhibit beta in the RH to train horizontally. In such cases it is important to increase the enhancement of beta in the LH as it will tend to reduce in amplitude. Training a frequency in one direction in either direction tends to train it in the same direction on the contralateral side. Our goal is to maintain or encourage normal asymmetry between alpha and beta. However, we must allow room for compensatory excursions in amplitude and coherence as the system reorganizes itself around successive attractor states in the progressive iterations that lead to optimal functioning. Alpha should tend to be higher in the right hemisphere and beta should tend to be higher in the left hemisphere when reviewing the grand averages on the training screen.

At the same time we train horizontally, we are watching for slow to fast wave ratios to normalize along the Vertical Axis. Montgomery, Robb, Dwyer, and Gontkovsky (1998) reported that with eyes open, delta and theta should dominate the spectral distribution with beta being approximately half the amplitude of the slow wave at the vertex. In our analysis of hundreds of normal QEEGs at our clinic we noted that this relationship roughly held across the scalp. This was confirmed more recently by Almurshedi and Ismail (2014). With this in mind, can train along the Vertical Axis of arousal in the LH by inhibiting theta while training along the Horizontal Axis of arousal in the RH by increasing alpha or SMR. Thus both Axes can be trained at the same time through different combinations of enhancements and inhibits. We expect hemispheres to shift symmetry continuously during a session as a reflection of plasticity in the system and healthy mood fluctuation in response to the narrative in the training video. Typically, abnormally high amplitudes in slow waves such as alpha or theta will shift down as symmetry is normalized and vice versa. We have thousands of training records to support this observation.

From this discussion it should be clear that we are training regional network dynamics in selected bilateral networks based on statistical determination of the most dysregulated bilateral network. The question then arises regarding which network is most dysregulated.

Upstream and Downstream Contributions to Dysregulation

Through analyzing the abnormal standard deviations of each location in each dimension of magnitude (or power), dominant frequency, asymmetry, coherence, and phase, we can determine which locations are most globally abnormal with respect to dimensions of analysis. Typically, training in the worst bilateral network will generate a positive response. However, Honey et al. (2007) and others have noted that upstream or downstream networks that are dysregulated may be the actual source of dysregulation for a network hub. Typically, the disturbing input is from upstream, which is usually a more posterior location. By training the most dysregulated network posterior to the identified worst network, better responses can be obtained. The only way to determine this is to actually try training both networks to find the optimal response. In either case, the clinician is training the brain toward a more normal distribution based on the QEEG findings. By training each network into a more balanced Vertical and Horizontal relationship, several iterations of training and mapping can be done. Typically each remap shows between 30–40% change, when degree of deviation is tallied, between pre and post maps when things are proceeding well. Fifty percent change is usually the most we usually encounter in our system of analysis. The percent change is not additive, as each map represents a new configuration of the entire system as it cycles through progressive iterations of adjustment. This is difficult for new clinicians to grasp as they tend to expect change in a linear succession. The good news is that at each remapping we characteristically observe fewer compensatory changes away from the mean and more changes toward the mean.

Methods

Bio-Psycho-Social Assessment and Tracking

The first step in our assessment process is to statistically determine the most deviant locations based on a weighting method that assigns a value for each location based on its z score in magnitude, dominant frequency, phase, coherence, and symmetry. The symmetry value used in the calculation is derived from magnitude difference between left and right hemispheres rather than a z score value. Each location is paired with its homologous site on the contralateral hemisphere. Frequently these rank next to each other in the list. Eight sites are listed in rank order with the top sites selected as the most likely location to generate maximum results.

Once a site is selected it is evaluated for frequency deviances and an initial training strategy is generated. A typical example would be downtraining theta in the left hemisphere and uptraining Lo Beta in the right hemisphere. The training strategy is further adjusted by clinical rules considerations based on the Quadrant Rules (Demos, 2005; Soutar, 1999; Soutar & Longo, 2011), i.e. not to train beta up in the right posterior quadrant. A third stage of analysis is also applied in which typical compensatory dynamics are considered within the existing frequency context in each hemisphere under applied protocol conditions as observed in the past. A final complex three-frequency approach results typically involving low, mid, and high frequency ranges for each hemisphere. Training is typically done based on separately referenced ears but using linked ears has also generated favorable results.

A monopolar montage is utilized for each hemisphere and either a one or two channel protocol is employed in each hemisphere. Once the first session has been run the training graph is evaluated for significant changes in amplitude in the normative direction. The review screen or trend screen is also evaluated for compensatory patterns such as theta dropping when beta is downtrained in the right hemisphere or alpha decreases in the left hemisphere resulting in beta increases in the right hemisphere. In terms of vertical movement, changes of 2 to 5 μv are significant for delta, theta, and alpha, and changes of 0.5 to 2 μv are significant for beta. These trends are best considered in artifact free analysis with regression lines calculated but can be visually estimated by a practiced clinician. In terms of horizontal movement, trends showing increases of alpha on the left and beta on the right are monitored. The convergence of each left and right hemisphere combination for each component band is usually a sign of enhanced plasticity and tends to correspond with normalization of amplitude on the vertical plane. Maximum normalization typically occurs in the first 15 sessions, marking the end of the acquisition period of training.

Clients are usually observed to have the most significant symptom changes during this period but degrade or backslide partially between sessions. The consolidation period begins with progressive retention of symptom reduction between sessions until symptoms are permanently diminished. Clients are typically backed off to one session per week to confirm retention.

Symptom Tracking

Symptom tracking in our methodology is a critical component of the training process as clients tend to quickly habituate to their new level of function and forget their prior level of distress. The Cognitive Checklist is used to evaluate clients on each dimension for function monitored with EEG map analysis. The checklist provides a series of questions designed to tap constructs utilized in fMRI research and locate regions noted in the research to be hubs and nodes of various networks associated with specific functions such as short-term memory or sequential memory. A projected map of locations associated with client-endorsed items indicating problems is generated and then compared to the EEG generated map for correlations. Correlated items are then sent to an automated symptom tracker, confirmed by therapist and client as significant, and then tracked over time using various graphs. Clients then report level of symptom changes.

Pre/Post Maps

The assumption has been typically made that the brain changes that occur have a linear function and move progressively toward the normative pattern in steady increments over the course of training. This has never been found to be the pattern in the majority of cases we have reviewed over the past three years with our new analysis methods used in approximately 300 clinics. We have seen dramatic cases of rapid shifts in the normal direction but they are the exception. This could be considered a consequence of our training protocols but we have a large number of clinicians also using LENS, Infra Low Protocols, z score, and a host of others while conducting pre and post maps with similar outcomes. None of these has consistently produced linear changes toward the norm across sessions and clients. Consequently, in our opinion, claims to the contrary should be met with skepticism.

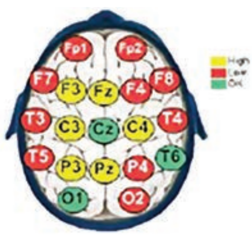
Our pre/post map method reviews the changes toward and away from the norm across all dimensions of analysis and computes a percent change as well as an over percentage of change in any direction. The changes we observe across NFB methods reflect what compensatory theories predict; that there is considerable movement in many locations away from the norm as other areas move toward the norm. It is only reasonable to expect a nonlinear dynamical autocorrelative system with a 1/f power function and over 30 billion neurons with minimal degrees of separation to have a complex solution space. As attractor states shift in each network and allostasis is renegotiated there is considerable compensatory movement away from normative expectations and back again. We observe several cycles of change with considerable retrograde movement during initial acquisition phases of learning and then progressive movement toward the norm in the consolidation phases. These cannot be easily correlated directly with symptom changes and symptom intensity. We typically observe robust changes in QEEG measures in the 35% range within the first 15 sessions and exceptional levels of change in 45–50% range. These changes typically reflect dramatic changes in symptomology.

A Case Study

The subject is a 9-year-old girl diagnosed with anxiety disorder who is afraid to go to school where she frequently has panic attacks. We had her parents fill out the Cognitive Emotional Checklist (CEC) and her symptoms included Worry, Whining, Scary Thoughts, Stuck on Thoughts, Stuck on Behaviors, Argumentative, Bargaining Behavior, Procrastination, Minimal Patience, Disorganized, Careless Mistakes, Lack of Motivation, Poor Follow Through, and Difficulty with Attention and Focus. Some of these features are more anxiety based and some depression based. The lack of motivation, poor follow through, argumentative and bargaining behavior all suggested considerable emerging depression. This was confirmed by a pronounced alpha asymmetry in the headmaps.

The Test of Variables of Attention (TOVA) showed normal performance with an increase in omission errors in the beginning of the second half of the test. We find this is common in children with anxiety disorders.

The CEC (Figure 19.1) indicated that anxiety features dominated the symptom ranking system with 12 items selected at a high score in the anxiety category but also 7 items ranked high in the depression category. Memory items ranked high in 9 items. Attention ranked lowest with 6 items. This suggested that anxiety was the driving force generating loss of attentional function as a consequence of degraded memory functions due to elevated glucocorticoid and cortisol degradation of hippocampal



CEC Response Assessment

Category	Response Count	Average Response
Anxiety	12	2.50
Memory	9	2.11
Impulsive	8	1.75
Depression	7	2.29
Attention	6	2.17

CEC Responses

Answer	Question
3	Procrastination/Puts Things Off
3	Careless Mistakes
3	Disorganization/Forgets & Loses Things
3	Constant Worry
3	List Learning Problems
3	Restless/Impatient/Easily Frustrated
3	Argumentative
3	Scary Thoughts
3	Poor Math Skills
3	Whines Excessively
3	Stuck On Thoughts
3	Stuck On Behaviors
3	Bargains Constantly
3	Fearful Rumination
2	Short Attention Span & Focus

Figure 19.1 The Cognitive Emotional Checklist rank orders questions regarding cognitive and emotional problems by categories of anxiety, memory, impulse control, depression and attention based on responses measured with a Likert like scale. The category of anxiety has the higher average response. Dysregulated locations are predicted in the headmap to the left based on response level to questions regarding symptoms and behaviors that correlate with those locations as indicated in fMRI research.

function. This is a common feature in anxiety and typically shows up as a loss of theta to parietal regions and deficient hippocampal input to Short-Term and Sequential Memory regions.

The client received 40 sessions over a five-month period, training approximately twice weekly. She was trained using a two channel BrainMaster Atlantis amplifier and software with a two channel protocol at C3-C4 with alpha 8–12 inhibited, high beta 20–30 inhibited, and beta 15–20 enhanced at C3, and beta 15–20 inhibited, beta 20–30 inhibited, and alpha 8–12 enhanced at C4. The training location was based on a statistical analysis of amplitude, dominant frequency, phase, coherence, and asymmetry to locate the most deviant locations on all measures. At session 9 we shifted to a bipolar montage using C3-Fz and C4-P4 in order to focus more on the left front and right posterior quadrant. We find this often enhances training when working at C3-C4. The choice of frequency inhibits and enhancements was made in order to shift alpha symmetry, reduce depression, and reduce beta elevations in the right hemisphere related to anxiety. Reinforcement rate changes, such as increasing right hemisphere beta inhibits or decreasing left hemisphere beta inhibits and increasing left hemisphere alpha inhibits, were made on the fly from session to session as needed while observing the trend screen in real time. We view this as "riding the ratios" in response to interhemispheric compensatory changes taking place during the session. These efforts were initiated to enhance movement of trend lines in the desired direction.

The second map (Figure 19.2) shows a 36% change, which is a good average. Note, however, that 80% of the changes were away from the normal distribution and 20% toward the normal distribution.

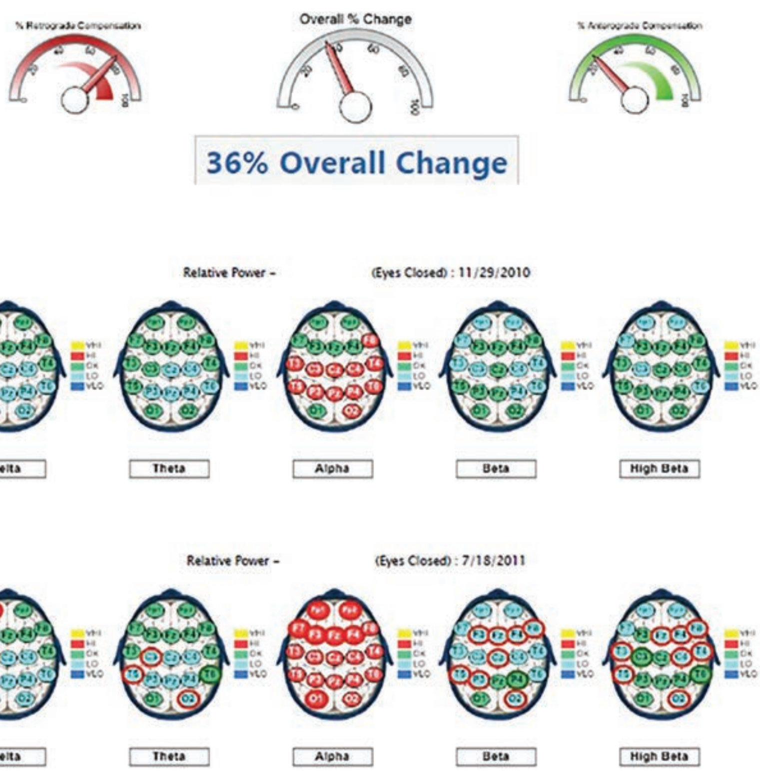


Figure 19.2 The Pre-Post assessment compares the first QEEG with those that follow by calculating the total percent change in a terms of standard deviation in five neurometric dimensions including magnitude, dominant frequency, coherence phase and asymmetry in all 10–20 locations. Of the 36% change approximately 25% of that change was toward the norm (anterograde) and approximately 75% was away from the norm (retrograde).

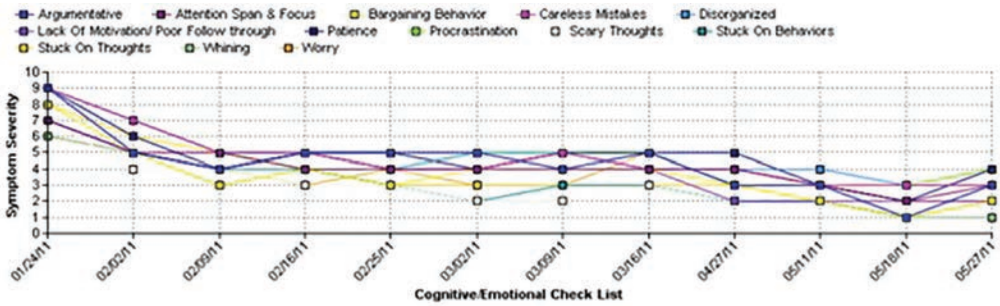


Figure 19.3 This time series analysis shows client symptom severity based on a 10 point Likert rating scale with higher numbers representing greater symptoms. Clients rate themselves during each visit with a tablet linked to their client folder. This graph indicates significant reductions in symptoms over a period of months with greatest changes occurring in the first few weeks and toward the end of the training.

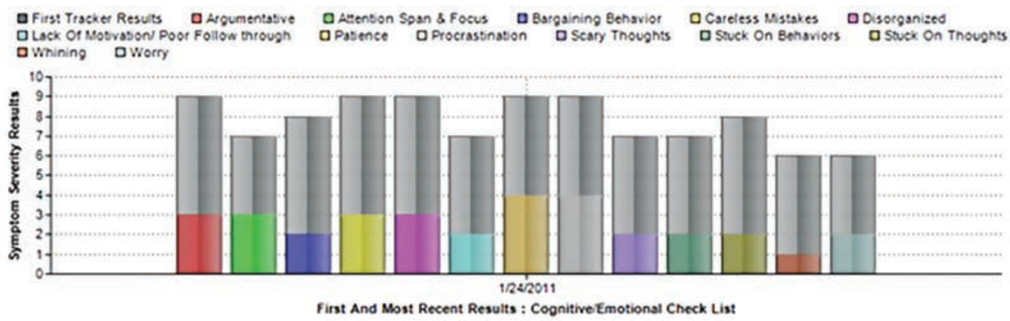


Figure 19.4 This graph shows the same information as the previous one but in the form of bar graphs with the uniform grey component of each bar representing where they started out and the lighter/darker grey (or color) part where they finished up with respect to symptom severity. This graph shows symptom reductions of 60% or more on average with each symptom.

This metric is based upon the simple total percentage of change in measures across all dimensions of analysis. Also note the significant and steady improvement in symptomology during this period (Figure 19.3). This demonstrates how reorganization of any kind with positive changes in symptoms is the most important aspect in the short run. Note that in the next remap (Figure 19.4), the majority of the changes were 60% in the normative direction or anterograde direction while 40% were in the abnormal direction or retrograde direction. This constitutes a distinct improvement in QEEG measures from a statistical perspective with continued improvement in symptoms. The client was enjoying school and singing solos in the school choir. Her insomnia had dissipated considerably along with the majority of her other symptoms. I would like to make special note that she had significant symptom changes by session 15 (in fact, the graph shows the global pattern is 6–8 sessions), which is typical, but we consider this the latter part of the acquisition phase and our experience shows that if we do not train at least another 15 sessions the gains may not consolidate, i.e. become permanent. Also note that we achieved further gains in the last sessions which we would have missed if we had discontinued prematurely.

While the map still showed a dominant diffuse alpha slightly above one standard deviation which did not diminish, her alpha asymmetry resolved and her beta asymmetry diminished (Figure 19.5). These are the most reliable markers, based on the reviewed research, of anxiety and depression and the changes were consistent with her changes in symptomology. This overall pattern of change confirms our observations that each iteration of training and mapping demonstrates a progression from dominant retrograde changes to dominant anterograde changes.

In reviewing the metabolic analysis (Figure 19.6), we concluded that the diffuse elevated alpha that did not respond to NFB was likely due to metabolic issues after reviewing and ruling out problems

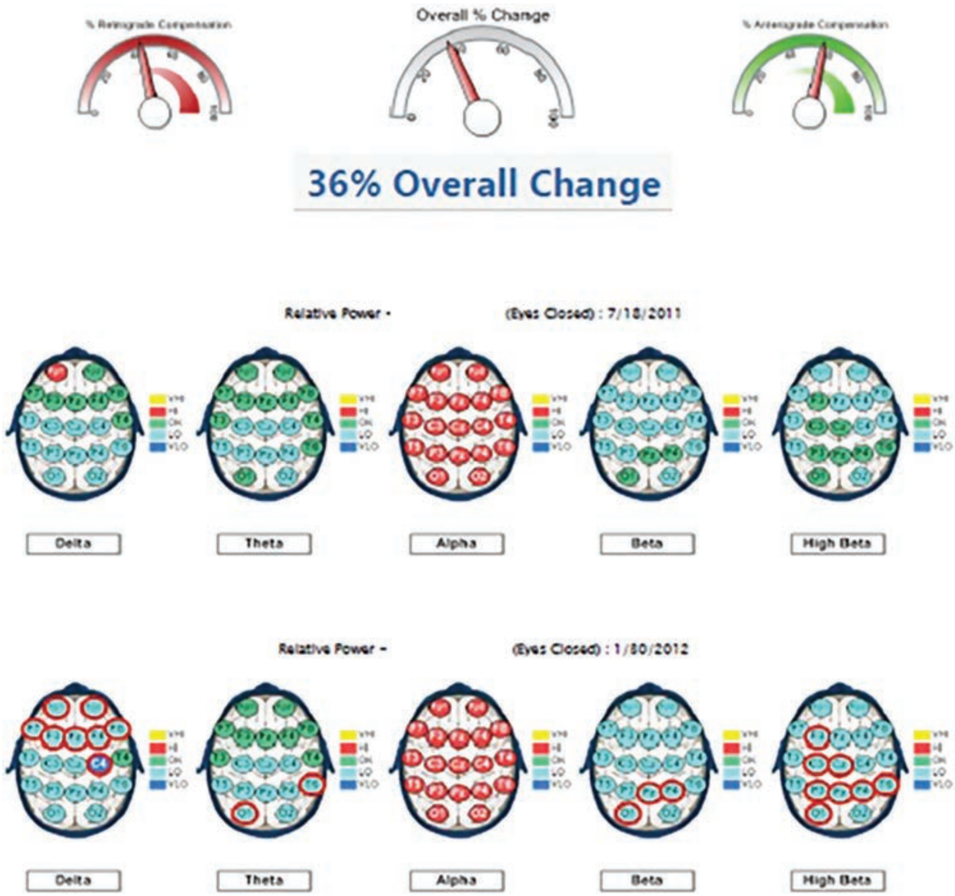


Figure 19.5 This pre-post QEEG compares the last two maps and shows a consistent 36% overall change (which is in the average range statistically) but this time with reduced retrograde movement away from the norm (now only 40%) and increased anterograde movement toward the norm (now 60%). This pattern of change in later phases of training is common in the clinics we have monitored.

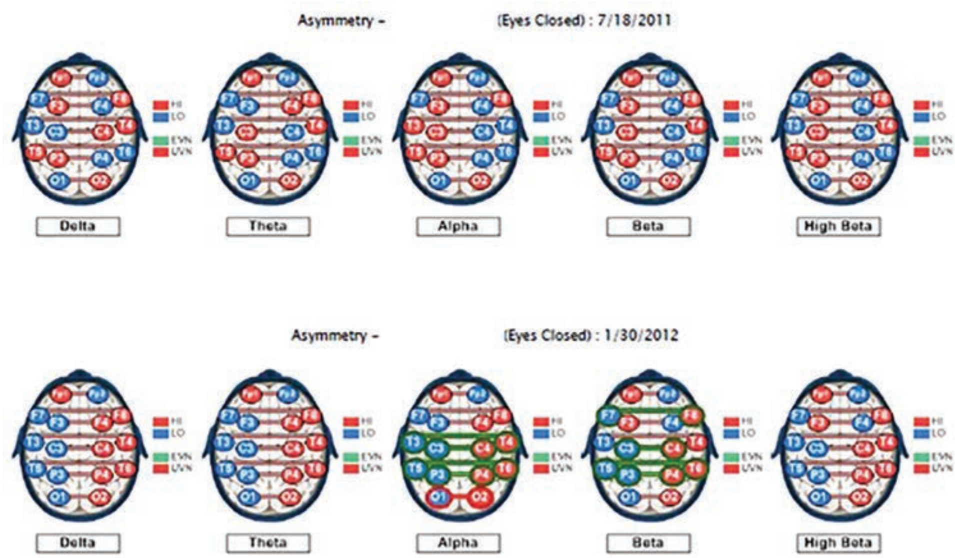


Figure 19.6 The asymmetry headmaps are based on magnitude and not standard deviation and show either red, if the value is highest in that side, or blue if the value is lowest in that side. This map shows improved asymmetry with alpha showing higher on average on the right and beta showing higher on the left. These changes correlate with reductions in depression and anxiety.

with the family system. The high number of symptoms relating to blood sugar regulation as well as gastrointestinal distress and food sensitivities further supported the hypothesis that metabolic sources were responsible for the diffuse elevated alpha. The elevated alpha was in the range of one standard deviation as well so it did not constitute an extreme deviation but rather a mild one.

Discussion

It should be clear from the preceding material that the efficacy of this method is considerable. This case is representative of thousands of cases from hundreds of clinics using the identical analysis methods. By standardizing assessment and protocol implementation methods we have been able to more carefully observe and control outcomes as well as share them. Allowing the brain to determine its own limits of deviation during repeated trials rather than using a normative constraint model is clearly as effective as other more complex modalities. It does not utilize more intrusive measures involving microcurrent stimulation, microtesla induction, or entrainment, but could be used in conjunction with these approaches. Early trials on a large scale involving thousands of cases from clinics around the country have been encouraging and some vendors are using this approach with photic stimulation and what appears to be greater efficacy. The most important advantage of this methodology is that it is grounded in standard NFB peer reviewed methodology and QEEG research and draws heavily from neuroimaging research. It trains clearly identified meta-networks based on recent research and focuses the training emphasis on a complex nonlinear dynamical process rather than just focusing on an ROI. It does not have the accuracy of LORETA training methods but anatomical specificity may be of limited value with EEG biofeedback. The resources required to engage this technology are considerably less expensive than more complex approaches and provides a good springboard for learning theory and advancing into these more complex approaches should future research demonstrate superiority. Typically, entering clinicians are counselors without technical backgrounds and extensive complexity reduces the potential for the expansion of neurofeedback into these professional domains. Several years of experience with this market segment suggests that this approach is a very effective introductory method that addresses these issues.

References

Abarbanel, A., & Evans, J. R. (1999). *Introduction to quantitative EEG and neurofeedback.* New York: Academic Press.

Almurshedi, A., & Ismail, A. K. (2014). Cross coherence independent component analysis in resting and action states EEG discrimination. *Journal of Physics: Conference Series*, *546.* doi:10.1088/1742–6596/546/1/012019

Alstott, J., Breakspear, M., Hagmann, P., Cammoun, L., & Sporns, O. (2009). Modeling the impact of lesions in the human brain. *PLoS Computational Biology, 5*(6), e1000408. doi:10.1371/journal.pcbi.1000408

Arns, M., de Ridder, S., Strehl, U., Breteler, M., & Coenen, A. (2009). Efficacy of neurofeedback treatment in ADHD: The effects on inattention, impulsivity and hyperactivity: A meta-analysis. *Clinical EEG and Neuroscience, 40*, 180–189.

Avram, J., Baltes, F. R., Miclea, M., & Miu, A. C. (2010). Frontal EEG activation asymmetry reflects cognitive biasesin anxiety: Evidence from an emotional face stroop task. *Applied Psychophysiology Biofeedback, 35*, 285–292.

Baehr, E., Rosenfeld, J. P., & Baeher, R. (1997). The clinical use of an alpha asymmetry protocol in the neurofeedback treatment of depression: Two case studies. *Journal of Neurotherapy, 3*, 12–23.

Baehr, E., Rosenfeld, J. P., Baehr, R., & Earnest, C. (1999). Clinical use of an alpha asymmetry protocol in the treatment of mood disorder. In J. R. Evans & A. Abarbanel (Eds.), *Introduction to quantitative EEG and neurofeedback* (pp. 181–200). San Diego: Academic Press.

Bassette, D. S., Meyer-Lindenberg, A., Archard, S., Duke, T., & Bullmore, E. (2006). Adaptive reconfiguration of fractal small-world human brain functional networks. *PNAS, 103*(51), 19518–19523.

Beck, A. T. (1979). *Cognitive therapy and the emotional disorders.* Cleveland, OH: Meridian.

Be'langer, M., Allaman, I., & Magistretti, P. J. (2011). Brain energy metabolism: Focus on astrocyte-neuron metabolic cooperation. *Cell Metabolism, 14*, 724–738.

Breakspear, M., Jirsa, V., & Deco, G. (2010). Computational models of the brain: From structure to function. *NeuroImage, 52*, 727–730.

Buckner, R. L., Andrews-Hanna, J. R., & Schacter, D. L. (2008). The brain's default network: Anatomy, function, and relevance to disease. *Annals of the New York Academy of Sciences, 1124*, 1–38.

Buzsaki, G. (2006). *Rhythms of the brain.* New York: Oxford University Press.

Choi, S. W., Chi, S. E., Chung, S. Y., Kim, J. W., Ahn, C. Y., & Kim, H. T. (2011). Is alpha wave neurofeedback effective with randomized clinical trials in depression? A pilot study. *Neuropsychobiology, 63*, 43–51.

Davidson, R. J. (1995). Cerebral asymmetry, emotion, and affective style. In R. J. Davidson, & K. Hugdahl (Eds.), *Brain asymmetry* (pp. 361–387). Cambridge: MIT Press.

Demasio, Antonio. (1994). *Descartes' error: Emotion, reason, and the human brain.* New York: Avon Books.

Demasio, Antonio. (1999). *The feeling of what happens.* New York: Harcourt Brace.

Demos, J. M. (2005). *Getting started with neurofeedback.* New York: W. W. Norton & Company.

Diamond, D. M., Campbell, A. M., Park, C. R., Halonen, J., & Zoladz, P. R. (2007). The temporal dynamics model of emotional memory processing: A synthesis on the neurobiological basis of stress-induced amnesia, flashbulb and traumatic memories, and the Yerkes-Dodson Law. *Neural Plasticity*, *2007*(60803). doi:10.1155/2007/60803. Web. 14 June 2016. (Published Online).

Engels, A. S., Heller, W., Mohanty, A., Herrington, J. D., Banich, M. T., Webb, A. G., & Miller, G. A. (2007). Specificity of regional brain activity in anxiety types during emotion processing. *Psychophysiology, 44*, 352–363.

Epel, E. S., Blackburn, E. H., Lin, J., Dhabhar, F. S., Adler, N. E., Morrow, J. D., & Cawthon, R. M. (2004). Accelerated telomere shortening in response to life stress. *PNAS, 101*(49), 17312–17315.

Fan, J., Xu, P., Van Dam, N., Eilamstock, T., Gu, X., Luo, Y., & Hof, P. (2012). Spontaneous brain activity relates to autonomic arousal. *The Journal of Neuroscience, 32*(33), 11176–11186.

Freeman, W. J., Ahlfors, S. P., & Menon, V. (2009). Combining fMRI with EEG and MEG in order to relate patterns of brain activity to cognition. *International Journal of Psychophysiology, 73*(1), 43–52.

Gilbert, M. E., & Zoeller, R. T. (2010). Thyroid hormones—impact on the developing brain: Possible mechanisms of neurotoxicity. In G. Jean Harry & Hugh A. Tilson (Eds.), *Neurotoxicology* (pp. 79–112). New York: Informa Healthcare.

Hammond, D., Walker, J., Hoffman, D., Lubar, J., Trudeau, D., Gurnee, R., & Horvat, J. (2004). Standards for the use of quantitative electroencephalography (QEEG) in neurofeedback: A position paper of the international society for neuronal regulation. *Journal of Neurotherapy, 8*(1), 5–27.

Heller, W., Nitschke, J. B., Etienne, M. A., & Miller, G. A. (1997). Patterns or regional brain activity differentiate types of anxiety. *Journal of Abnormal Psychology, 106*(3), 376–385.

Honey, C. J., Kotter, R., Breakspear, M., & Sporns, O. (2007). Network structure of cerebral cortex shapes functional connectivity on multiple time scales. *Proceedings of the National Academy of Sciences of USA, 104*, 10240–10245.

Horvat, J. (2009). Coherence and the quirks of coherence/phase training: A clinical perspective. In J. R. Evans (Ed.), *Handbook of neurofeedback* (pp. 213–227). New York: Informa.

John, E. R., Prichep, L. S., Fridman, J., & Easton, P. (1988). Neurometrics: Computer assisted differential diagnosis of brain dysfunctions. *Science, 293*, 162–169.

Kilner, J. M., Mattout, J., Henson, R., & Friston, K. J. (2005). Hemodynamic correlates of EEG: A heuristic. *NeuroImage, 28*, 280–286.

Kirk, I. J., & MacKay, J. C. (2003). The role of theta-range oscillations in synchronizing and integrating activity in distributed mnemonic networks. *Cortex, 39*, 993–1008.

Laird, A. R., Fox, P. M., Eickhoff, S. B., Turner, J. A., Ray, K. L., McKay, D. R., Glahn, D. C., . . . Fox, P. T. (2012). Behavioral interpretations of intrinsic connectivity networks. *Journal of Cognitive Neuroscience, 23*(12), 1–16.

Lubar, J. F., & Lubar, Judith. (1999). Neurofeedback assessment and treatment for attention deficit/hyperactivity disorders. In James R. Evans & Andrew Abarbanel (Eds.), *Introduction to quantitative EEG and neurofeedback* (pp. 243–310). New York: Academic Press.

McIntosh, A. R., & Korostil, M. (2008). Interpretation of neuroimaging data based on network concepts. *Brain Imaging and Behavior, 2*, 264–269.

Meehan, T. P., & Bressler, S. L. (2012). Neurocognitive networks: Findings, models, and theory. *Neuroscience and Biobehavioral Reviews, 36*(10), 2232–2247.

Monastra, V. J. (2005). Electroencephalographic biofeedback (neurotherapy) as a treatment for attention deficit hyperactivity disorder: Rationale and empirical foundation. *Child Adolescent Psychiatric Clinics of North America, 14*, 55–82.

Montgomery, D. D., Robb, J., Dwyer, K. V., & Gontkovsky, S. T. (1998, Spring). Single channel QEEG amplitudes in a bright, normal young adult sample. *Journal of Neurotherapy, 2*(4), 1–7.

Morillas-Romero, A., Tortella-Feliu, M., Bornas, X., & Aguayo-Siquier, B. (2013). Resting parietal electroencephalogram asymmetries and self-reported attentional control. *Clinical EEG and Neuroscience, 44*(3), 188–192.

Nakagawa, T., Jirsa, V., Spieglar, A., McIntosh, A., & Deco, G. (2013). Bottom up modeling of the connectome: Linking structure and function in the resting brain and their changes in aging. *NeuroImage, 80*, 318–329.
Niedermeyer, E., & Lopes da Silva, F. H. (2005). *Electroencephalography: Basic principles, clinical applications, and related fields*. New York: Lippincott Williams & Wilkins.
Nunez, P. L., & Srinivasan, R. (2006). *Electric fields of the brain: The neurophysics of EEG* (2nd ed.). New York: Oxford University Press.
Othmer, S., Othmer, S. F., & Kaiser, D. A. (1999). EEG biofeedback: An emerging model for its global efficacy. In James R. Evans & Andrew Abarbanel (Eds.), *Introduction to quantitative EEG and neurofeedback* (pp. 243–310). New York: Academic Press.
Pascual-Leone, A., Amedi, A., Fregni, F., & Merabet, L. B. (2005). The plastic human brain cortex. *Annual Review of Neuroscience, 28*, 377–401.
Pop-Jordanova, N., & Pop-Jordanov, J. (2005). Spectrum-weighted EEG frequency ("brain rate") as a quantitative indicator of mental arousal. *Contributions, Macedonian Academy of Sciences and Arts, Section of Biological and Medical Sciences, 26*(2), 35–42.
Raichle, M. E., & Gusnard, D. A. (2002). Appraising the brain's energy budget. *PNAS, 99*(16), 10239.
Rossiter, T. (2004). The effectiveness of neurofeedback and stimulant drugs in treating AD/HD: Part II. Replication. *Applied Psychophysiology and Biofeedback, 29*(4), 233–243.
Sapolsky, R. M. (1999). Glucocorticoids, stress, and their adverse neurological effects: Relevance to aging. *Experimental Gerontology, 34*(6), 721–732.
Schore, A. N. (1994). *Affect regulation and the origin of the self: The neurobiology of emotional development*. Hillsdale, NJ: Lawrence Erlbaum Associates.
Schwartz, M., & Andrasik, F. (Eds.) (2003). *Biofeedback: A practitioners guide* (3rd ed.). New York: Guildford Press.
Soutar, R. (1999). *Doing neurofeedback: A workshop manual*. Roswell, GA: New Mind Publications.
Soutar, R., & Longo, R. (2011). *Doing neurofeedback: An introduction*. SanRafael, CA: ISNR Research Foundation.
Sterman, M. B. (2000). Basic concepts and clinical findings in the treatment of seizure disorders with EEG operant conditioning. *Clinical Electroencephalography, 31*(1), 45–55.
Sterman, M. B. (1996). Physiological origins and functional correlates of EEG rhythmic activities: Implications for self-regulation. *Biofeedback and Self–Regulation, 21*, 3–33.
Sterman, M. B., & Egner, T. (2006). Foundation and practice of neurofeedback for the treatment of epilepsy. *Applied Psychophysiology and Biofeedback, 31*(1).
Teipal, S. J., Pogarell, O., Meindl, T., Dietrich, O., Sydykova, D., Hunklinger, U., Georgii, B., et al. (2009). Regional networks underlying interhemispheric connectivity: An EEG and DTI study in healthy ageing and amnestic mild cognitive impairment. *Human Brainmapping, 30*, 2098–2119.
Tomasia, D., Wang, G., & Volkowa, N. D. (2013). Energetic cost of brain functional connectivity. *PNAS, 110*(33), 13642–13647.
Trentin, A. G. (2006). Thyroid hormone and astrocyte morphogenesis. *Journal of Endocrinology, 189*, 189–197.
Zubin, J., & Spring, B. (1977). Vulnerability: A new view of schizophrenia. *Journal of Abnormal Psychology, 86*, 103–126.