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### EEG Spectral-Power and Coherence: LORETA Neurofeedback Training in the Anterior Cingulate Gyrus

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## EEG Spectral-Power and Coherence: LORETA Neurofeedback Training in the Anterior Cingulate Gyrus

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**ABSTRACT.** *Introduction.* This study examines the EEG spectral power and coherence changes that occur as a result of LORETA neurofeedback (LNFB) training, which is a recently developed spatial-specific neurofeedback protocol in which it has been demonstrated that human beings can learn to change activity in their own anterior cingulate gyrus. We trained individuals to increase low-beta (14-18 Hz) activity in the cognitive division of the anterior cingulate gyrus (ACcd).

*Methods.* This study was conducted with eight non-clinical students with a mean age of 22. The participants completed over 30 sessions of LNFB training. We utilized the WAIS-III for pre- and post-psychometric measures to assess the influence of this training protocol.

*Results.* We selected training Sessions 5, 10, 15, 20, 25, and 30 for comparison to Session 1. There are significant increases in absolute power and coherence over sessions. There is significant increase in the working memory and processing speed subtest scores.

*Discussion.* The anterior regions of the cortex increase in the low-beta frequency relative to the ACcd at significant levels. The superior prefrontal cortex and occipital regions increase in the higher beta frequencies, but not in the trained frequency. The improvements in the working memory and processing speed scores suggest that LNFB had an overall positive effect in attentional processes, working memory, and processing speed. doi:10.1300/J184v10n01\_02

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**KEYWORDS.** Neurofeedback, LORETA, anterior cingulate gyrus, electroencephalograph, biofeedback, spectral power and coherence, coherence, cognitive enhancement, attention

### INTRODUCTION

The anterior cingulate gyrus (AC) is a subject of intense interest to researchers, due in part to its location in the brain and its involvement in executive functions and reported activation in tasks involving attention, cognition, motivation, personality and numerous psychological processes. However, attentional processes are probably the most investigated function of the AC (Devinsky, Morrell, & Vogt, 1995; Pardo, Pardo, Janer, & Raichle, 1990; Bench et al., 1993; Posner & Petersen, 1990). The AC contains both cognitive and affective areas. The cognitive division consists of areas 24 and 32, while the affective division consists of areas 25, 33 and rostral area 24. This affective region is reported to be involved with regulating endocrine and autonomic functions as well as conditioned emotional learning, vocal communication of internal states and emotional valence concerning external and internal stimuli (Devinsky et al., 1995). The AC sends afferents to the amygdala and thalamo-cortical structures that possibly influence internal states associated with emotions (Devinsky et al., 1995; Carlson, 2001). A recent study reports activation of the affective division of the AC and the right inferior frontal, uncus and hippocampal formation during a self-generated anger memory recall experiment (Cannon et al., 2005). In this respect, it is considered part of the rostral limbic system, which includes the ventral striatum, the amygdala, periaqueductal grey, anterior insular cortex and the dorsolateral prefrontal and orbitofrontal cortices. Seizure disorders involving the anterior cingulate are reported to result in altered levels of attention and consciousness (Devinsky et al., 1995).

One prominent theory proposes that the AC detects the need for executive control and signals the prefrontal cortex (PFC) to execute the control (Markela-Lerenc et al., 2004). Executive functions (EF) are suggested to be an enveloping process that includes all the cognitive processes associated with goal completion, anticipation, goal selection, planning, and initiation of activity, self-regulation and monitoring

and use of feedback (Sohlberg & Mateer, 1989). This suggests EF are not only instrumental in cognitive processes but also crucial in attentional effort and maintenance. The dorsolateral prefrontal cortex and in particular the AC are suggested to be instrumental in executive functions and are established to be active during short-term memory tasks and auditory, visual and somesthetic information processing and to be involved in cognitive processes (Roland, 1984; Rubia, Smith, Brammer, & Taylor, 2003). The prefrontal cortex and AC are known to play a specific role in executive functions, and are physiologically connected and commonly activated in functional imaging studies. Moreover, lesion studies report damage to regions in the dorsolateral prefrontal cortex (DLPFC) lead to deficits in planning, reasoning, and problem solving (Heyder, Suchan, & Daum, 2004).

Studies involving sustained attention report frequent engagement of the right hemisphere in frontal and parietal regions and suggest a fronto-parietal thalamic network to be associated with sustained attention. Moreover, the prefrontal cortex and AC are suggested to be associated with response selection (Coull, Frackowiak, & Frith, 1998; Stathopoulou & Lubar, 2004; Pardo, Fox, & Raichle, 1991). Activation of the prefrontal cortex, AC, bilateral parietal cortex and occipital areas is reported in functional magnetic resonance imaging (fMRI) experiments involving sustained attention and counting (Ortuño et al., 2001). Studies report significant activation of the supplementary motor area (SMA) during attentional tasks and suggest that the SMA, dorsolateral PFC, inferior parietal lobes and the AC would be related to attentional effort as a general factor (Carr, 1992).

EEG coherence is suggested to reflect a number of synaptic connections between electrode sites and the magnitude of these connections (Thatcher, Krause, & Hrybyk, 1986; Thatcher, McAlaster, Lester, Horst, & Cantor, 1983; Nunez, Wingeier, & Silberstein, 2001). EEG measurements are regularly made using multiple electrodes on the scalp, using such

methods as the autocorrelation, mutual correlation functions, spectra, and mutual coherence functions as diagnostic indicators of structure in these signals (Nunez, 1995; Nunez et al., 1997; Nunez & Silberstein, 2000). These methods are frequently used to probe cognitive events and information processing (Nunez, 1995; Nunez et al., 1997; Nunez & Silberstein, 2000; Thatcher et al., 1986). Hence, it appears that linear coherence and correlation analyses capture the majority of interactions between neuronal populations as measured by scalp EEG. High coherence values between two or more EEG signals suggest synchrony among neuronal populations; however, these values do not define the source of the interactions (Knyazeva & Innocenti, 2001; Towle et al., 1993). Alternatively, low-coherence values suggest that neuronal populations are operating more independently. Increased amplitude and differentiation of neuronal populations are suggested to be correlated with intelligence (Thatcher, North, & Biver, 2005). It is also reported that increased complexity and neural efficacy are positively correlated with intelligence measures (Thatcher et al., 2005). Quantitative EEG and coherence provide important information regarding cortico-cortical functioning during cognitive tasks (Hogan, Swanwick, Kaiser, Rowan, & Lawlor, 2003) and a quantitative measure of the association between pairs of electrodes as a function of frequency (Schack, Griesbach, & Krause, 1999). Studies regarding coherence changes during cognitive tasks have focused mainly on hemispheric asymmetries (Tucker, Roth, & Bajr, 1986). It is reported that EEG coherence reveals the degree of correlation between two or more signals and that coherence can reveal anatomical connections (Fein, Ruz, Brown, & Merrin, 1988).

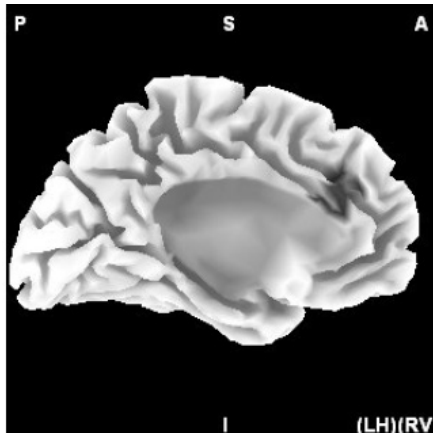
Neurofeedback techniques have been utilized in clinical and research settings for treatment of epilepsy (Serman, 2000), attentional disorders (Lubar, 1984, 1985, 1995a, 1995b; Lubar, Swartwood, Swartwood, & O'Donnell, 1995; Lubar & Lubar, 1999; Barnea, Rassis, & Zaidel, 2004; Barnea, Rassis, Raz, Othmer, & Zaidel, 2004) alcoholism and posttraumatic stress disorders (Peniston & Kulkosky, 1989, 1990, 1991) and are demonstrated to be effective in influencing and normalizing neurologi-

cal functioning in children and adults with attention deficit disorder (ADD) and attention deficit hyperactivity disorder (ADHD; Lubar, 1985, 1991, 1995a, 1995b; Lubar & Lubar, 1984; Lubar, Swartwood, Swartwood, & O'Donnell, 1995). Neurofeedback is an operant learning technique in which subjects learn to condition their own EEG using computer interfaces to allow the individual to become aware of and change their own brain's electrical activity. The participant is provided auditory or visual reward to initiate changes and achieve the desired effect (Thornton & Carmody, 2005). Studies report improvements in attention as a result of increasing low beta activity in healthy subjects during inhibition of sensorimotor processes (Egner, Zech, & Gruzelier, 2004). Changes in EEG power and coherence are reported in experiments involving complex mental problems (Jausovec & Jausovec, 1999), reading tasks (Efthymios & Lubar, 2002), working memory processes (Cairo, Liddle, Woodward, & Ngan, 2004) and visuomotor tasks (Aoki, Fetz, Shupe, Lettich, & Ojemann, 2001). Studies report successful interventions using neurofeedback techniques in treating reading disabilities and traumatic brain injury (Thornton & Carmody, 2005).

Following Congedo, Lubar, and Joffe (2004) we aimed at improving attentional processes using LORETA neurofeedback (LNFB; Pascual-Marqui, 1995, 1999; Pascual-Marqui, Michel, & Lehmann, 1994; Pascual-Marqui, 1999; Congedo, 2003; Congedo et al., 2004; Cannon et al., 2005), which is a recently developed, spatial-specific neurofeedback training protocol. We trained individuals to increase 14-18 Hz (low beta) power activity in a seven-voxel cluster defining the ACcd (cytoarchitectural regions 24b', 24c', 32'), within Brodmann Area (BA) 32 with center coordinates at  $X = -3$ ,  $Y = 31$ ,  $Z = 29$ . Figure 1 is a LORETA/MRI image showing this functional subdivision of the ACcd that is shown to be instrumental in executive functions and to play a crucial role in cognitive and affective processing; moreover, there is suspected involvement of frontostriatal network abnormalities in the etiology of ADHD. This definition of the region of interest (ROI) followed indications of Devinsky et al., 1995.

This study investigates the changes that occur in the EEG spectral power and coherence

FIGURE 1. It shows the region of interest of LNFB training. The image is of the left hemisphere. The red in the image shows (ROI) the target of seven voxels<sup>3</sup> with center coordinates at ( $x = -3$ ,  $y = 31$ ,  $z = 29$ ) Brodman Area 32, Cingulate Gyrus, Limbic lobe.



resulting from LNFB training within the ACcd. The efficacy of the training was assessed by means of pre/post training psychometric testing using subtests of the Wechsler Adult Intelligence Scale-Third Edition (WAIS-III, Wechsler, 1997). In a prior work, we extracted the activity in the ACcd and other identified generators of the surface EEG using LORETA and assessed the learning effect by statistical modeling (Cannon et al., 2005). The results indicate significant learning occurred in the ACcd and in cortical regions known to share afferent and efferent connections with the AC and are shown to be active with the AC in cognitive and attentional tasks. More specifically, learning occurred in specific clusters of neuronal populations in the bilateral dorsolateral prefrontal cortex and the right post central gyrus. The anterior regions appear to increase in activity in synchrony with or as a function of the ACcd, while many posterior regions decrease in the trained frequency but increase in higher beta activity.

Current research reporting the EEG spectral power and coherence changes resulting from neurofeedback training in normal subjects is sparse. However, studies report that neurofeedback training protocols that reward the SMR frequency improved perceptual sensitivity, reduced commission errors and improved

speed in sustained attention tasks, recall in semantic memory and accuracy in focal attention and musical tasks (Egner & Gruzelier, 2001, 2003). Studies show that the theta frequency is related to encoding and retrieval in short-term memory tasks, and the upper alpha frequency (10-12 Hz) is related to sensory processes involving semantic memory, while the lower alpha frequency (8-10 Hz) SMR are inferred to be related to attention (Vernon et al., 2003). Thornton and Carmody (2005) report significant improvement using neurofeedback techniques in coherence and magnitude interventions for traumatic brain injury. Our hypothesis proposes that LNFB training in the ACcd significantly influenced changes in both spectral absolute power and coherence in the EEG of these participants.

## METHOD

### Participants

This study was conducted with eight participants, four male and four female non-clinical students at the University of Tennessee, Knoxville. The mean age was 22, range 20 - 26, SD = 1.92. Seven of the participants were right handed and one was ambidextrous. All participants read and signed an informed consent to protocol approved by the University of Tennessee Institutional Review Board. All received extra course credit for participating in this study. Exclusionary criteria for participation included previous head trauma, history of seizures, drug or alcohol use and any previous psychiatric diagnosis.

### Procedures

Participants were prepared for EEG recording using a measure of the distance between the nasion and inion to determine the appropriate cap size for recording (Electrocap, Inc.; Blom & Anneveldt, 1982). The head was measured and marked prior to each session to maintain consistency. The ears and forehead were cleaned for recording with NuPrep, a mild abrasive gel used to remove any oil and dirt from the skin. After fitting the caps, each electrode site was injected with electrogel and prepared so that im-

pedances between individual electrodes and each ear were  $< 6 \text{ K}\Omega$ . The LNFB training was conducted using the 19-leads standard international 10/20 system (FP1, FP2, F3, F4, Fz, F7, F8, C3, C4, Cz, T3, T4, T5, T6, P3, P4, Pz, O1 and O2) with the Deymed Truscan EEG acquisition system using traditional EEG frequencies. The data were collected and stored with a band pass set at 0.5–64.0 Hz at a rate of 256 samples per second. Thirty-three training sessions were conducted three times per week in a comfortably lit, sound attenuated room in the Neuropsychology and Brain Research Laboratory at the University of Tennessee, Knoxville. Lighting and temperature were held constant for the duration of the experiment. Each session required approximately forty minutes to complete.

### ***Neurofeedback Protocol***

In a preliminary session, the participants were instructed to control tongue and eye movements, eye blinks, and muscle activity from forehead, neck, and jaws. This enabled the subjects to minimize the production of extra-cranial artifacts (EMG, EOG, etc.) during the sessions. In the same session, they were informed of the inhibitory and reward aspects of the training. Thresholds were then set and maintained for each participant. The thresholds were:

- a. Electrooculogram (EOG)  $< 15.0 \text{ (}\mu\text{V)}$ : Suppress
- b. Electromyogram (EMG)  $< 6.0 \text{ microvolts (}\mu\text{V)}$ : Suppress
- c. Region of Interest (ROI)  $> 5.0 \text{ microamperes (}\mu\text{A)}$ : Enhance

The participants were provided visual and auditory feedback and points were achieved when they were able to simultaneously: (a) decrease 1–3 Hz activity in a linear combination of six frontal channels, FP1, FP2, F3, F4, F7, F8, and (b) decrease 35–55 Hz activity in a linear combination of six temporal and occipital channels, T3, T4, T5, T6, O1, and O2, while (c) increasing current source density (14–18 Hz) in the intra-cranial region of seven voxels<sup>3</sup> (ROI).

Maintaining the condition for .75 seconds achieved one point. The targets for inhibiting artifacts were selected based on current knowledge, such that the frontal channels are frequently contaminated by ocular movement, blinks and forehead movement and these often register in the lower frequencies. Similarly, the temporal and occipital sites were chosen due to the higher frequency contamination that occurs from jaw and teeth clenching, tongue movement and neck contractions. The auditory stimuli consisted of both positive and negative reinforcement, an unpleasant splat sound when the conditions were not met and a pleasant tone when they were. Similarly, the visual stimuli were activated when the criteria were being met (i.e., a car or a spaceship driving faster and straighter). Alternatively, a slower car, driving in the wrong lane or the spaceship flying slow and crooked occurred when the criteria were not being met. The score for meeting the criteria was also seen by the participants in a small window of the game screen.

### ***Data Collection***

Three-minute eyes-closed baselines were collected before and after training for analysis. Likewise, three-minute eyes-opened baseline recordings were collected before and after each session. The LNFB training involved four rounds, each four minutes in length. In addition, for each session, the participants in this study provided a written record of their experience, strategies, and mental processes employed to obtain points for each session during this training. The reports include attention to muscle and eye movement, the visual characteristics of the game, the pleasant tone and making the unpleasant splat stop, working memory, long and short-term memory, counting, mental verbalization (talking to the game, themselves or singing songs), thoughts of daily stresses, frustration relating to performance, sexual imagery and breathing and visualization techniques.

### ***Psychometric Pre-Training Measures***

We administered the WAIS-III for a pre-training measure. The mean Full Scale Index Score (FSIQ) is 124, range (118–139), SD = 6.79. We selected the Working Memory Index

(WMI) and Processing Speed Index (PSI) scores for post-training comparison. The mean pre-WMI score is 118, range 94-141, SD = 5.81. The mean pre-PSI score is 107, range 88-120, SD = 3.93. The WMI score consists of the sum of scaled scores in the Arithmetic (A), Digit Span (DS) and Letter-number sequencing (LN) subtests. The PSI score consists of the sum of scaled scores in Digit-Symbol Coding (CD) and Symbol Search (SS). We used these combinations of subtest scores following indications of Sattler (2001).

### **Data Analysis**

The obtained data were rigorously artifact rejected, with extra attention given to the frontal and temporal areas. All episodic eye blinks, eye movements, teeth clenching, jaw tension, body movements and possible EKG (electrocardiogram) were removed from the EEG stream. We then imported the artifacted EEG files into NeuroGuide (Applied Neuroscience) version 2.07 for group analysis. We compared Sessions 5, 10, 15, 20, 25, and 30 to Session 1. We also compared pre and post eyes-closed baselines. We conducted an ANOVA for the obtained pre and post psychometric scores controlling for subject as a random factor.

The statistical procedures in NeuroGuide utilize the following parameters. The EEG values are computed to z scores, the FFT parameters are 2s epochs at a sample rate of 128 samples per second or 256 digital time points at a frequency range from 0.5 to 40 Hz with a resolution of 0.5 Hz using a cosine taper window to minimize leakage. Each two second FFT consists of 81 rows (frequencies 0 to 40 Hz) by 19 columns (electrode locations) that equates to a 1,539 element cross-spectral matrix for each subject. To minimize the effects of windowing, the FFT (Serman & Kaiser, 2001) an EEG sliding average of the cross-spectral matrix is computed for each normal subject, editing the EEG by advancing in 64-point steps.

The FFT is recombined with the 64-point sliding window of 256 point FFT cross-spectrum for the entire EEG record. Each of the 81 frequencies for each 19 channels is  $\log_{10}$  transformed to better approximate a normal distribution. The total number of two-second windows is entered into the analysis of variance, t-test

and used to calculate the degrees of freedom for a given statistical test. The mean, variance, standard deviation, sum of squares, and squared sum of the real (cosine) and imaginary (sine) coefficients of the cross-spectral matrix are computed across the sliding average of edited EEG for all 19 leads for the total number of 81 and 1,539 log transformed elements for each subject. NeuroGuide utilizes independent t-tests, assuming population variances are not equal (Winer, 1962).

## **RESULTS**

### **Absolute Power Description of Figures**

Figures 2A through 2F show the results for the absolute power comparisons for Sessions 5, 10, 15, 20, 25, and 30 to session 1, respectively. The figures represent the probability of obtaining the t-value for the session comparisons. The colors indicate the significance level, the red in the graphs indicates probabilities toward zero while the blue indicates values equal to or greater than  $\alpha = .05$ . These figures show only the results for the training rounds across sessions. Figures 2G and 2H show the linear measures of absolute power increase across sessions; similarly, Figure 2I shows the increase of specific frequencies over sessions.

Figure 2A shows the results for absolute power differences between Session 5 and Session 1. The results indicate there are no significant differences in the delta (1-3 Hz) frequency. There are differences in the theta (4-8 Hz) frequency in the right frontal, right inferior temporal region and parietal areas. The alpha frequency (8-12 Hz) shows increases in the right parietal and central regions. A pattern begins developing in the 13 Hz and training frequency (14-18 Hz). This pattern involves FP1, FZ, F3, F4, CZ, PZ, in this session; however, in subsequent sessions includes FP2, P3, P4 and T6. There are moderate increases in the higher beta frequencies (19-32 Hz) in the left parieto-occipital and temporal regions.

Figure 2B shows the results for absolute power differences between session 10 and session 1. There is a moderate increase in the 3 Hz frequency in the left superior and dorsolateral

FIGURE 2A. The absolute power changes for the comparison between Session 5 and Session 1 are shown. The colors indicate the significance level, the red in the graphs indicate probabilities toward zero while the blue indicates values equal to or greater than  $\alpha = .05$ .

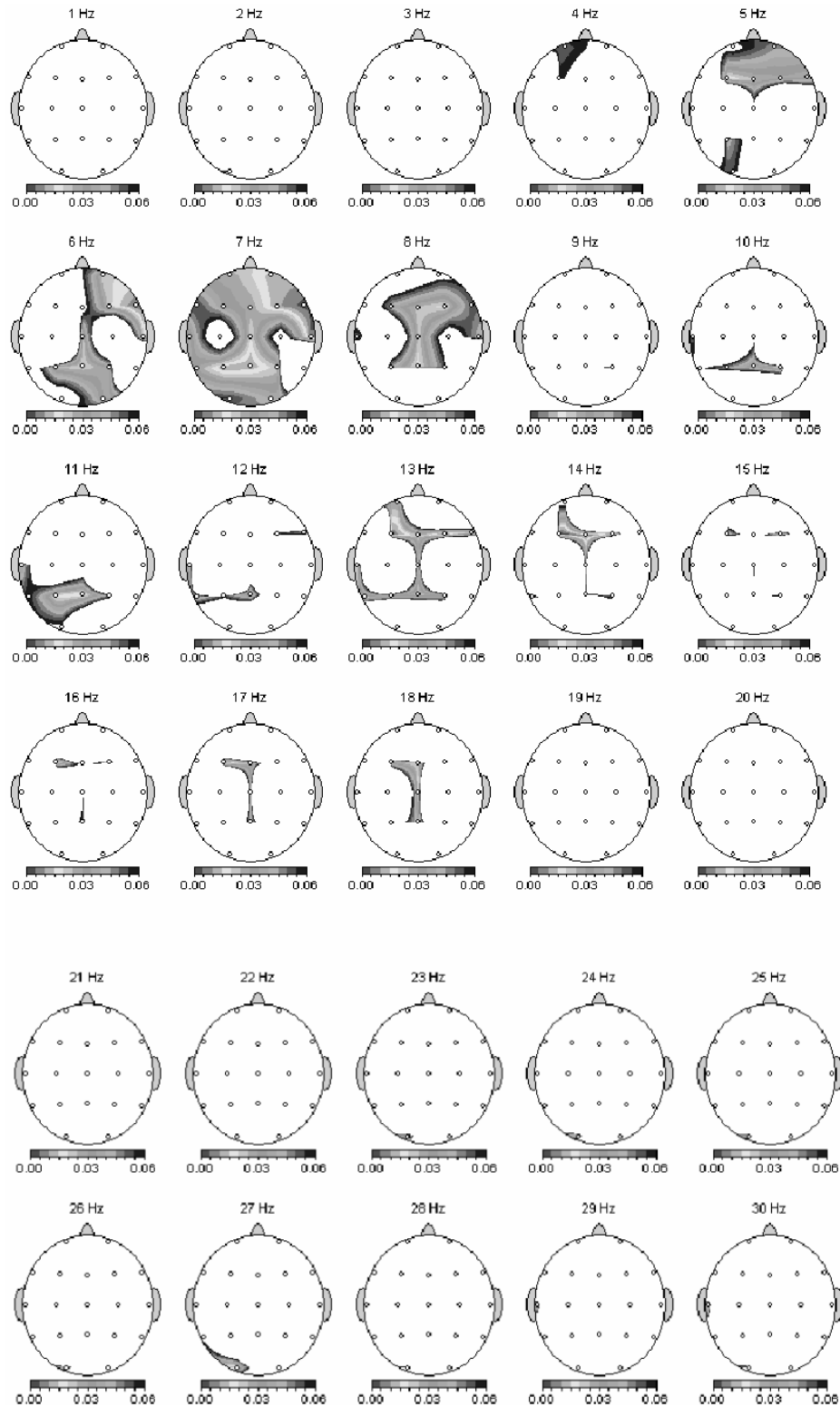




FIGURE 2B. The absolute power changes for the comparison between Session 10 and Session 1 are shown. The colors indicate the significance level, the red in the graphs indicate probabilities toward zero while the blue indicates values equal to or greater than  $\alpha = .05$ .

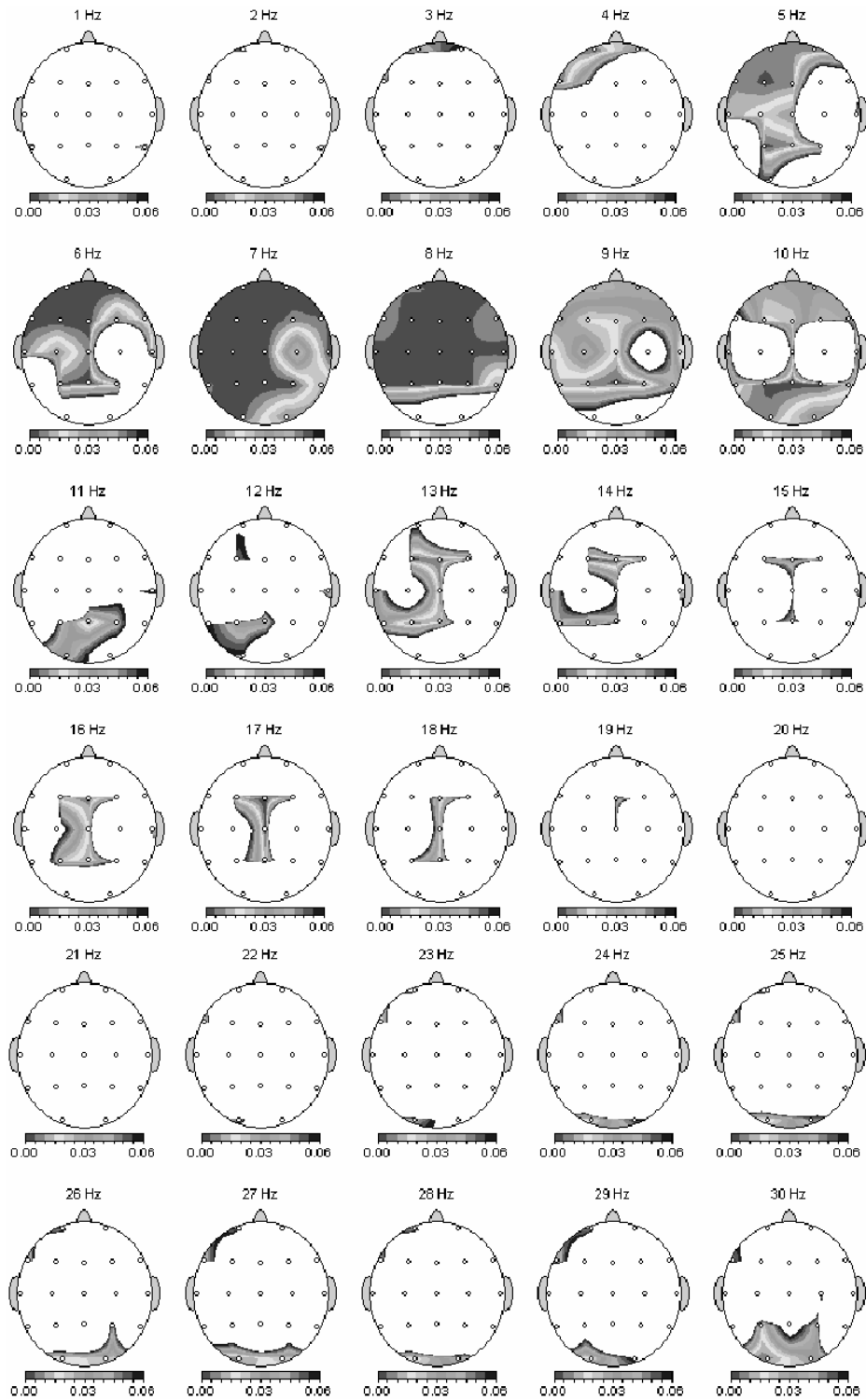


FIGURE 2C. The absolute power changes for the comparison between Session 15 and Session 1 are shown. The colors indicate the significance level, the red in the graphs indicate probabilities toward zero while the blue indicates values equal to or greater than  $\alpha = .05$ .

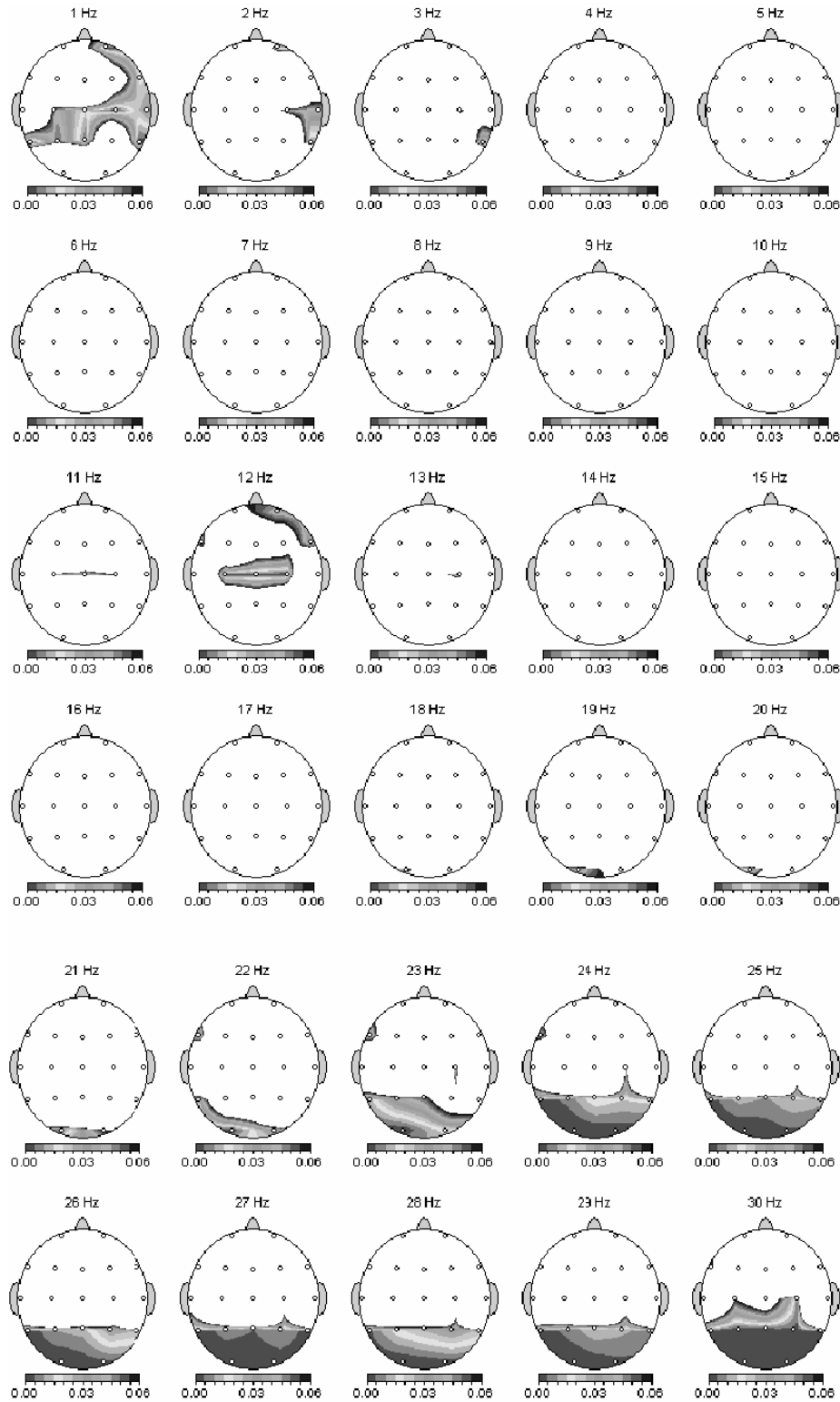


FIGURE 2D. The absolute power changes for the comparison between Session 20 and Session 1 are shown. The colors indicate the significance level, the red in the graphs indicate probabilities toward zero while the blue indicates values equal to or greater than  $\alpha = .05$ .

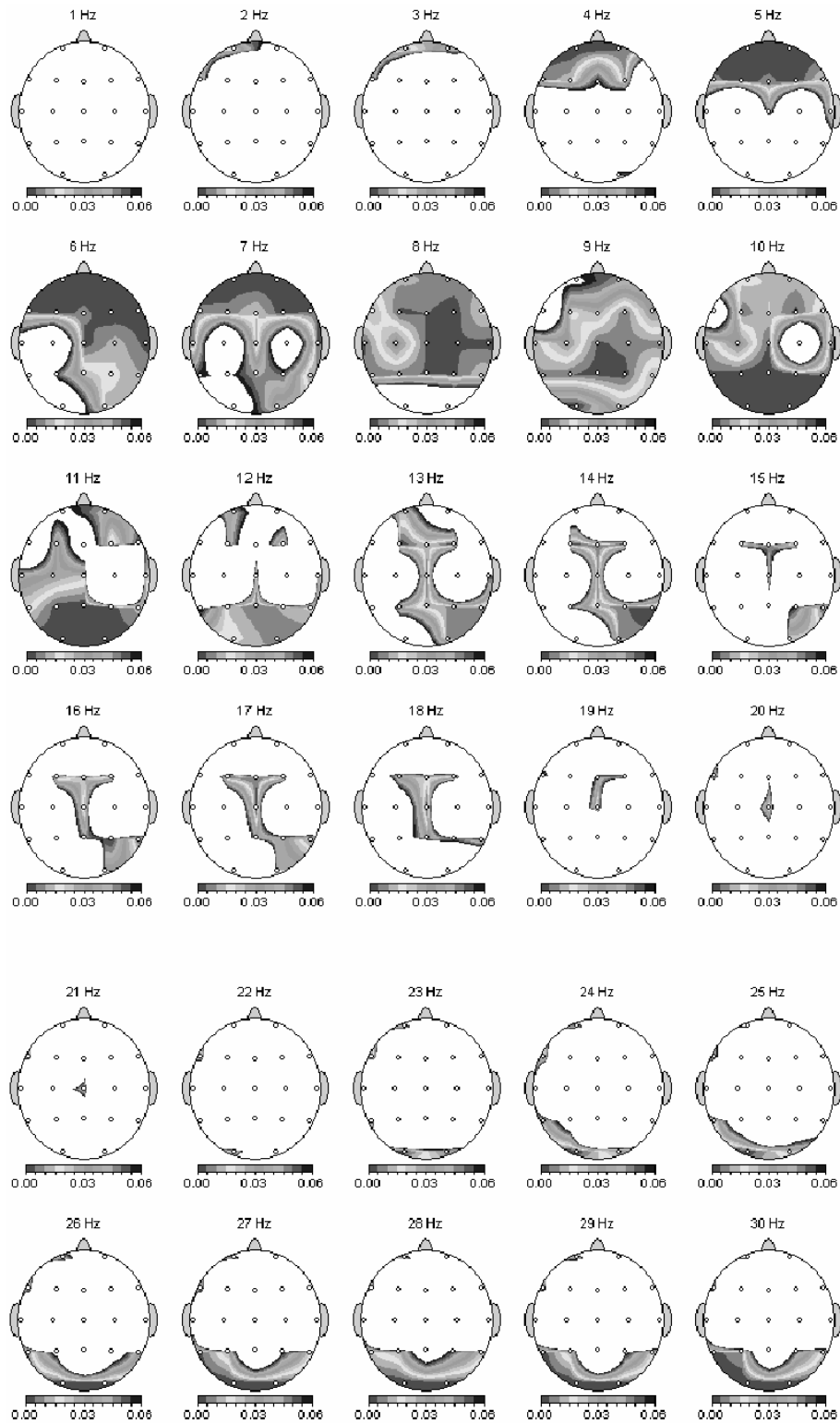


FIGURE 2E. The absolute power changes for the comparison between Session 25 and Session 1 are shown. The colors indicate the significance level, the red in the graphs indicate probabilities toward zero while the blue indicates values equal to or greater than  $\alpha = .05$ .

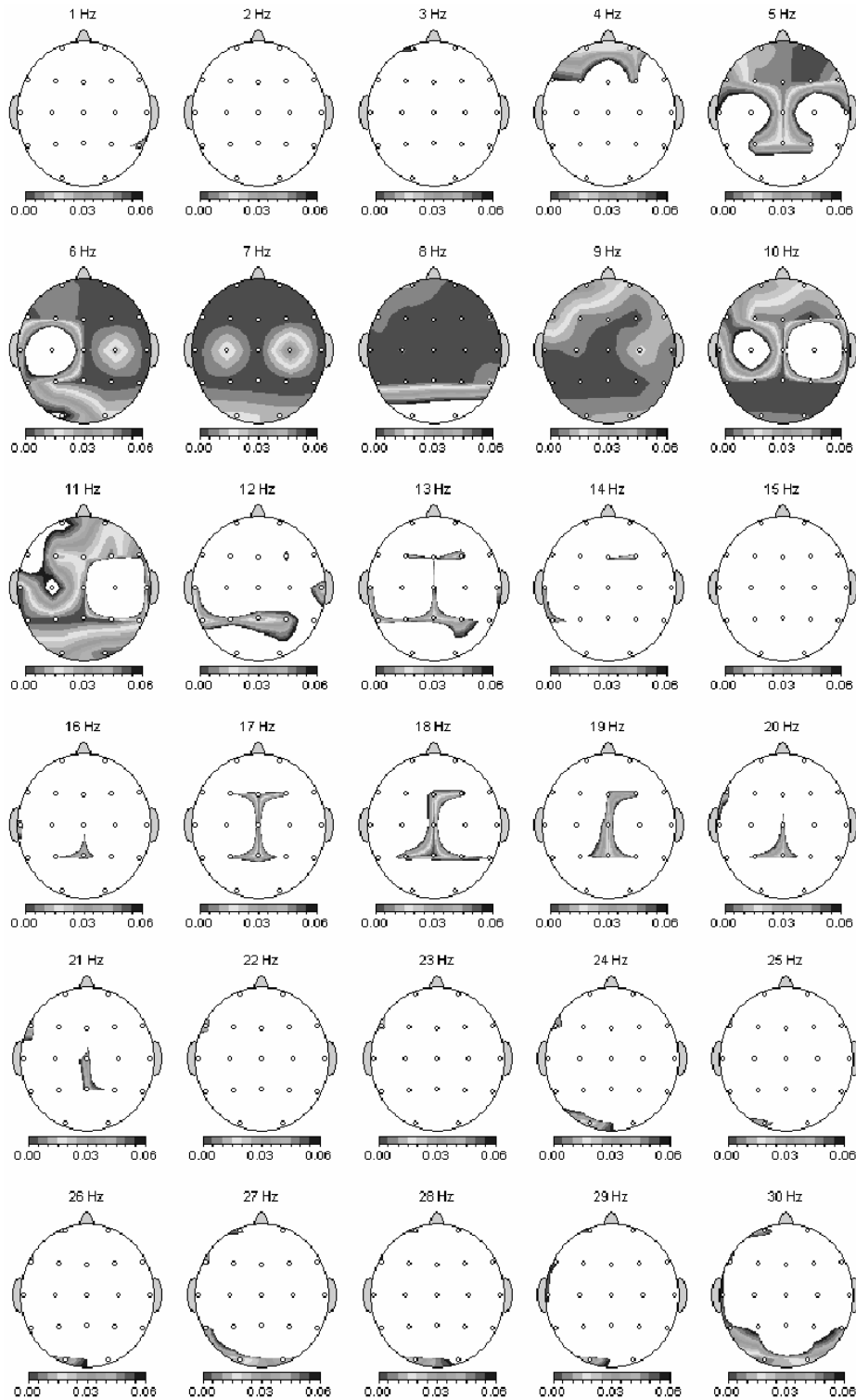


FIGURE 2F. The absolute power changes for the comparison between Session 30 and Session 1 are shown. The colors indicate the significance level, the red in the graphs indicate probabilities toward zero while the blue indicates values equal to or greater than  $\alpha = .05$ .

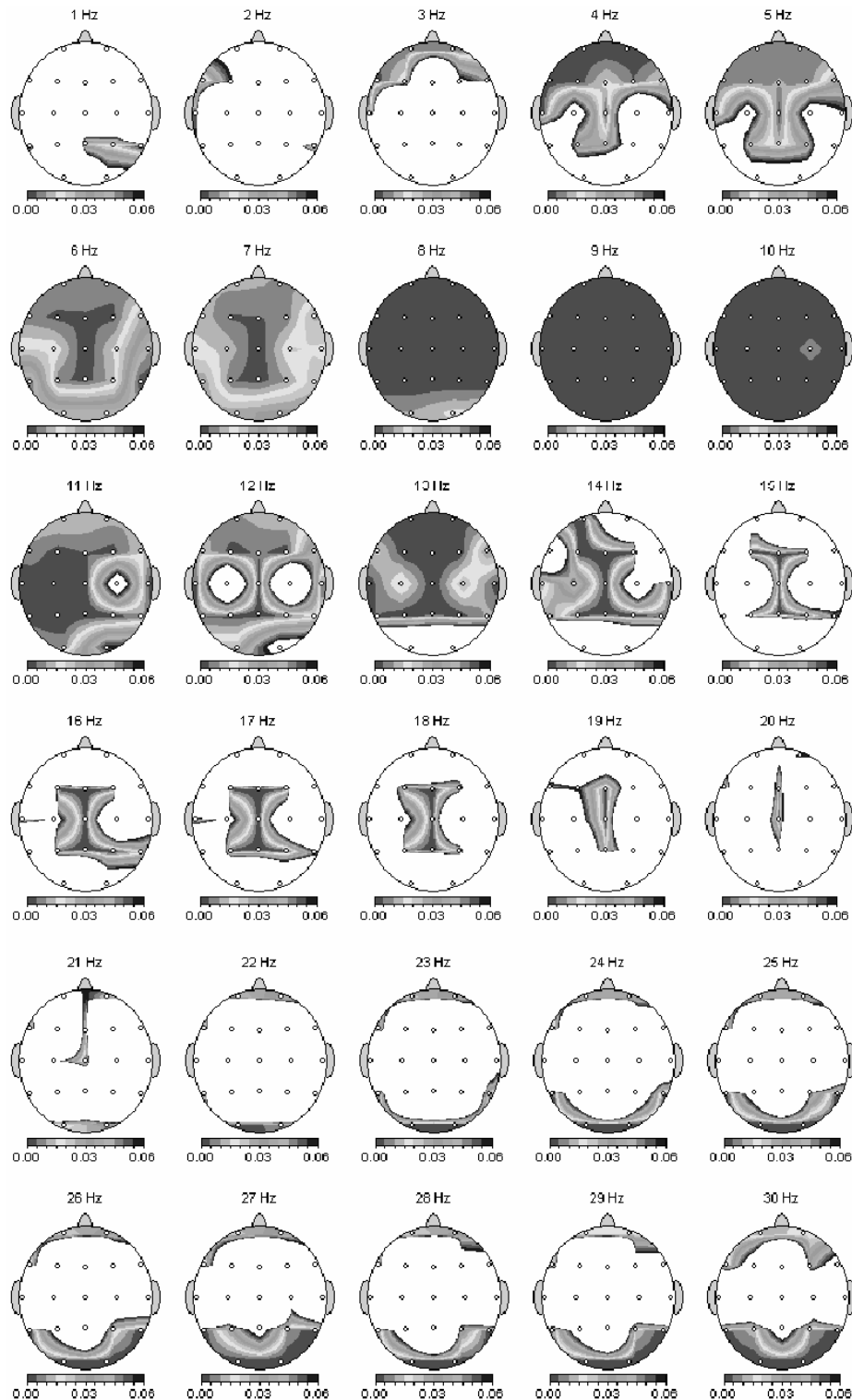


FIGURE 2G. The linear increase for the beta 1 (12-15 Hz) over Sessions 1, 5, 10, 15, 20, 25, and 30 is shown. There is a significant increase in this frequency over sessions as the topographical maps illustrate.

Absolute Power Increase (uVs<sup>2</sup>) for Beta 1 (12-15 Hz) at ACcd, Bilateral DLPFC and Centro-Parietal Regions Across Sessions

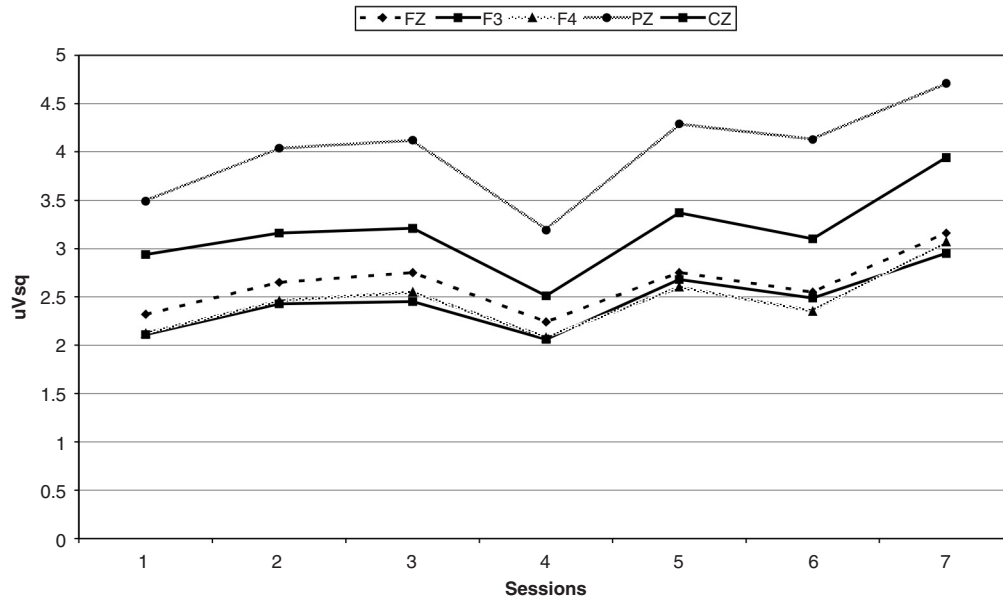


FIGURE 2H. The linear increase for the beta 2 (15-17.5 Hz) over Sessions 1, 5, 10, 15, 20, 25, and 30 is shown. There is a significant increase in this frequency over sessions as the topographical maps illustrate.

Absolute Power Increase Over Sessions Beta 2 (15-17.5 Hz) uVs<sup>2</sup> for ACcd, Frontal, Centro-Parietal Regions

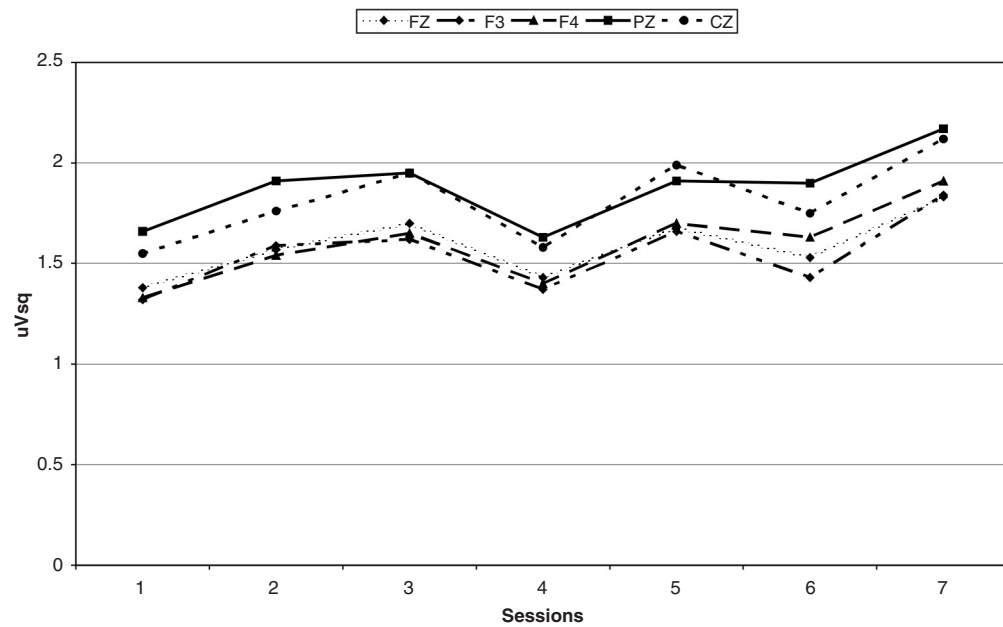
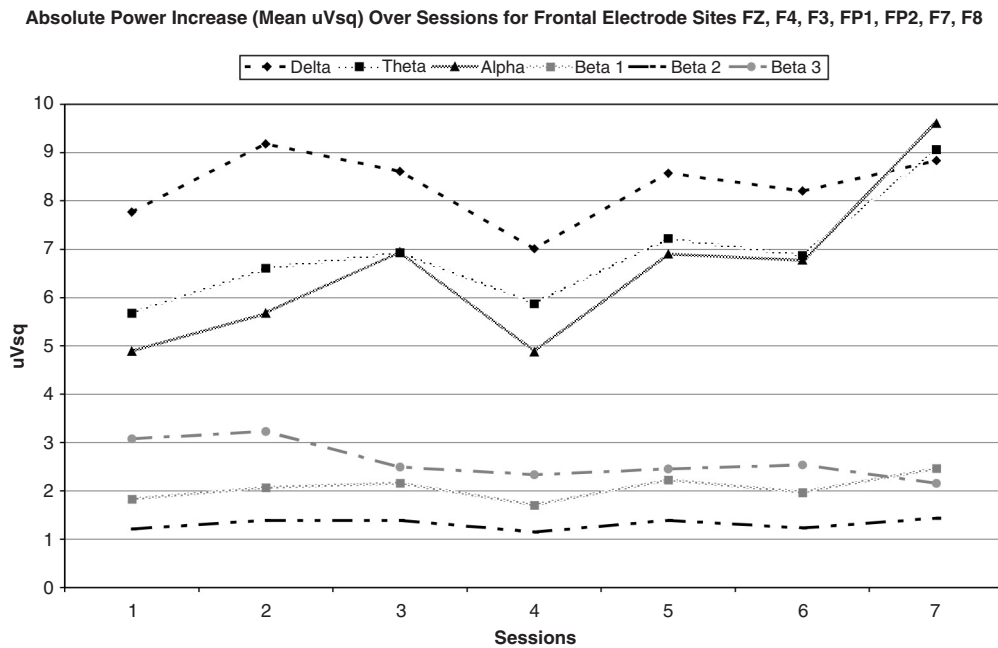


FIGURE 2I. The mean  $\mu\text{Vs}^2$  linear increase for the delta, theta, alpha, beta 1, beta 2 and beta 3 frequencies over sessions for frontal regions relative to the topographical maps is shown.



prefrontal region. The theta and alpha frequencies increase in the left frontal regions and become more pronounced in the frontal, temporal and parietal cortices. The pattern involving the trained frequency continues to develop in this session. The higher beta frequencies increase in the occipital, parietal and left dorsolateral prefrontal cortices.

Figure 2C shows the results for the absolute power differences between Session 15 and Session 1. There is significant increase in the delta frequency in the right frontal and bilateral parietal regions. The theta and alpha frequencies show no significant differences in this session. The trained frequency shows no difference in this session. There is, however, a significant increase in the higher beta frequencies in the occipital and parietal lobes.

Figure 2D shows the results for the absolute power differences between session 20 and session 1. There is significant increase in the delta (2 and 3 Hz) frequency in the left dorsolateral and superior prefrontal cortices. The theta and alpha frequencies increase in the occipital, parietal and frontal cortices. The 13 Hz pattern returns in this comparison, as does the trained frequency. The increases involve electrodes FP1,

F3, Fz, F4, CZ, PZ, P3, P4, T6, and O2. The higher beta frequencies increase moderately in the left frontal lobe and continue to increase at significant levels in the occipital and parietal lobes.

Figure 2E shows the absolute power differences between Session 25 and Session 1. There are no significant differences in the delta frequency. The theta and alpha frequencies in this session increase in frontal, parietal and occipital regions. The 13 Hz pattern returns in this comparison; however, it involves more of the left parietal and temporal regions than in previous sessions. The trained frequency appears less organized in this session, but does appear in the 17, 18, and 19 Hz beta frequencies. The higher beta frequencies continue to increase in the occipital regions in this session.

Figure 2F shows the absolute power differences between Session 30 and Session 1. There is an increase in the delta frequency in the superior frontal and right parietal cortices. The theta frequency increases in the frontal and central regions at significant levels and moderate levels in the occipital regions. The alpha frequency increases appear more global in this session. The 13 Hz frequency appears to engage the en-

tire frontal cortex in this session; meanwhile, the trained frequency shows the pattern more significantly than in previous sessions. It includes FP1, F3, F4, FZ, CZ, P3, P4, and T6. The higher beta frequencies increase in the occipital region and in the bilateral superior prefrontal cortices in this session.

Figure 2G (beta 1) and 2H (beta 2) show the linear increase in absolute power across sessions. These graphs show the average group mean microvolts squared (uVsq) levels for Sessions 1, 5, 10, 15, 20, 25, and 30 for electrodes FZ, F3, F4, PZ, and CZ. Figure 2I shows the increase for mean group uVsq increases over sessions in the same fashion.

### *Coherence Figure Descriptions*

NeuroGuide computes the coherence values for both interhemispheric and intrahemispheric electrode relationships. The output for the analysis consists of topographical maps for the probability of the obtained t value for the comparison. The blue indicates decreased coherence while the red indicates increased coherence. The size of the line dictates the probability of the obtained t value. The thinner line represents a  $p$  at less than or equal to .05. The middle size line indicates  $\alpha$  less than or equal to .025, and the larger line indicates values at a less than or equal to .010. In all figures, the interhemispheric results are the top four images from left to right, for the delta, theta, alpha, and beta frequencies. The next two rows from top to bottom and left to right show the intrahemispheric results in the same fashion. These figures show the results from the training rounds across sessions. Figures 3A through 3F show the coherence changes over sessions for the training rounds only.

Figure 3A shows the coherence changes between Session 5 and Session 1. In this comparison there is increased coherence in the delta frequency between FP1, FP2, P3, and P4. The coherence between electrodes in the right hemisphere show increase in the theta frequency in the superior frontal and right parietal lobe. Coherence in the alpha frequency increases in the left parietal and occipital region. The coherence decreases in the beta frequency in this comparison. The intrahemispheric co-

herence results show increases in delta, theta and alpha activity between hemispheres and between temporal, parietal, and frontal regions. The trained frequency increases in the superior prefrontal cortex. The higher beta frequencies decrease throughout the cortex.

Figure 3B shows the results for the coherence changes between Session 10 and Session 1. The coherence in the delta and theta frequencies increases throughout the frontal, central, right parietal, and occipital regions. The alpha frequency shows significant increase centrally and in frontal regions, while decreased coherence occurs in parietal and occipital regions. The beta frequency increases within the frontal regions, including the trained frequency, it also increases between central and parietal regions.

Figure 3C shows the coherence changes for Session 15 compared to Session 1. The interhemispheric changes show coherence increase in the delta frequency occurs between the left dorsolateral prefrontal, temporal, and occipital region, and in the right superior and dorsolateral prefrontal regions. The alpha frequency increases in fronto-temporal and parietal regions, while the beta frequency increases in the between central and left parieto-frontal and the left superior frontal to occipital regions. The intrahemispheric results show increased coherence between the left parietal and right frontal regions in the delta frequency. The theta frequency increases in the left parietal and occipital regions and right superior frontal region. The alpha and beta frequencies increase between the occipital, parietal and temporal regions and the superior and dorsolateral right prefrontal cortex. The trained frequency increases between central, occipital and prefrontal regions. There is also decreased coherence between hemispheres and between the right hemisphere and central regions.

Figure 3D shows the results for the comparison of Session 20 to Session 1. The interhemispheric results show coherence increases in the delta frequency between the right parieto-temporal areas and the left frontal, parietal and occipital regions; there is a contralateral increase between the temporal and parietal lobes. The theta frequency increases in coherence between hemispheres in the prefrontal and posterior regions; there is a significant coherence increase between FP1 and FP2 in the superior



frontal lobes and between O1 and O2 in the posterior occipital lobes. The alpha frequency shows no decrease in coherence but significant increases between occipital and frontal regions as well as between parietal, temporal and frontal areas. The beta frequency increases between left frontal and right parietal areas. For the intrahemispheric results, coherence in the delta frequency increases between occipital, right parietal, temporal and frontal regions, while there is a decrease between left frontal and right parietal regions. The theta coherence increases within the right hemisphere, and between the left prefrontal cortex and left central-parietal regions. The alpha frequency increases globally between hemispheres in both anterior and posterior regions. The trained low-beta frequency increases centrally and in the left prefrontal regions. The higher beta frequencies decrease in coherence between many locations, but continue to increase between the left frontal, temporal and parietal regions.

Figure 3E shows the results of the comparison between Session 25 and Session 1. The interhemispheric results show the delta frequency increases in coherence within the right hemisphere between frontal, temporal and occipital regions; there is also an increase between the left temporal-occipital and a contralateral coherence increase between the superior frontal and occipital regions. The theta frequency increases in the right hemisphere and between contralateral parieto-temporal regions. The alpha frequency increases between occipital and frontal regions, and within the right hemisphere. The beta frequency increases between the bilateral frontal and parietal regions, while decreasing in temporal regions. For the intrahemispheric results, the delta frequency coherence increases between the right frontal-temporal and parietal regions and between the left parieto-occipital regions. Similarly, the theta frequency increases within the right hemisphere but decreases between the left frontal to temporal and parietal cortices. The right hemisphere coherence increases within the alpha and beta frequencies between the frontal and central regions, especially in the trained frequency, but decreases between parietal and occipital regions.

Figure 3F shows results for the comparison of Session 30 to Session 1. The interhemis-

pheric results show an increase in all frequencies in this comparison. There is a decrease in coherence in the beta frequency from T5 to F3. The intrahemispheric results show an increase in all frequencies between the frontal, central, temporal and parietal regions favoring the right hemisphere. The high and middle beta frequencies decrease between the right to central left regions and in the parieto-occipital regions.

Figures 3G and 3H show the linear increase in coherence for the beta frequency (12-32 Hz) between selected frontal regions across Sessions 1, 5, 10, 15, 20, 25, and 30. Figure 3G shows the intrahemispheric increase across sessions between electrode pairs FP1-FP2, F3-F4, and O1-O2. Figure 3H shows the interhemispheric increase between electrode pairs across sessions for electrode pairs FP2-F8, FP2-F4, FP1-F3, and FP1-F7. These data are for training rounds only.

### *Pre- and Post-Coherence Changes*

Figure 4 shows the results for the pre and post eyes-closed baseline comparison. The first set of images shows the interhemispheric pairs from left to right. The coherence in the delta frequency increases between the dorsolateral prefrontal cortex and right parietal region. The theta frequency increases between the right superior to right parietal regions. The alpha frequency increases in the right post central gyrus region. The beta frequency increases in coherence contralaterally and within the inferior parietal lobe and right post central gyrus. In the intrahemispheric comparison, there is an increase between the bilateral DLPFC to the RPCG.

Similarly, the coherence in the theta frequency increases between the DLPFC, superior prefrontal cortex and in the right post-central gyrus, while the beta-frequency increases between the frontal regions and the ACCd. The beta 3 frequency is of particular interest in that it increases within the right hemisphere and contralaterally to the DLPFC and left anterior parietal regions. There is significant coherence increase between the right inferior temporal, right frontal, and right central and parietal regions.

FIGURE 3A. The significant coherence changes between Session 5 and Session 1 are shown. The top set of four images show the homologous pairs, while the remaining two rows of four images, from left to right show the intrahemispheric coherence changes.

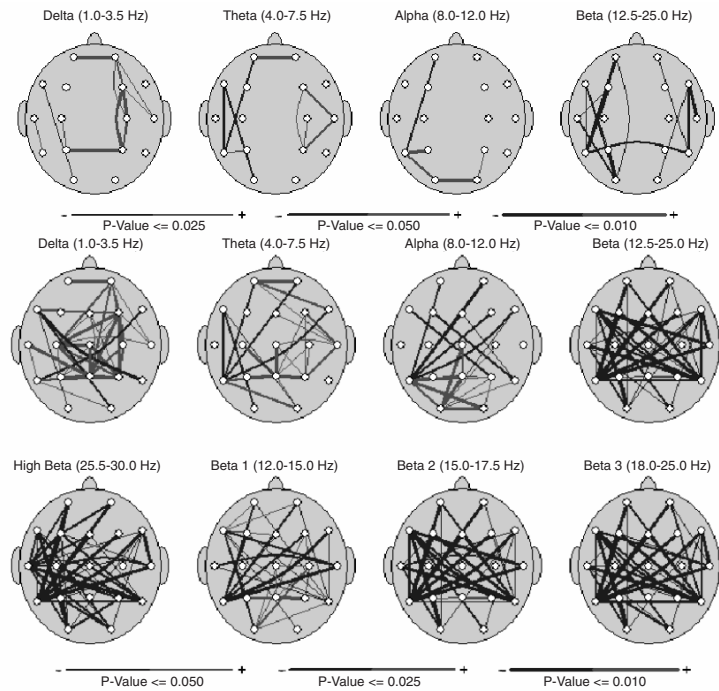


FIGURE 3B. The significant coherence changes between Session 10 and Session 1 are shown. The top set of four images show the homologous pairs, while the remaining two rows of four images, from left to right show the intrahemispheric coherence changes.

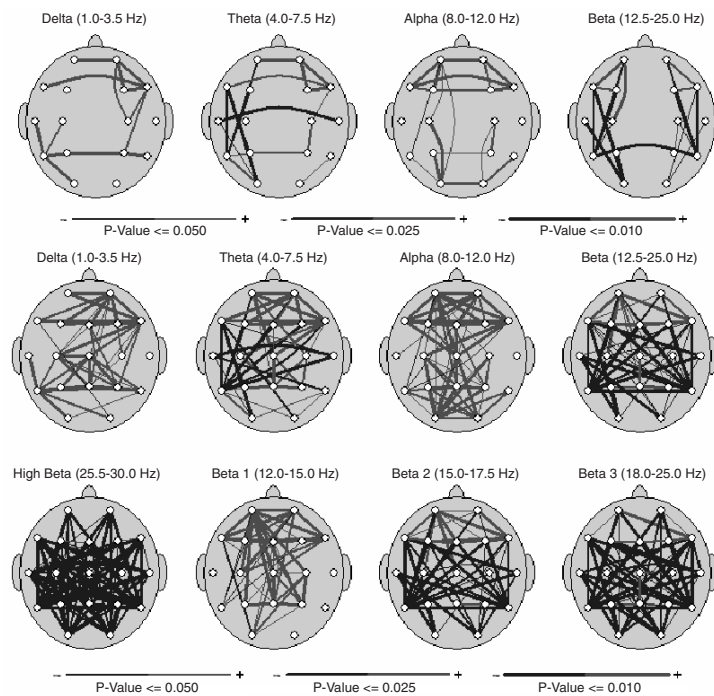


FIGURE 3C. The significant coherence changes between Session 15 and Session 1 are shown. The top set of four images show the homologous pairs, while the remaining two rows of four images, from left to right show the intrahemispheric coherence changes.

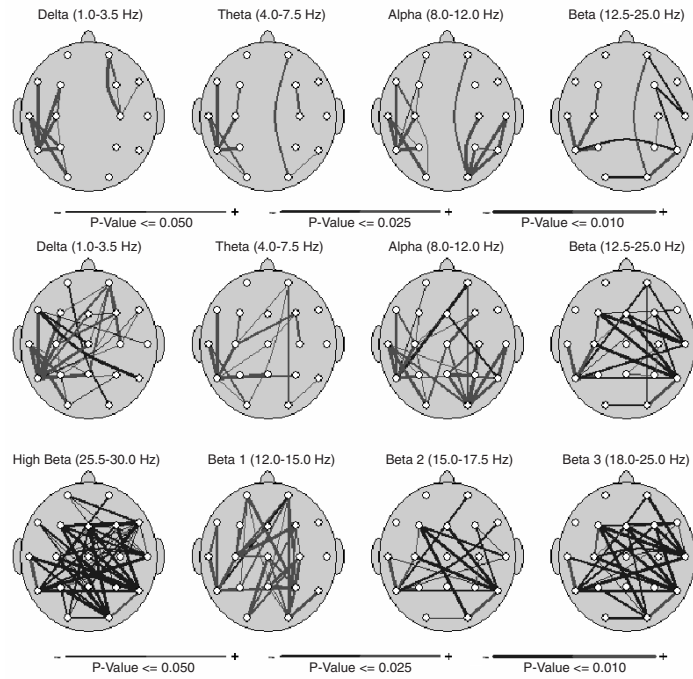


FIGURE 3D. The significant coherence changes between Session 20 and Session 1 are shown. The top set of four images show the homologous pairs, while the remaining two rows of four images, from left to right show the intrahemispheric coherence changes.

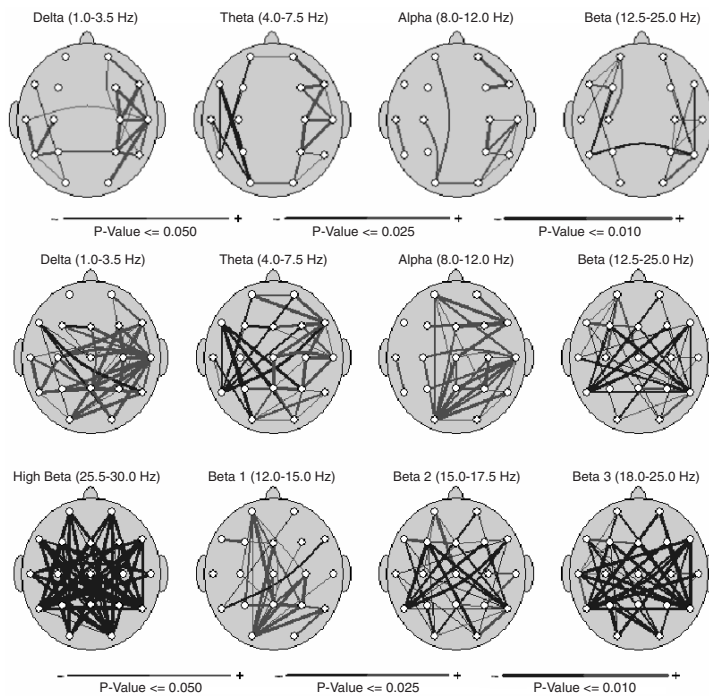


FIGURE 3E. The significant coherence changes between Session 25 and Session 1 are shown. The top set of four images show the homologous pairs, while the remaining two rows of four images, from left to right show the intrahemispheric coherence changes.

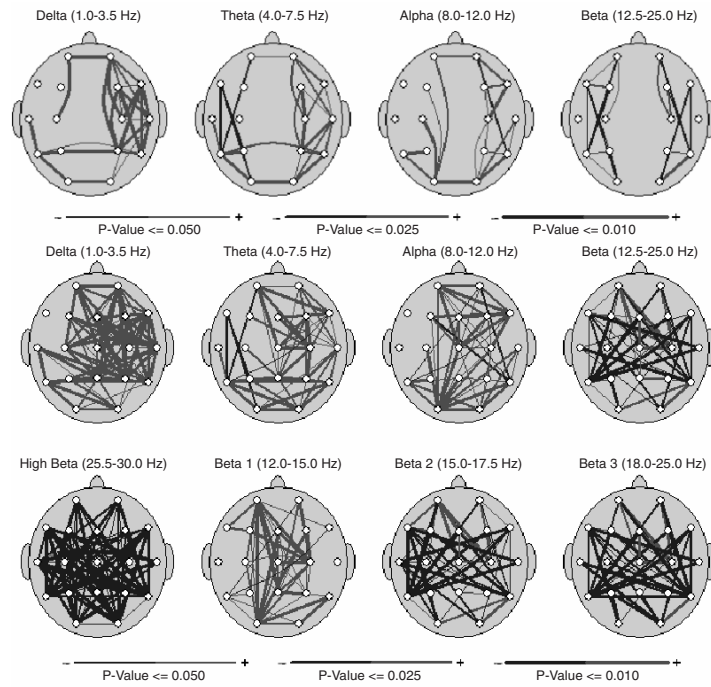


FIGURE 3F. The significant coherence changes between Session 30 and Session 1 are shown. The top set of four images show the homologous pairs, while the remaining two rows of four images, from left to right show the intrahemispheric coherence changes.

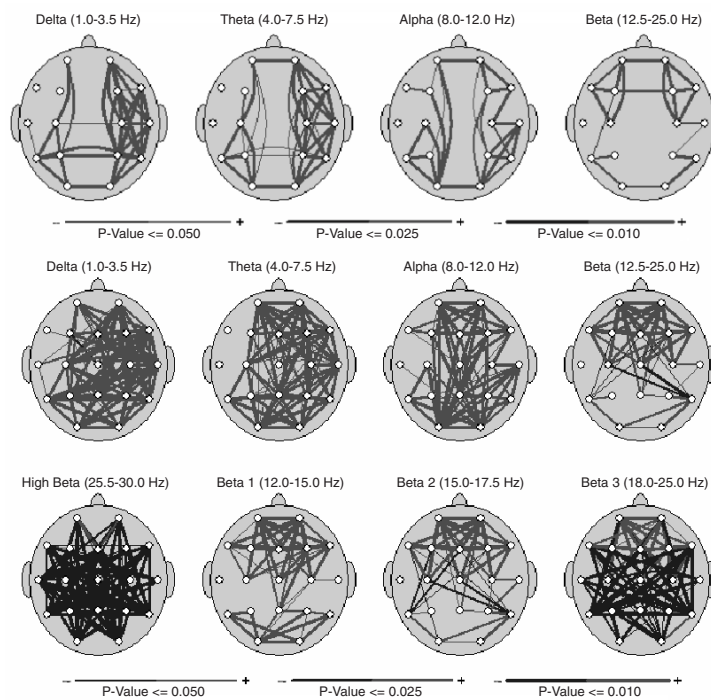


FIGURE 3G. The mean uVs<sub>q</sub> linear increase in coherence across sessions for the beta frequency between intrahemispheric electrode sites is shown.

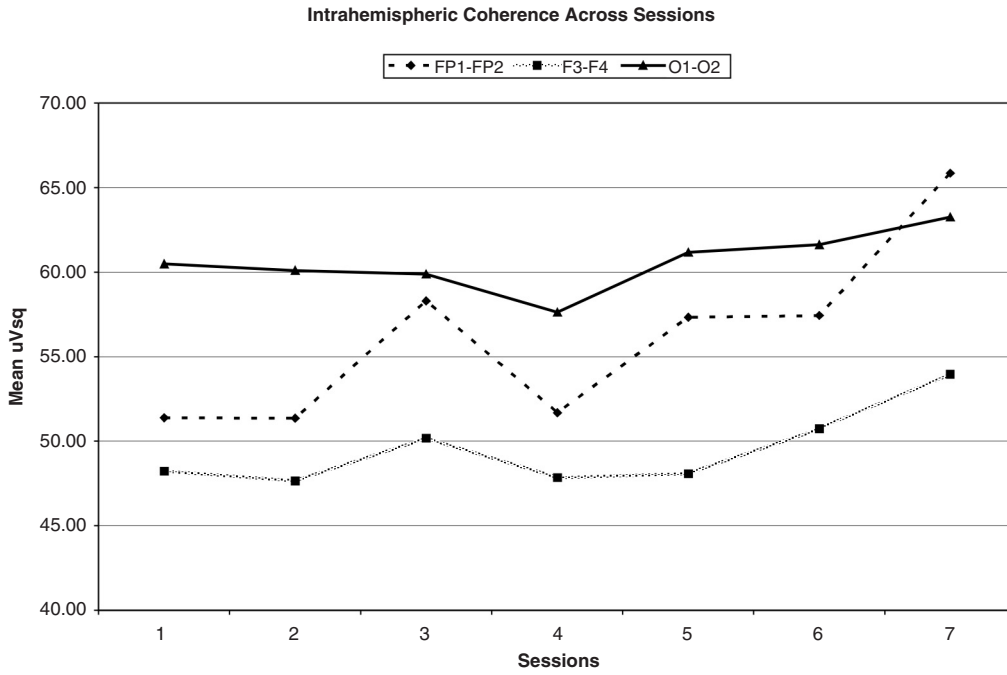


FIGURE 3H. The mean uVs<sub>q</sub> linear increase in coherence across sessions for the beta frequency between interhemispheric electrode sites is shown.

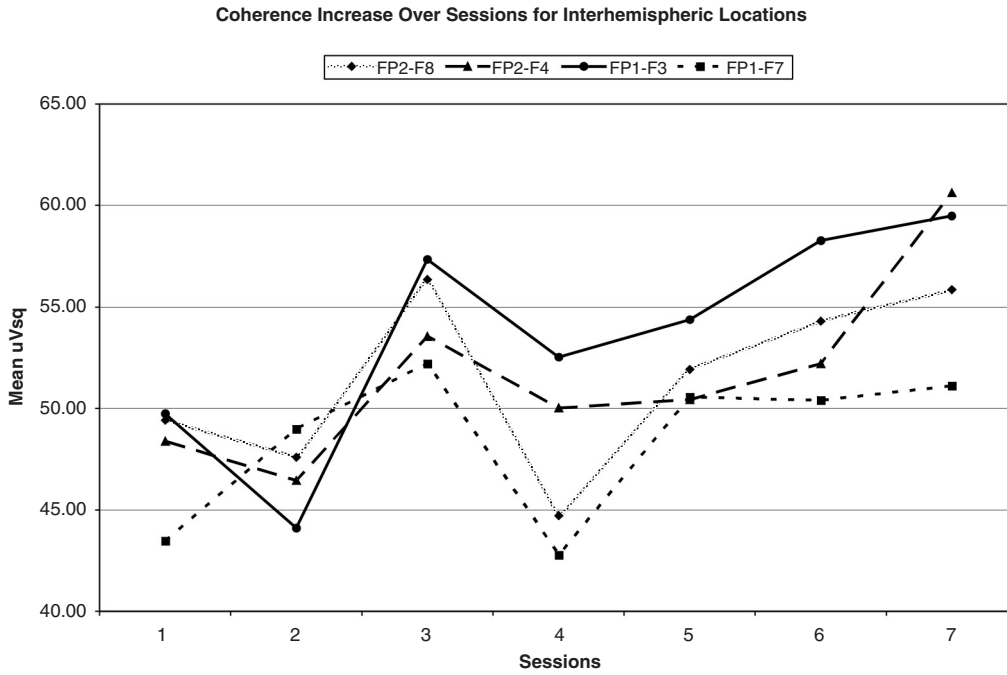
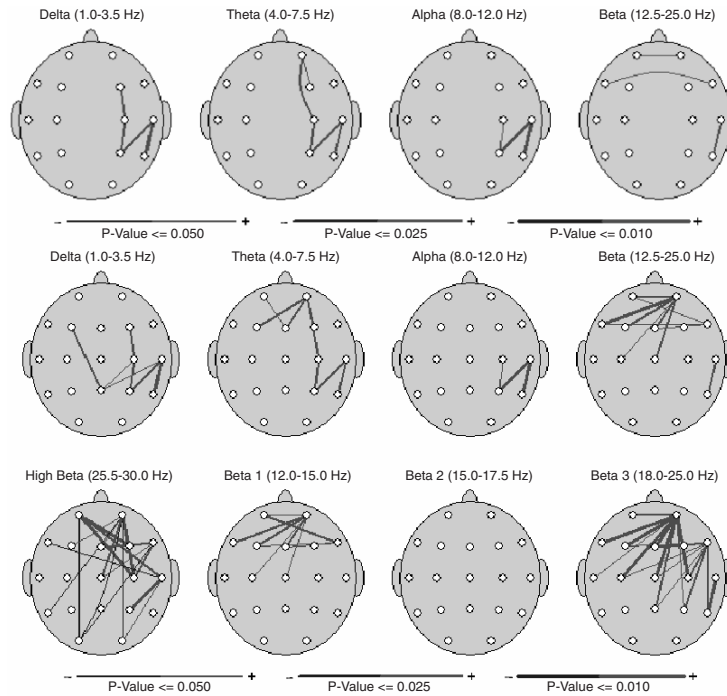


FIGURE 4. The results for the pre- and post-eyes-closed baseline comparison are shown. The output consists of topographical maps of the probability of the obtained t value for the comparison. The blue indicates decreased coherence while the red indicates increased coherences. The size of the line dictates the probability of the obtained t value. The thinner line represents  $\alpha$  at less than or equal to .05. The middle size line indicates  $\alpha$  less than or equal to .025, and the larger line indicates values at  $\alpha$  less than or equal to .010.



### Psychometric Post Measures

The post psychometric measures for all subjects were taken at Session 30, which was one week prior to the end of the spring semester. We opted for this time to avoid the possible confounding effects of the stress and anxiety associated with finals. Table 1 shows the results for the analysis of the pre- and post-obtained subtests, WMI and PSI scores. Included in the table are the pre and post subtest scaled scores, the mean, standard deviation, 95% confidence intervals (lower-upper), the difference between the pre- and post-subtests, the degrees of freedom, the F value and the probability of F. In psychometric testing, there is the consideration of practice effect and test/retest reliability. For the WAIS-III, the test/retest gains and losses for the age group 16-29 are reported as: Coding (+ 1.2),  $p < .001$ , Arithmetic (+ 0.6),  $p < .001$ , Digit Span (+ 0.5),  $p < .05$ , Symbol Search (+ 1.0),  $p < .001$ , Letter-Number Sequencing (+

0.1),  $p > 0.05$ , Working Memory Index (+ 2.9),  $p < .01$ , Processing Speed Index (+ 6.0),  $p < .001$  (Sattler, 2001). The average gains in test/retest are measured for a six-month period. The highest test increase for this practice effect occurs in Perceptual Organization (2.7 to 7.4) and the smallest increase occurs in Working Memory (1.3 to 3.1) in the Full Scale test/retest administration (Sattler, 2001). The Spearman rank-order correlation for our subject age group for test/retest gains at six months is  $-.56$  which suggests that practice effects are likely to be smaller for the most reliable subtests (Sattler, 2001). Our test/retest period was at four months, and allowing the possibility that the practice effect would increase to some degree; however, we consider the magnitude of change in the WMI and PSI scores considerably higher than might be accounted for by this effect alone. The differences between the pre and post measure scores are significantly higher in our group than in the test/retest group. Except in the Arith-

TABLE 1. The differences between pre- and post-psychometric measures are shown. There was no change in arithmetic and the change in LN sequencing is in the desired direction.

Group Pre- Post-WAIS III Subtest and Index Scores							
Subtest *Index	Mean	SD	95% Lower-Upper	Diff	df	F	p
Pre C	11	2.77	(8.67-13.32)				
Post C	13	3.20	(10.31-15.68)	+2	1,6	51.32	0.0004
Pre A	13	3.02	(11.09-16.05)				
Post A	13	1.99	(11.95-15.29)	0	1,6	2.83	0.1438
Pre DS	12	2.99	(10.36-15.38)				
Post DS	15	4.36	(11.59-18.90)	+3	1,6	11.48	0.0147
Pre SS	11	1.84	(9.83-12.91)				
Post SS	13	2.66	(11.14-15.60)	+2	1,6	14.01	0.0096
Pre LN	12	3.70	(9.53-15.71)				
Post LN	14	2.54	(12.61-16.88)	+2	1,6	4.89	0.0691
*Pre WMI	117	16.44	(103-131)				
*Post WMI	128	17.64	(114-143)	+11	1,6	45.12	0.0005
*Pre PSI	106	11.13	(97-115)				
*Post PSI	117	14.17	(105-129)	+11	1,6	23.93	0.0027

metic subtest, there was no change in the mean score. The Letter-Number sequencing scores for our group were higher than the test/retest group and changed in the desired direction but not at significant levels.

Table 1 shows the differences between pre- and post-psychometric measures. There was no change in arithmetic and a desired change in LN sequencing.

## DISCUSSION AND CONCLUSION

This is the first study of its kind to present the changes that occur in EEG power and coherence in normal subjects resulting from approximately thirty sessions of spatial-specific neurofeedback training. The obtained data illustrate the significant changes in spectral power and coherence that occur as a result of this training protocol, and add support to the suggestion that human beings can learn to activate and change activity in their own anterior cingulate gyrus (Congedo, 2003; Congedo, Lubar, & Joffe, 2004; Cannon et al., 2005). More importantly, the data illustrates that LNFB increases absolute power and influences differentiation and integration of neuronal populations in specific frequencies. There appears to be a reorganization of all frequencies in numerous cortical regions over sessions. The data suggest that LNFB in the ACCd increases absolute power in

many regions known to share anatomical connections with the AC and that are shown to be active during attentional, cognitive and executive processes (Tzourio, Massiou, Joliot, Renault, & Mazoyer, 1997; Stathopoulou & Lubar, 2004; Pardo, Fox, & Raichle, 1991; Cabeza et al., 2003; Towle et al., 1993).

It is reported that beta activity (13-21 Hz) is associated with active processing of information and focused attention (Neidermeyer & da Silva, 1987). The data obtained in this study identify a specific pattern of absolute power increase in the frontal-central, bilateral dorso-lateral prefrontal cortex and right parietal regions at FP1, FZ, F3, F4, P3, P4, and T6 over sessions in the trained frequency (14-18 Hz). This increase in absolute power specific to these regions can possibly be attributed to an executive circuit that activates in the trained frequency relative to the AC. This pattern increases in absolute power and coherence at significant levels at or between electrodes FZ, PZ, F3, F4, CZ, P4, and T6, as well as the in the superior prefrontal at FP1 and FP2 and parietal-occipital regions at O1 and O2 over sessions and becomes clearly discernable in the topographical maps. Of particular interest is the significant change in the 13 Hz frequency in earlier sessions and the resulting involvement of the entire frontal and central cortices in later sessions. Figures 3G and 3H show the increase in coherence between specific pairs of electrodes in the beta frequency over sessions and figure 2I shows the increase or decrease for each specific frequency over sessions. The topographical maps show this protocol influenced integration among neuronal populations in the delta, theta, alpha and trained frequency (14-18 Hz), while increasing complexity and differentiation among neuronal populations in the higher beta frequencies.

It is suggested that several independent circuits operate to control attention, cognition and executive functions. Alternatively, executive functions are suggested to include all the processes of attention, cognition, memory, initiation and drive, response inhibition, task persistence, organization, generative thinking and awareness (Sohlberg & Mateer, 1989). It is our speculation that the data obtained in this study offers support to this second suggestion, and maps a plausible circuit of executive function,

as it pertains to central attention, involving these anterior regions and the AC. If the AC is indeed a gating mechanism, as suggested by Pizzagalli, Oakes, and Davidson (2003), then sustained activity in the cluster of voxels in this region makes sense. It is possible that neuronal populations in the AC achieve a ceiling effect and initiate facilitation of cortical areas that are known to receive projections from the AC (Cannon et al., 2005). In effect, that it is plausible to train this executive circuit by training one discriminate area in the AC. It is suggested that AC abnormalities are involved in mood disorders, schizophrenia and attentional disorders, and studies report reduced glial density and blood flow, neuronal size and synaptic subtypes in the AC in many of these disorders (Eastwood & Harrison, 2001). It is foreseeable that LNFB training would be as efficacious in the treatment of clinical syndromes as it was in normal subjects. Our future research directions include studies designed to clarify this speculation. One benefit to this study would have been to include a control group for comparison. We plan to implement control groups in our future studies.

There are significant increases in coherence between electrodes in the right prefrontal cortex and the left prefrontal, central, and right parietal cortices. Neuronal populations in the RPFC correlate at significant levels with neuronal populations in the right inferior temporal, right parietal and left prefrontal regions, offering support for right hemispheric dominant activation during attentional tasks (Tzourio, Massiou, Joliot, Renault, & Mazoyer, 1997; Garavan, Ross, & Stein, 1999; Fazio et al., 1992). The coherence within the right hemisphere and contralaterally to the LPFC increases over sessions. fMRI studies of attention and episodic memory report that a right lateralized fronto-parietal network remains active during attentional tasks and the left prefrontal is more active during episodic memory tasks (Cabeza et al., 2003; Tucker, Dawson, Roth, & Penland, 1983). In our study, the sustained activity in the RPFC, RPCG and right inferior temporal cortex across sessions can be attributed to this network of attention, and its contribution to concentration on motor control, spatial information, muscle activity, attention to surroundings, the game itself, and visual and

auditory stimuli and possibly cognitive processes. Since we know that the left hemisphere of the prefrontal cortex is involved in the analysis of information, controlling serial behaviors and recognizing serial events and the right prefrontal cortex is involved in synthesis (putting things together as a whole), inhibition and emotional behavior (Carlson, 2001).

Event related coherence studies report increased synchronization in the bilateral DLPFC in a GO/NO GO experiment and suggest that it is related to both decision-making and motor inhibition processes (Shibata et al., 1998). Our data supports this bilateral prefrontal effort associated with inhibition of movement and decision-making, however, we would include the processes of attention and using cognitive and mnemonic processes as goal directed behaviors. The increases in coherence between hemispheres and within the right hemisphere suggest that LNFB positively influenced synchrony and complexity among the neuronal populations in this study (Cannon et al., 2005). There is a significant increase between the bilateral DLPFC, RPCG and ACcd in the trained frequency. There is also a decrease in coherence for high beta activity within the bilateral and central posterior parietal lobes; however, the superior frontal and posterior occipital cortices increase in high beta activity and not the trained frequency.

The psychometric results offer support for the effects of LNFB. The increase in PSI scores suggests that the neurofeedback training positively influenced processes involving visual motor coordination, attention, concentration, visual acuity, visual scanning and tracking and short-term memory for learning new tasks. Similarly, the increased WMI index score suggests a positive influence in short-term memory, auditory memory and attentional processes, which would be aided by the LPFC and ACcd (Kondo et al., 2003). The results suggest that LNFB training in the ACcd had an overall positive influence in both the Working Memory and Processing Speed tasks. We suspect that the increased coherence between occipital and superior frontal regions, especially the right frontal and inferior temporal lobes is involved in this increase.

The attentional and cognitive aspects of executive function remain a focal point for re-



searchers. Neurofeedback techniques are of interest in neurological improvement and enhancement of specific functions and treatment of psychological disorders and traumatic brain injury. This study illustrates that LORETA neurofeedback in the cognitive division of the AC produces significant changes in the spectral power and coherence in the topographical EEG of normal subjects as compared to baseline and the beginning session. The data suggest that this training significantly influenced EEG coherence between anterior and posterior regions and within and between hemispheres, as well as increasing absolute power amplitude throughout cortical regions that are associated with the AC.

We have demonstrated that increasing 14-18 Hz low beta activity in a seven voxel cluster in the cognitive division of the AC does result in significant changes in absolute power and coherence in the bilateral dorsolateral prefrontal cortices and the right post central gyrus. This study supports the idea of the AC being a control mechanism for regions of the cortex and the data demonstrate that the AC influences the anterior regions in the trained frequency (14-18 Hz). This power and coherence increase over sessions suggests that the AC is instrumental in recognizing, planning and learning a particular task and thereby increases or decreases activity in the cortex in the same trained frequency to elicit the desired response. In effect, it is possible this is a goal directed and motivational management of cortical activity.

There are many locations such as the insular cortex, parahippocampal, lingual, fusiform, and orbital-frontal gyri, and other regions at the base of the brain where it is doubtful that surface EEG neurofeedback would have much influence and LNFB would be the best methodology for activating or deactivating these areas. LNFB is more complicated than conventional neurofeedback. Conventional neurofeedback involves directly increasing or decreasing microvolt levels of specific frequencies in raw EEG. LNFB derives through complex mathematical computations of current sources in the brain gray matter, which is believed to generate the surface EEG (Nunez, 1995). In our implementation LNFB is based on 19 or more electrodes and three-dimensional vector based measurements from 2394 7 mm<sup>3</sup> voxel. LNFB

requires inhibiting surface EEG activity from linear combinations of EEG channels in addition to EMG and EOG activity monitored from multiple sites. It is much more demanding than conventional neurofeedback in regard to inhibiting artifacts. LNFB requires intensive effort on the part of subjects, and may require even more sessions than were conducted in this study. More studies are needed cross correlating LORETA with fMRI and PET for both normal cognitive activity and clinical syndromes.

The data suggest that the AC can be trained to activate and initiate specific regions of the cortex relative to task. There appears to be a multi-dimensional attentional and cognitive network involving the AC, DLPFC and the RPCG and activation of this network in the 14-18 Hz frequency in the anterior regions of the cortex decreases the same frequency in posterior parietal and occipital regions, but increases these latter regions in higher beta frequencies. The intricacies of the human brain are many and this study demonstrates the dynamic relationship between the AC and other regions of the cortex involved in attention, cognition and motivational processes. Further study is important to understand the relationship between the areas of the cortex identified in this study in executive functions and the contribution of the AC to all aspects of human behavior. Our future research will involve training individuals to activate the clusters of neuronal populations in the LPFC and RPFc identified in this paper to perform a cortical-sub-cortical comparison with the training in the ACcd and these two groups.

## REFERENCES

- Aoki, F., Fetz, E., Shupe, L., Lettich, E., & Ojemann, G. (2001). Changes in power and coherence of brain activity in human sensorimotor cortex during performance of visuomotor tasks. *Biosystems*, *63*, 89-99.
- Barnea, A., Rassis, A., & Zaidel, E. (in press). Effect of neurofeedback on hemispheric word recognition. *Brain and Cognition*.
- Barnea, A., Rassis, A., Raz, A., Othmer, E., & Zaidel, E. (in press). Effects of neurofeedback on hemispheric attention networks. *Brain and Cognition*.
- Bench, C., Frith, C., Graby, P., Friston, K., Paulesu, E., Frackowiak, R., et al. (1993). Investigations of the

- functional anatomy of attention using the Stroop Test. *Neuropsychologia*, 31, 907-922.
- Blom, J. L., & Anneveldt, M. (1982). An electrode cap tested. *Electroencephalography and Clinical Neurophysiology*, 54, 591-594.
- Cabeza, R., Dolcos, F., Prince, S., Rice, H., Weissman, D., & Nyberg, L. (2003). Attention-related activity during episodic memory retrieval: A cross-function fMRI study. *Neuropsychologia*, 41, 390-399.
- Cairo, T., Liddle, P., Woodward, T., & Ngan, E. (2004). The influences of working memory load on phase specific patterns of cortical activity. *Cognitive Brain Research*, 21, 377-387.
- Cannon, R., Lubar, J. F., Congedo, M., Gerke, A., Thornton, K., Kelsay, B., et al. (2006). The effects of neurofeedback training in the cognitive division of the anterior cingulate gyrus. *International Journal of Science* (in press).
- Cannon, R., Lubar, J., Thornton, K., Wilson, S., & Congedo, M. (2005) Limbic beta activation and LORETA: Can hippocampal and related limbic activity be recorded and changes visualized using LORETA in an affective memory condition? *Journal of Neurotherapy*, 8 (4), 5-24.
- Carlson, N. (2001). *Physiology of behavior* (7th ed., pp. 79-80). Boston, MA: Allyn and Bacon.
- Carr, T. (1992) Automaticity and cognitive anatomy: Is word recognition automatic? *American Journal of Psychology* 105, 201-237.
- Congedo, M. (2003). *Tomographic neurofeedback: A new technique for the self-regulation of brain electrical activity*. Unpublished doctoral dissertation. University of Tennessee, Knoxville.
- Congedo, M., Lubar, J., & Joffe, D. (2004). Tomographic neurofeedback: A new technique for the self-regulation of brain electrical activity [Abstract]. *Journal of Neurotherapy*, 8 (2), 141-142.
- Coull, J., Frackowiak, R., & Frith, C. (1998). Monitoring for target objects: activation of right frontal and parietal cortices with increasing time on task. *Neuropsychologia*, 36 (12), 1325-1334.
- Devinsky, O., Morrell, M., & Vogt, B. (1995). Review article: Contributions of anterior cingulate cortex to behaviour. *Brain*, 118, 279-306.
- Eastwood, S., & Harrison, P. (2001). Synaptic pathology in the anterior cingulate cortex in schizophrenia and mood disorders. A review and a Western blot study of synaptophysin, GAP-43 and the complexins. *Brain Research Bulliten*, 55 (5), 569-578.
- Efthymios, A. & Lubar, J. (2002). Quantitative electroencephalographic amplitude measures in young adults during reading tasks and rest. *Journal of Neurotherapy*, 6 (2), 5-19.
- Egner, T., & Gruzelier, J. (2001). Learned self-regulation of EEG frequency components affects attention and event-related brain potentials in humans. *NeuroReport*, 12, 4155-4159.
- Egner, T., & Gruzelier, J. (2003). Ecological validity of neurofeedback: Modulation of slow wave EEG enhances musical performance. *NeuroReport*, 14, 1221-1224.
- Egner, T., Zech, T., & Gruzelier, J. (2004). The effects of neurofeedback on the spectral topography of the electroencephalogram. *Clinical Neurophysiology*, 115, 3452-3460.
- Fazio, F., Perani, D., Gilardi, M. C., Colombo, F., Cappa, S. F., & Vallar, G. (1992). Metabolic impairment in human amnesia: A PET study of memory networks. *Journal of Cerebral Blood Flow Metabolism*, 12, 353-358.
- Fein, G., Raz, J., Brown, F., & Merrin, L. (1988). Common reference coherence data are confounded by power and phase effects. *Electroencephalography and Clinical Neurophysiology*, 69 (6), 581-584.
- Garavan, H., Ross, T. J., & Stein, E. I. (1999). Right hemispheric dominance of inhibitory control: An event related functional MRI study. *Proceedings of the National Academy of Science, USA*, 96, 8301-8306.
- Heyder, K., Suchan, B., & Daum, I. (2004). Cortico-subcortical contributions to executive control. *Acta Psychologica*, 115, 271-289.
- Hogan, M., Swanwick, G., Kaiser, J., Rowan, M., & Lawlor, B. (2003). Memory-related EEG power and coherence reductions in mild Alzheimer's disease. *International Journal of Psychophysiology*, 49 (2), 147-163.
- Jausovec, N., & Jausovec, K. (1999). EEG activity during the performance of complex mental problems. *International Journal of Psychophysiology*, 36, 73-88.
- Knyazeva, M., & Innocenti, G. (2001). EEG coherence studies in the normal brain and after early-onset cortical pathologies. *Brain Research Reviews*, 36, 119-128.
- Kondo, H., Morishita, M., Osaka, N., Osaka, N., Fukuyama, H., & Shibasaki, H. (2003). Functional roles of the cingulo-frontal network in performance on working memory. *Neuroimage*, 21, 2-14.
- Lubar, J. F. (1984). Electroencephalographic biofeedback of SMR and beta for treatment of attention deficit disorders in a clinical setting. *Biofeedback and Self-Regulation*, 9, 1-23.
- Lubar, J. F. (1985). Changing EEG activity through biofeedback applications for the diagnosis and treatment of learning disabled children. *Theory into Practice, Ohio State University*, 24, 106-111.
- Lubar, J. F. (1991). Discourse on the development of EEG diagnosis and biofeedback treatment for attention deficit/hyperactivity disorders. *Biofeedback and Self-Regulation*, 16, 201-225.
- Lubar, J. F. (1995a). Neurofeedback treatment of attention deficit hyperactivity disorder: Research and clinical implications. *Biobehavioral self-regulation in the East and West* (pp. 312-323). Tokyo: Springer-Verlag.
- Lubar, J. F. (1995b). Neurofeedback for the management of attention deficit-hyperactivity disorders. In M.S. Schwartz (Ed.), *Biofeedback: A practitioners'*

- guide (2nd ed., pp. 493-522). New York: Guilford Publications, Inc.
- Lubar, J. F., Swartwood, M. O., Swartwood, J. N., & O'Donnell, P. (1995). Evaluation of the effectiveness of EEG in neurofeedback training for ADHD in a clinical setting as measured by changes in T.O.V.A. scores, behavioral ratings, and WISC-R performance. *Biofeedback and Self-Regulation*, *20*, 83-99.
- Lubar, J. F. & Lubar, J. (1999). Neurofeedback assessment and treatment for attention deficit/hyperactivity disorders. In A. Abarbanel & J. R. Evans, (Eds.), *Introduction to quantitative EEG and neurofeedback* (pp. xxi, 103-143, 406). San Diego, CA: Academic Press, Inc.
- Markela-Lerenc, J., Ille, N., Kaiser, S., Fiedler, P., Mundt, C., & Weisbrod, M. (2004). Prefrontal-cingulate activation during executive control: Which comes first? *Cognitive Brain Research*, *18*, 278-287.
- Niedermeyer, E., & Lopes da Silva, F. (Eds.). (1987). *Electroencephalography: Basic principles, clinical applications and related fields* (2nd ed.). Baltimore, MD: Urban & Schwarzenberg.
- Nunez, P. (1995). *Neocortical dynamics and human EEG rhythms*. New York: Oxford University Press.
- Nunez, P. L., & Silberstein, R. B. (2000). On the relationship of synaptic activity to macroscopic measurements: Does co-registration of EEG with fMRI make sense? *Brain Topography*, *13* (2), 79-96.
- Nunez, P. L., Srinivasan, R., Westdorp, A. F., Wijesinghe, R. S., Tucker, D. M., Silberstein, R. B., et al., (1997). EEG coherency I: Statistics, reference electrode, volume conduction, laplacians, cortical imaging, and interpretation at multiple scales. *Electroencephalography and Clinical Neurophysiology*, *103*, 499-515.
- Nunez, P. L., Wingeier, B. M., & Silberstein, R. B. (2001). Spatial-temporal structures of human alpha rhythms: Theory, microcurrent sources, multiscale measurements, and global binding of local networks. *Human Brain Mapping*, *13* (3), 125-164.
- Ortuño, F., Ojeda, N., Arbizu, J., Lopez, P., Marti-Climent, J.M., Peñuelas, I., et al., (2001). Sustained attention in a counting task: Normal performance and functional neuroanatomy. *Neuroimage*, *17*, 411-420.
- Pardo, J. V., Fox, P. T., & Raichle, M. E. (1991). Localization of a human system for sustained attention by positron emission tomography. *Nature*, *349*, 61-64.
- Pardo, J. V., Pardo, P., Janer, K., & Raichle, M. (1990). The anterior cingulate cortex mediates processing selection in the Stroop attentional conflict paradigm. *Proceedings of the National Academy of Science, USA*, *87*, 256-259.
- Pascual-Marqui, R. D., Michel, C. M., & Lehmann, D. (1994). Low-resolution electromagnetic tomography: a new method for localizing electrical activity in the brain. *International Journal of Psychophysiology*, *18*, 49-65.
- Pascual-Marqui, R. D. (1995). Reply to comments by Hämäläinen, Ilmoniemi and Nunez. In source localization: Continuing discussion on the inverse problem. In W. Skrandies (Ed.). *ISBET Newsletter*, *6*, 16-28.
- Pascual-Marqui, R. D. (1999). Review of methods for solving the EEG inverse problem. *International Journal of Bioelectromagnetism*, *1* (1), 75-86.
- Peniston, E. G., & Kulkosky, P. J. (1989). Brainwave training and beta-endorphin levels in alcoholics. *Alcoholism: Clinical and Experimental Research*, *13* (2), 271-279.
- Peniston, E. G., & Kulkosky, P. J. (1990). Alcoholic personality and alpha-theta brainwave training. *Medical Psychotherapy: An International Journal*, *3*, 37-55.
- Peniston, E. G., & Kulkosky, P. J. (1991). Alpha-theta brainwave neuro-feedback therapy for Vietnam veterans with combat-related post-traumatic stress disorder. *Medical Psychotherapy: An International Journal*, *4*, 47-60.
- Pizzagalli, D., Oakes, T. R., & Davidson, R. J. (2003). Coupling of theta and glucose metabolism in the human rostral anterior cingulate cortex: An EEG/PET study of normal and depressed subjects. *Psychophysiology*, *40*, 939-949.
- Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience*, *13*, 25-42.
- Rubia, K., Smith, A., Brammer, M., & Taylor, E. (2003). Right inferior prefrontal cortex mediates response inhibition while mesial prefrontal cortex is responsible for error detection. *Neuroimage*, *20*, 351-358.
- Roland, P. E. (1984). Metabolic measurements of the working frontal cortex in man. *Trends in Neuroscience*, *7*, 430-435.
- Sattler, J. (2001). *Assessment of children: Cognitive applications* (4th ed., pp. 382-395). San Diego, CA: Jerome M. Sattler, Publishers Inc.
- Schack, B., Griezbaum, G., & Krause, W. (1999). The sensitivity of instantaneous coherence for considering elementary comparison processing. Part I: The relationship between mental activities and instantaneous coherence. *International Journal of Psychophysiology*, *31*, 219-240.
- Sohlberg, M., & Mateer, C. (1989). *Introduction to cognitive rehabilitation: Theory and practice* (pp. 236-237). New York: The Guilford Press.
- Stathopoulou, S., & Lubar, J. (2004). EEG changes in traumatic brain injured patients after cognitive rehabilitation. *Journal of Neurotherapy*, *8* (2), 21-51.
- Sterman, B. (2000). EEG markers for attention deficit disorder: Pharmacological and neurofeedback applications. *Child Study Journal*, *30* (1), 1-23.
- Sterman, B., & Kaiser, D. (2001). Comodulation: A new QEEG analysis metric for structural and functional disorders of the central nervous system. *Journal of Neurotherapy*, *4* (3), 73-83.
- Thatcher, R., McAlaster, R., Lester, M. I., Horst, R. L., & Cantor, D. S. (1983). Hemispheric EEG asymmetries related to cognitive functioning in children. In A. Porecman (Ed.), *Cognitive processing in the right hemisphere* (pp. 125-146). New York: Academic Press.

- Thatcher, R., Krause, P. J., & Hyrbyk, M. (1986). Cortico-cortical associations and EEG coherence: A two-compartmental model. *Electroencephalography and Clinical Neurophysiology*, *64*, 123-143.
- Thatcher, R., North, D., & Biver, C. (in press). EEG and intelligence: Relations between EEG coherence, EEG phase delay and power. *Clinical Neurophysiology*.
- Thornton, K., & Carmody, D. (2005). Electroencephalogram biofeedback for reading disability and traumatic brain injury. *Child and Adolescent Psychiatric Clinics of North America*, *14*, 137-162.
- Towle, V. L., Bolaños, J., Suarez, D., Tan, K., Grzeszczuk, R., Levin, D. N., et al. (1993) The spatial location of EEG electrodes: Locating the best fitting sphere relative to cortical anatomy. *Electroencephalography and Clinical Neurophysiology*, *86*, 1-6.
- Tucker, D., Dawson, S., Roth, D., & Penland, J. (1983). Regional changes in EEG power and coherence during cognition: Intensive study of two individuals. *Behavioral Neuroscience*, *99* (3), 564-577.
- Tucker, D., Roth, D., & Bajr, T. (1986). Functional connections among cortical regions: Topography of EEG coherence. *Electroencephalography and Clinical Neurophysiology*, *63*, 242-250.
- Tzourio, N., Massiou, F., Joliot, M., Renault, B., & Mazoyer, B. (1997). Functional anatomy of human auditory attention studied with PET. *Neuroimage*, *5*, 63-77.
- Vernon, D., Egner, T., Cooper, N., Compton, T., Neilands, C., Sheri, A., et al. (2003). The effect of training distinct neurofeedback protocols on aspects of cognitive performance. *International Journal of Psychophysiology*, *47*, 75-85.
- Wechsler, D. (1997). *Wechsler Adult Intelligence Scale-3rd Edition*, Psychological Corporation, San Antonio, TX.
- Winer, B. (1962). *Statistical principles in experimental design*. New York: McGraw Hill.

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