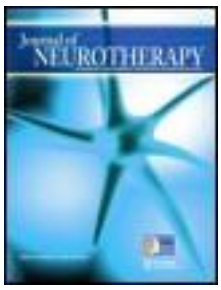

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Neurofeedback Efficacy in the Treatment of a 43-Year-Old Female Stroke Victim: A Case Study

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ABSTRACT. *Introduction*. A 43-year-old Caucasian woman presented with a series of physical and mental deficits following a right hemisphere cerebral artery embolus suffered at age 42.

Method. For both the pretreatment and posttreatment evaluation, the client's EEG data were collected. Prior to beginning neurofeedback a self-developed symptom checklist was provided to the participant and was repeated every 10 sessions. The participant received 52 neurofeedback sessions with the use of Neurocybernetics equipment. To determine statistical changes between the pretreatment and posttreatment conditions, average cross-spectral matrices were computed for bands delta (1–3.5 Hz), theta (3.5–7.5 Hz), alpha (7.5–12.5 Hz), beta1 (12.5–25 Hz), beta2 (25–32 Hz), and gamma (37–47 Hz). In this study the pretreatment cross-spectra for each epoch were then compared to the posttreatment epoch cross-spectra using the previously mentioned frequency band ranges. For each condition, cross-spectral matrices were computed and averaged over 2-s epochs resulting in one cross-spectral matrix for each epoch and for each of the discrete frequencies within each band. Based on previous LORETA analyses, we used a rectangular window. No time frame or frequency wise normalization was performed.

Results. Following treatment, comparative QEEG and eLoreta analyses illustrated significant decreases in the absolute and relative power theta measures and significant elevations of absolute and relative power occipital beta. These findings correspond to client self-report data demonstrating improvement in cognitive functioning and depressed mood.

Conclusion. Overall, findings suggest the utility of neurofeedback for the treatment of stroke, with particular gains noted in the areas of cognitive functioning, sleep quality, emotional regulation, and energy.

KEYWORDS. Brodmann's areas, eLORETA, embolus, neurofeedback, qEEG, quantitative EEG, stroke

INTRODUCTION

Strokes occur when blood clots or broken blood vessels disrupt the flow of blood to areas of the brain, causing the death of blood cells and subsequent damage to the brain. When isolated from other cardiovascular diseases, stroke remains the third most

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deadly disease for men and women living in the United States (American Heart Association [AHA], 2009). Sadly, for those that live through the stroke itself, regaining function and dealing with the loss of previous ways of being can be nearly as devastating. Although there are many therapies available to stroke patients that have been found to be highly effective at treating physical and emotional symptoms, limited research has been done to evaluate the efficacy of using biofeedback or neurofeedback in the treatment of stroke victims (Hammond, 2006). What research does exist tends to support the ability of neurofeedback to improve speech, coordination, attention, memory, and concentration (Nelson, 2007; Putman, 2001; Rozelle & Budzynski, 1995). Research has also indicated that mood disregulation, particularly anxiety and depression, which is frequently associated with stroke, can be improved with the use of neurofeedback (Putman, 2001; Rozelle & Budzynski, 1995).

Although stroke can occur at any age, much like other diseases, the prevalence of stroke increases dramatically with age. This is particularly true as men and women reach the age of 60, where rates for each are around 7% (AHA, 2009). Prior to this age limit, stroke prevalence is 0.3% and 2.9% for women in the age groups of 20 to 39 and 40 to 59, respectively (AHA, 2009). What is missing in the research at large is an evaluation of treatment options for stroke victims who do not fall into higher risk categories. In particular, stroke victims, perceived as ''young'' with regard to risk standards, may carry particular risks for emotional consequences of stroke due to their age and lifestyle standards. That is, those who are younger and perceived to be healthier may be more greatly impacted by disruptions from a stroke in their physical function and daily lives. In a study conducted by Neau et al. (1998), it was found that poststroke depression occurred in 48.3% of young adult victims aged 15 to 45. Factors that appear to contribute to poststroke depression include localization of the infarct, the severity of any disabilities, a bad general outcome, and an inability to return to work (Neau et al., 1998).

The stroke recovery process and prognosis is as varied as the stroke itself. In the landmark Copenhagen Stroke Study (Jorgensen et al., 1995), 1,197 acute stroke patients were examined for neurological deficits and functional difficulties at the time of admission, following rehabilitation, and 6-months poststroke. The results of this study indicated that functional recovery occurred in 95% of stroke patients within 12.5 weeks (Jorgensen et al., 1995). Although not surprising, the degree of stroke recovery and speed with which this was achieved was largely dependent on the severity of the initial stroke. However, even patients within the severe stroke category maximized their functional recovery by 20 weeks. Neurological recovery was found to precede functional recovery by an average of 2 weeks (Jorgensen et al., 1995). Unfortunately, utilizing traditional rehabilitation, these researchers found that there would be no neurological or functional recovery expected after 5 months (Jorgenson et al., 1995). Although this study does provide some hope for the prognosis of stroke patients, it does not address whether recovery returns to prestroke levels. Further, it supports conventional medical wisdom that indicates limited recovery options beyond the initial recovery period. For patients who seek to return to a prestroke lifestyle, additional therapies and alternatives may be warranted.

Quantitative electroencephalography (QEEG) and other imaging techniques such as Low-resolution electromagnetic tomography (LORETA) are methods for localizing electrical activity in the brain based on multichannel scalp EEG recordings (Pascual-Marqui, Michel, & Lehmann, 1994). To our knowledge, this technique has not been implemented to a great degree in the neuroelectrical investigation of stroke patients' rehabilitation following neurofeedback intervention.

QEEG analysis has been used in other literature for the purpose of understanding the impact from stroke (Claassen et al., 2004; Nuwer, Jordan, & Ahn, 1987) and has been used as a technique for evaluating specific EEG features in a comparative methodology in stroke as well (Szelies,

Mielke, Kessler, & Heiss, 2002). The LORETA method specifically has been refined since its original inception resulting first in sLORETA (standardized LORETA) and most recently eLORETA (exact LORETA), providing now a three-dimensional distributed, linear solution with exact localization of EEG sources (Pascual-Marqui, 2007; Pascual-Marqui et al., 2006). To our knowledge, this is the first investigation using the eLORETA method for the evaluation of the effects of neurofeedback in any stroke group or case evaluation.

METHOD

Participant

The participant was a 43-year-old Caucasian woman who presented with a series of physical and mental deficits following a right hemisphere cerebral artery embolus she suffered at age 42. The participant indicated that she had no prior health issues. Prior to the stroke, the participant was an avid bicycle rider and soccer player, was in excellent health, and fit none of the risk factors that would put her at risk for a stroke (AHA, 2009). On November 14, 2007, while at work, she suffered from a right hemisphere stroke in the T6 region (International 10–20 System; Niedermeyer & Lopes da Silva, 2004) that resulted in numbness and paralysis to the left side of her body, particularly her left arm and hand. Other symptoms included severe exhaustion, lack of focus, distractibility, and poor energy. Following the stroke she developed the secondary symptom of depression and was placed on 150 mg of Effexor XR. She was out of work for 3 months following the stroke.

Following an initial, and unsuccessful, attempt at talk therapy, the participant opted to pursue neurofeedback as a means to address her feelings of being "scattered" and "depressed." She indicated that her goals were to address the depression and sense of distractibility that developed following the stroke. In particular, she struggled with how this could have happened to her at such a young age and given the type of health she was in. Further, her inability to engage in bicycling and soccer following the stroke served to intensify her feelings of despair and frustration. She began neurofeedback therapy 1 year 1 month following her stroke.

Procedures

EEG data collection. For both the pretreatment and posttreatment evaluation, the client's EEG data were collected continuously in a dimly illuminated and sound attenuated room. The EEG was sampled with 19 electrodes in the standard 10–20 International placement referenced to linked ears. Data were collected for 10 min of baseline eyes-closed condition and 10 min of baseline eyes-open condition. Only data during baseline eyes-closed condition were evaluated for the implementation of neurofeedback protocols.

QEEG. Acquired digitized EEG data were plotted and carefully inspected visually using manual artifact rejection. All episodic artifacts including eye blinks, eye movements, teeth clenching, body movements, or EKG artifact was removed from the stream of EEG using the Eureka software (Congedo, 2005). For both the pretreatment and posttreatment conditions the data were then submitted to quantitative analysis software programs NXLink/Neurometrics (John, Prichep, & Easton, 1987), Neuroguide (Thatcher, Walker, Biver, North, & Curtin, 2003) and EureKa (Congedo, 2005), which computed the fast Fourier transform providing cross-spectral output. The data analysis evaluated for the development of the neurofeedback protocols included spectral power, percentage spectral power, frequency band ratios, and coherence and phase measures, as well as the comparison to normative database samples for the same measures. The normative database comparison analysis was age matched to the client based on the methods and variety of frequency bands employed within the specific software (Congedo, 2005; John et al., 1987; Thatcher et al., 2003). For this study, due to the presence of electromyography (EMG) artifact in the eyes-open condition, only the eyes-closed condition was used for comparative analysis.

Both the eyes-closed and eyes-open data were examined for determining clinical indications and protocol development taking into consideration contribution from EMG artifact.

Symptom checklist. Prior to beginning neurofeedback a self-developed symptom checklist was provided to the participant. The initial checklist was used to gauge severity of presenting symptoms and assessed both mood and physical symptoms on a 7-point Likert scale from 1 (*not problematic*) up to 7 (*very problematic*). This same checklist was modified to later assess progress as neurofeedback sessions continued (1 = *much better*, 2 = *better*, 3 = *somewhat better*, 4 = *no change*, 5 = *somewhat worse*, 6 = *worse*, 7 = *much worse*). The symptom checklist was provided to the participant every 10 sessions to assess changes in both mood and physical symptoms. Each checklist also provided short-answer prompts to allow the participant to discuss any changes in her environment or medication, as well as any additional comments/observations she would like to make about her progress.

Client interview. Approximately 1 month following the final neurofeedback session the primary researcher conducted a one-on-one interview with the participant to assess her neurofeedback experience and perceived progress throughout treatment.

Treatment procedures. The participant received 52 neurofeedback sessions with the use of Neurocybernetics equipment (Lightstone, 2009). Treatment was terminated at that point based on the client's satisfaction with her results. All training sessions were conducted for a 30-min period and were carried out at a rate of twice per week.

The selection of protocols and the order with which they were utilized was based on several criteria. The initial consideration was the likelihood of success at the training site. Protocol preference was first given to central sites where prior anecdotal and nonresearch population findings indicate that people tend to have an easier time responding to neurofeedback. The second criteria included protocol placements that were likely to have a general positive effect, based on the researchers' prior treatment experience. The third criteria factored in the relevance of the protocol placement to the clinical presentation and noted responses to training by the client. Initial symptom presentation was correlated with corresponding brain structures for initial placement and was modified as the client both did and did not respond to training over time. For example, after 10 sessions of training at site C3, the client reported feeling "on her game" with improved focus and awareness, particularly at work. Finally, the QEEG results, specifically deviation from the normative sample, were considered.

In response to the final criteria, protocol selections were based on noted aberrations in the initial QEEG that demonstrated localization and slowing (5–8 Hz) in the left parietal and central sites (Figure 1), as well as significant coherence and phase aberrations in the right temporal/parietal site of T6 (Figures 2 and 3). Cortical slowing, particularly in the theta frequencies, has previously been associated with stroke presentation (Bearden, Cassisi, & Pineda, 2003; Rozelle & Budzynski, 1995). Decisions on when to switch sites were based on client progress, symptom reports, and consistency

FIGURE 1. *Z*-score deviation from the reference population in the theta frequency band in the absolute power measure of the pretreatment QEEG assessment.

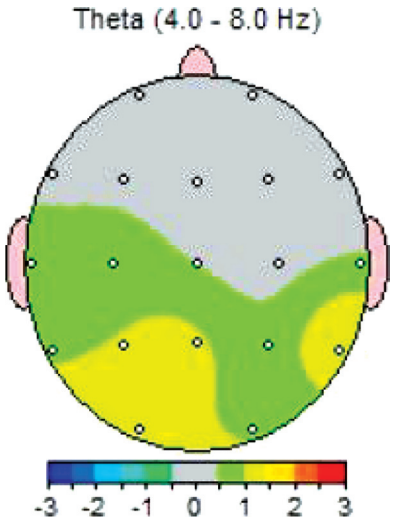
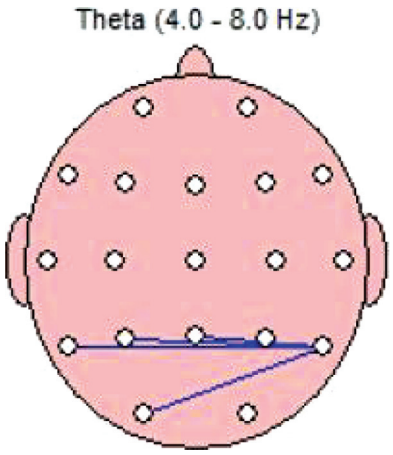
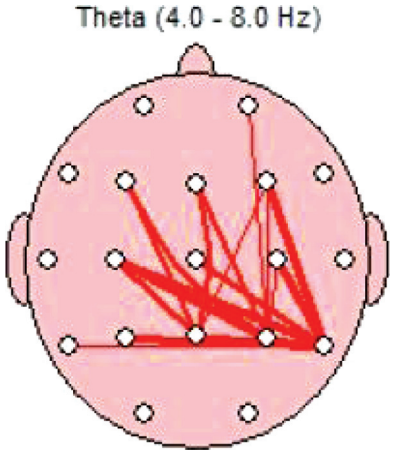


FIGURE 2. *Z*-score deviation from the reference population in the theta frequency band in coherence measure of the pretreatment QEEG assessment. *Note*. The blue line indicates a *Z*-score deviation of ≥ -2.58.



and modification of waveforms during and across sessions, as noted by the primary researcher. Sites were initially evaluated on change reported by the client and were maintained a minimum of 10 sessions thereafter to allow for permanent change to occur.

FIGURE 3. *Z*-score deviation from the reference population in the theta frequency band in the phase measure of the pretreatment QEEG assessment. *Note*. The thicker red lines indicate a *Z*-score value of $>/=+3.09$. The thinner red lines indicate a *Z*-score value of $>/=1.96$.



The first 32 sessions were completed using mono-polar montages in the eyes-open condition. Sessions 1 to 11 occurred at site C3. Theta (4–7 Hz) and high beta (22–36 Hz) were inhibited, whereas low beta (13–15 Hz) was rewarded. Sessions numbered 12 to 26 occurred at sites P3 and PZ, respectively. Fifteen minutes of training were done at each site, with both sites having a theta (4–7 Hz) and high beta (22–36 Hz) inhibit and a low beta (12–15 Hz) reward. Sessions 27 to 32 were trained at site POZ which included a theta (4–7 Hz) and high beta (15–36 Hz) inhibit and an alpha (9–12 Hz) reward.

The final 20 sessions were completed using bipolar montages in the eyes-closed condition. The movement from mono-polar to bipolar montages was done to address the multiple coherence and phase aberrations present in the initial QEEG. This change occurred once amplitude changes in the previous sites were deemed to have had their maximum benefit. It is worth noting that the hypercoherence issues primarily stemmed from the stroke site at T6. An attempt to decrease coherence between T6 and various sites was the goal of this training. Sessions 33 to 42 trained T6-T5 and included a beta inhibit (16–36 Hz) and slow wave (1–15 Hz) reward. Sessions 43 to 52 trained T6-PZ and also included a beta inhibit (20–36 Hz) and slow wave (1–15 Hz) reward.

Methods of comparative analysis of QEEG and Exact Low Resolution Electromagnetic Brain Tomography (eLORETA). A visual comparison was made between pretreatment and posttreatment QEEG differences by repeating the EEG data collection and QEEG methods described previously in this article. To determine statistical changes between the pretreatment and posttreatment conditions, average cross-spectral matrices were computed for bands delta (1–3.5 Hz), theta (3.5–7.5 Hz), alpha (7.5–12.5 Hz), beta1 (12.5–25 Hz), beta2 (25–32 Hz), and gamma (37–47 Hz) from both pre- and posttreatment assessments. In this study the pretreatment cross-spectra for each epoch were then compared to the posttreatment epoch cross-spectra using the previously mentioned frequency band ranges. For each condition,

cross-spectral matrices were computed and averaged over 2-s epochs resulting in one cross-spectral matrix for each epoch and for each of the discrete frequencies within each band. Based on previous LORETA analyses (Sherlin et al., 2007), we used a rectangular window. No time frame or frequency wise normalization was performed.

Quantitative EEG calculations were performed using the absolute and relative power measures. In this test observation the two groups are matched and the difference between each pair is computed. The mean of the differences is expected to equal zero under the null hypothesis. The test statistic is the well-known student t, with positive values indicating mean (A) > mean (B), and negative values indicating mean (A) < mean (B). These comparisons were made at the electrode level (Congedo, 2005).

The eLORETA calculations were performed using the latest version of the LORETA-KEY software distributed from the KEY Institute for Brain-Mind Research (Pascual-Marqui, 2007; Pascual-Marqui et al., 2006). This version of the LORETA KEY software implements exact low-resolution brain electromagnetic tomography, which is a true inverse solution with zero localization error (Pascual-Marqui, 2007). The new software package and viewer (Pascual-Marqui, 2007) were used to display the eLORETA current density in the frequency domain from the average cross-spectral matrix (Frei et al., 2001). This LORETA implementation incorporates a three-shell spherical head model registered to a recognized anatomical brain atlas (Talairach & Tournoux, 1988) and makes use of EEG electrode coordinates derived from cross-registration between spherical and realistic head geometry (Towle et al., 1993). The Montreal Neurological Institute brain volume was scanned at 5 mm resolution (Collins, Neelin, Peters, & Evans, 1994). The coordinates have been converted and corrected to Talairach coordinates (Brett, Johnsrude, & Owen, 2002). The solution space is restricted to cortical gray matter using the digitized probability atlas of the Brain Imaging Center at the Montreal Neurological Institute resulting in 6,239 voxels measuring $5 \times 5 \times 5$ mm (Collins et al., 1994).

The data permutation approach was used because it can adaptively account for the correlation structure of the variables, an embedded feature of all electrophysiological measurements (Holmes, Blair, Watson, & Ford, 1996). We performed 5,000 instances of randomization for each of the six frequency bands (delta, theta, alpha, beta1, beta2, and gamma) using the well-established techniques for nonparametric randomization (Nichols & Holmes, 2002). For the whole data set of both eLORETA (6,239 voxels $\times$ 6 frequency bands) and QEEG (19 electrodes $\times$ 6 frequency bands) a threshold of significance (if the global null hypothesis was false) was then computed using a two-tailed, independent pairs t test with an alpha rate of 0.05. For all bands, we tested the hypothesis that the mean eLORETA current source density per voxel, the mean power or mean percentage power per electrode, of the pretreatment condition differed from the posttreatment condition.

RESULTS

Symptom Checklist

Symptoms that the participant first indicated were moderately to very (4–7 on Likert scale) problematic to her prior to starting neurofeedback were tracked for progress across 10-session increments and are provided for review in Table 1. A final symptom checklist was provided to the participant following the 50th neurofeedback session but was not returned. It should be noted that the higher the number on the baseline assessment, the more problematic the symptom. Likewise, higher numbers on the follow-up assessments indicate a worsening of the symptom, as opposed to improvement. For marks made in between two numbers or inclusive of two numbers, an average is represented.

The results demonstrate that though none of her symptoms began as "very problematic," she had several that were "moderately problematic" or above. It is worth noting

TABLE 1. Changes in symptom checklist across 10-session intervals.

Symptom	Baseline Symptom Severity[a]	Progress After 10 Sessions[b]	Progress After 20 Sessions[b]	Progress After 30 Sessions[b]	Progress After 40 Sessions[b]
Not being organized	5	3.5	5	5	3
Unclear thinking	5	3	4	3	3
Slow reaction time	5	1	3	3	3
Poor attention	5	1	5	3	3
Spaciness or fogginess	5	2	4	2	3
Motivation	4	2	3	2	3
Energy	4	3	2	2	2
Feeling dull	4	4	3	3	3
Poor body awareness	4	4	4	3	3
Poor concentration	5	3	6	3	3

Note. MP = moderately problematic; VP = very problematic; MB = much better; NC = no change; MW = much worse.
[a]4 = MP; 7 = VP.
[b]1 = MB; 4 = NC, 7 = MW.

that the bulk of her symptoms dealt with cognitive processing issues. The results indicate that the participant noticed significant changes in many of her symptom categories within the first 10 sessions. Progress following those initial sessions waned, in some cases, but later continued to improve. Results following the 40th session indicate at least "somewhat better" improvement across all symptoms.

In addition to the numerical component of the Symptom Checklist, the participant and her family/friends/coworkers were allowed space on the back of the Symptom Checklist to comment on changes in the client and her environment. Comments provided by the participant on the short-answer prompts included, "I'm more with it, quicker to react"; "I'm told a lot that people can't tell I've stroked"; "I'm definitely more confident, sure, stable." Observations provided by her mother include, "She doesn't get lost in thought as often"; "She doesn't sound as tired"; "She's using her left hand more"; "No coma fog"; "She has more confidence"; "She's more emotional lately." Comments provided by her best friend include, "She's more expressive, more emotional, and frustrated by her limitations."

Client Interview

For those who are both researchers and clinicians, changes witnessed in topographic maps and symptom checklists are frequently considered secondary to the larger concept of "does my client *feel* better?" As a means to better address this question, the primary researcher conducted a posttreatment interview with the participant. Next are responses, in her own words, to questions posed by the researcher.

1. Why did you decide to do neurofeedback?

> What did I have to lose? I had no emotions except for my wit and humor. I had SIADD (stroke induced ADD) which made it tough to do anything, much less accomplish work. My house was a mess, I wasn't standing up for myself, something had to happen.

2. When did you start to notice changes with the neurofeedback?

   I started to notice changes right away, mostly that I was more 'with it,' more able to express myself. I know we did about 10 sessions for each spot. Most of the changes were noticed right away within the first 3–4 treatments on that spot. I really wish I had kept a blog on it, but I didn't. I do remember finally crying at something, which meant to me that my emotions were back. I seriously had not really just cried on my own about the stroke. By the time February came around, I was off the fattening anti-depressants [*Note*: by session 15, the participant had fully titrated off Effexor XR and was responding well] and was focusing better, but I still had no energy. I know there was a time when my sleep started to get better, I wouldn't wake up so many times at night. Then, we moved to the site where the stroke was, and almost immediately I had more energy. I was more alert, able to exercise and not be exhausted for 2 days later. Totally amazing! My mom could tell a difference in my voice right away. I was even sleeping less (from the 10 hrs I was sleeping prior down to 8–9 hours) and waking up earlier completely refreshed.

3. How would you describe those changes?

   It was weird, it wasn't like I could tell exactly what happened right away, but I could tell something happened. I definitely felt better, but couldn't exactly pinpoint why or what was better. I think a lot of it was just clearer thinking, better focus and the ability to express my thoughts. For the sleeping change, I noticed that I wouldn't toss and turn, I'd go to bed and go to sleep, right away, then I would stay asleep. The energy change was almost instant and especially noticeable after exercising. I was finally able to function the next day after riding my bike or that afternoon after riding that morning. It used to take 2–3 days to recover from the bike rides. I just felt 'normal' first time in 1.5 years. Having been a person who was always able to get things accomplished, the no energy part really hindered that.

4. Over the course of treatment, what changed, and at what intervals?

   Things that changed, not necessarily in this order: emotions came back, off the anti-depressants w/out slipping into a depression, clearer thinking, attention was better—better able to focus and not be easily distracted, better sleep, and energy. Things would change as we moved to a different spot. The change would happen within the day of the session, then it would taper off until the next session, then it would stay a bit longer.

5. Were you satisfied with your neurofeedback experience?

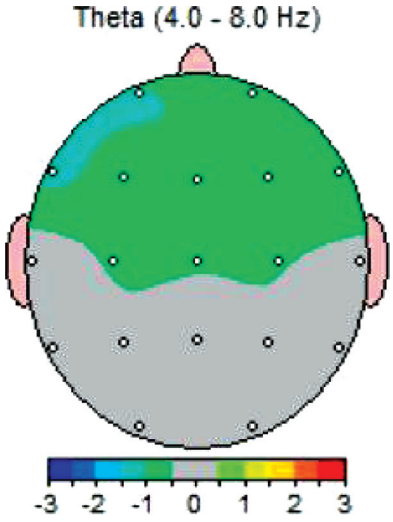
   Yes, I was extremely satisfied with the neurofeedback experience. It was painless and provided results I couldn't get elsewhere.

6. What symptoms do you continue to have following treatment?

   I still have a lot of physical issues—still no sense of touch in the left hand, pain from the muscles not relaxing, especially in the shoulder.

Pre- and Post-QEEG. It was previously mentioned that the protocols were based on noted aberrations in the initial analysis that demonstrated localization and slowing (5–8 Hz) in the left parietal and central sites, as well as significant coherence and phase

FIGURE 4. *Z*-score deviation from the reference population in the theta frequency band in the absolute power measure of the posttreatment QEEG assessment.



aberrations in the right temporal/parietal site of T6 (Figures 1–3). The follow-up QEEG results illustrate that there were objective changes in the differences between the pretreatment QEEG and the posttreatment QEEG that were based on the treatment protocols outlined in the methods section (Figures 4–6).

FIGURE 5. *Z*-score deviation from the reference population in the theta frequency band in coherence measure of the posttreatment QEEG assessment. *Note*. There are no deviations reaching $\geq \pm 1.96$ *Z*-score deviations.

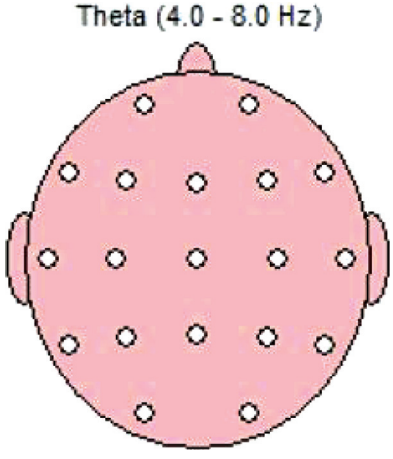
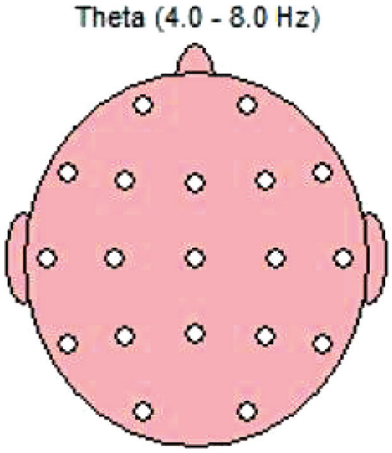


FIGURE 6. *Z*-score deviation from the reference population in the theta frequency band in phase measure of the posttreatment QEEG assessment. *Note*. There are no deviations reaching $\geq \pm 1.96$ *Z*-score deviations.



Results of comparative analysis of QEEG and eLORETA. The comparative QEEG analysis indicated 45 statistically significant electrode differences in the absolute power measures across the six frequency bands (Threshold = 4.3485, $M = -2.68$, Var = 1.955, $SD = 1.398$, Min = −5.82, Max = −0.0863, Skew = −0.744 and Kurt = 2.312), and these findings are summarized in Table 2. There were 23 statistically significant electrode differences in the relative power measures (Threshold = 4.2265, $M = 0.585$, Var = 1.133, $SD = 1.064$, Min = −2.669, Max = 2.686, Skew = −1.162, and Kurt = 5.554), and these findings are summarized in Table 3. The comparative analyses illustrate significant decreases in the absolute and relative power theta measures and significant elevations of absolute and relative power occipital beta.

The eLORETA *t* test resulted in having a significant difference threshold $\geq \pm 3.930$ in two-tailed A<>B at .05 alpha rate. These significant findings are illustrated in Figures 7 to 12. The delta frequency band has maximum decreases in Brodmann area 44, Precentral Gyrus of the frontal Lobe (Figure 7). Theta frequency differences are of significant decreases in the Brodmann area 11, Superior Frontal Gyrus of the Frontal Lobe (Figure 8). Alpha significant

TABLE 2. Absolute power differences.

	FP1	FP2	F7	F3	FZ	F4	F8	T3	C3
Delta	*Ns*	−6.15337399	−6.26773669	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*
Theta	−6.02825324	−7.05885821	−6.64945919	−5.87551967	−5.97341934	−5.72128721	−7.27402189	−4.54031344	−6.76576031
Alpha	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	−5.1575433
Beta	*ns*	*ns*	−6.30666321	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*
Hi Beta	*ns*	−5.00460508	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*
Gamma	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	−6.51526163	*ns*	*ns*

Note. This table illustrates the absolute power differences between the pretreatment and posttreatment conditions. Only those electrodes sites with significant changes are indicated.

TABLE 3. Relative power differences.

	FP1	FP2	F7	F3	FZ	F4	F8	T3	C3
Delta	*ns*	−5.04872398	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*
Theta	−3.85675985	*ns*	*ns*	−3.62174123	−3.96441276	−4.07649496	−5.31917018	*ns*	−4.77796317
Alpha	*ns*	3.84094406	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*
Beta	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*
Hi Beta	4.45290229	*ns*	*ns*	6.07447094	6.03593019	5.33248066	*ns*	3.43085455	6.50302917
Gamma	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	4.14015123

Note. This table illustrates the relative power differences between the pretreatment and posttreatment conditions. Only those electrodes sites with significant changes are indicated.

FIGURE 7. Delta frequency band statistically significant differences. *Note.* Blue indicates significant decreases in the posttreatment condition with the maximum value of −2.009460E + 0001 in the Brodmann area 44, Precentral Gyrus of the frontal lobe.

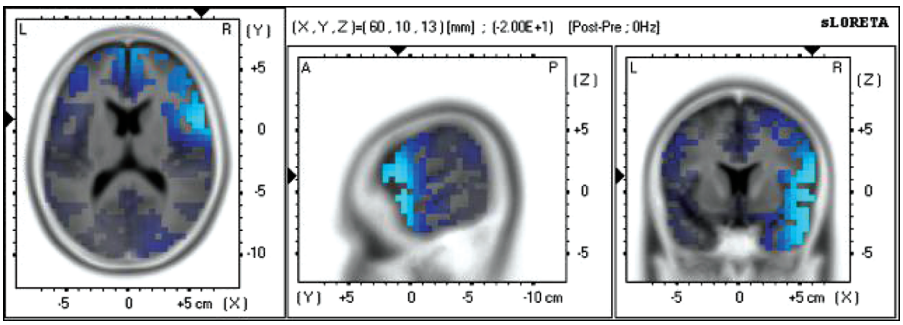


TABLE 2 (continued)

CZ	C4	T4	T5	P3	PZ	P4	T6	O1	O2
ns	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	−5.65391029	−5.41256783	*ns*
−6.22310532	−5.56243719	−7.43671063	−4.84748325	−7.0719101	−6.79843486	−6.87872936	−8.13339241	−8.51014322	−7.04575361
ns	*ns*	0	*ns*	−6.04323457	−4.57666761	−6.84896535	−13.05874874	−9.04087315	−8.19492187
ns	*ns*	−12.62425501	*ns*	−5.46576757	−5.34598589	−6.99595863	−13.26082143	−13.08889818	−6.93773813
ns	*ns*	−7.56183226	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	6.17653946
ns	*ns*	−8.45659387	9.93292111	*ns*	*ns*	6.34128804	7.58067	11.6820586	15.68513751

Nonsignificant sites are noted with *ns*. Most notable were the statistically significant decreases of theta frequency band.

TABLE 3 (continued)

CZ	C4	T4	T5	P3	PZ	P4	T6	O1	O2
ns	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*
−4.15468938	−3.46908631	*ns*	*ns*	−4.12551815	−4.16542578	*ns*	*ns*	*ns*	*ns*
ns	*ns*	4.11663762	*ns*	*ns*	*ns*	*ns*	−6.21770858	*ns*	−5.44374429
ns	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*
6.11145179	4.62142319	*ns*	4.53394304	6.37833166	5.46571097	6.33038313	8.81517458	7.90121416	9.96777544
ns	*ns*	−3.85760161	10.02346881	7.93135593	6.6253515	9.36212672	12.84094275	12.80182152	13.2932869

Nonsignificant sites are noted with *ns*. Most notable were the statistically significant decreases of theta frequency band.

FIGURE 8. Theta frequency band statistically significant differences. *Note*. Blue indicates significant decreases in the posttreatment condition with the maximum value = −1.83E + 1 in the Brodmann area 11, Superior Frontal Gyrus of the Frontal Lobe.

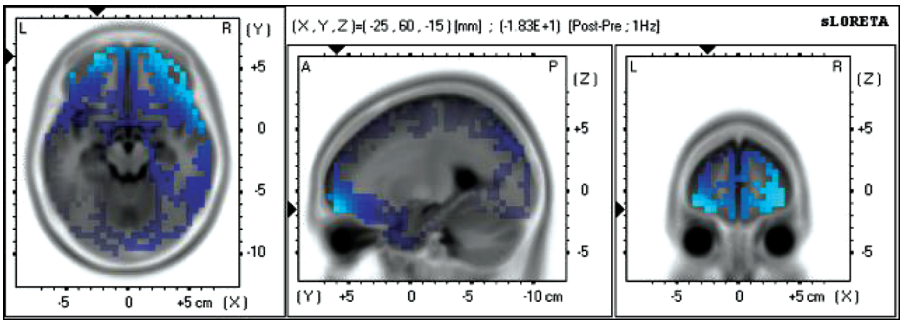
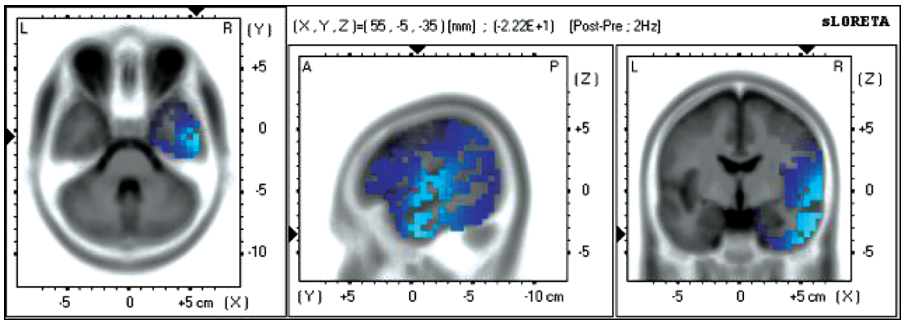


FIGURE 9. Alpha frequency band statistically significant differences. *Note*. Blue indicates significant decreases in the posttreatment condition with the maximum value = −2.22E + 1 in Brodmann area 20, Inferior Temporal Gyrus of the Temporal Lobe.



decreases were found in Brodmann area 20, Inferior Temporal Gyrus of the Temporal Lobe (Figure 9). Low beta and high beta significant decreases were found in Brodmann area 40, Inferior Parietal Lobule of the Parietal Lobe (Figures 10 and 11). Finally, the gamma frequency band has both differences of increases in Brodmann area 18,

FIGURE 10. Low beta frequency band statistically significant differences. *Note*. Blue indicates significant decreases in the posttreatment condition with the maximum value = −2.81E + 1 in Brodmann area 40, Inferior Parietal Lobule of the Parietal Lobe.

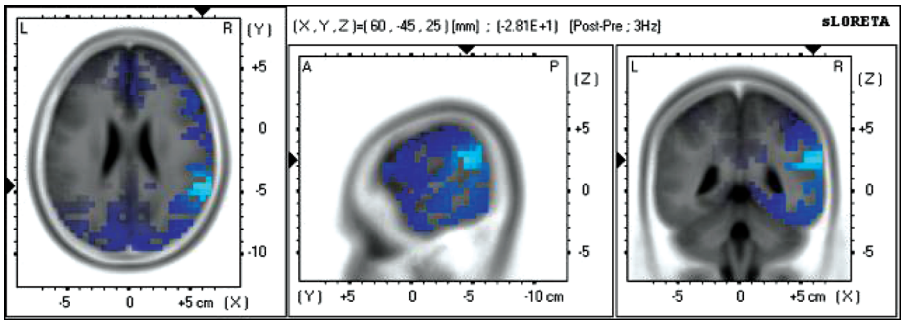


FIGURE 11. High beta frequency band statistically significant differences. *Note*. Blue indicates significant decreases in the post-treatment condition with the maximum value = − 1.53E + 1 in Brodmann area 40, Inferior Parietal Lobule of the Parietal Lobe.

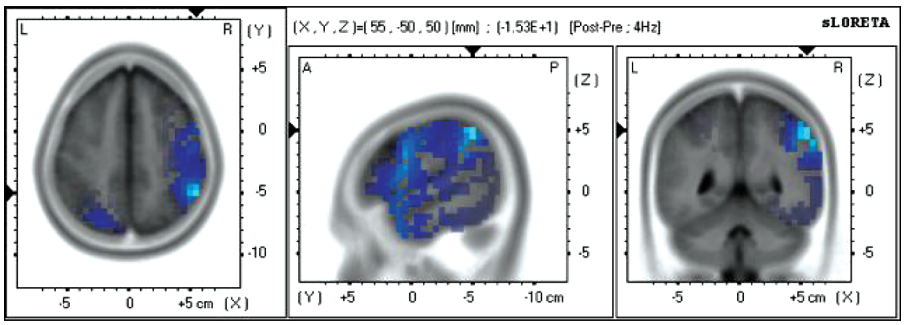
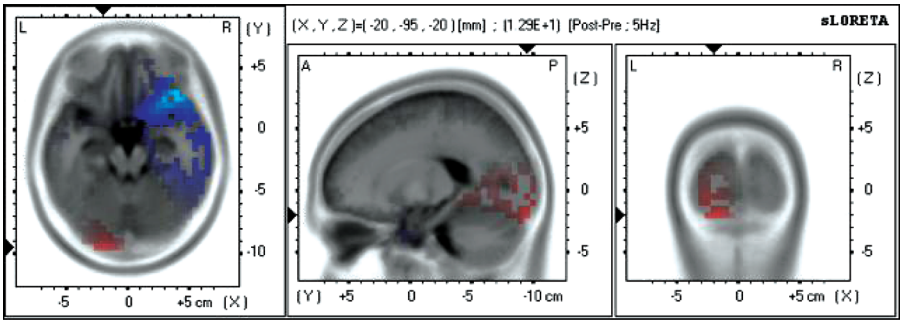


FIGURE 12. Gamma frequency band statistically significant differences. *Note.* Red indicates significant increases in the posttreatment condition with the maximum value = 1.29E + 1 in Brodmann area 18, Fusiform Gyrus of the Occipital Lobe. Blue indicates significant decreases in the posttreatment condition with the maximum value = −1.76E + 1 in Brodmann area 45, Inferior Frontal Gyrus of the Frontal Lobe.



Fusiform Gyrus of the Occipital Lobe and decreases in Brodmann area 45, Inferior Frontal Gyrus of the Frontal Lobe (Figure 12).

DISCUSSION

Following 6 months of regular neurofeedback therapy the participant self-reported significant improvement in cognitive-processing tasks, physical dexterity, and emotional regulation following her stroke the year before. She went from being unable to maintain the stamina needed to participate in weekly soccer games and cycling to being highly active both mentally and physically. Further she was able to completely titrate off her antidepressant with no lingering mood disturbance. Of particular relevance is that 3 weeks following the end of her treatment, the participant chose to travel alone to France to participate in a bicycling tour alongside the Tour de France. Though at times challenging, the participant made lengthy rides through mountainous terrain during the span of the 2 weeks she was there. For her efforts riding up the Cole de Romme, she was voted by her fellow tour group participants as the ''Most Determined Rider,'' a distinction she was immensely proud of. For this particular client the process of understanding how a stroke could happen to someone young and otherwise healthy was a large part of her healing process. Though we do not know the specific cause of her stroke, we did find that her desire to get better and willingness to try neurofeedback certainly made a large contribution to where she is today.

The correlation of symptoms, psychophysiology, previous literature findings and QEEG analysis provided an insightful training protocol. It should be heavily emphasized that the training protocol was not solely based upon standard deviations from the reference populations, but it was a synthesis of the findings from the clinical interview, the symptoms checklists, and all of the data acquired from the QEEG analysis including nonreference population findings. It is a broad understanding of the EEG and QEEG data and the incorporation of this information that provided an individualized and specific training protocol that was believed to maximize the intervention. We would like to point out that there were many other statistically significant findings that were not addressed, because we believed these findings to be less related to the presenting symptom picture, based on the current understanding of the client's injury and psychophysiology.

The QEEG findings and changes in symptom presentation are directly correlative to the occurrence of decreased theta across the cortex. They also illustrate both decreased slowing and increases of beta activity in cortical areas that are corroborative of increased cognitive processing ability (Sherlin, 2008). The eLORETA comparative findings were very intriguing, especially

when considering the QEEG findings. There were significantly more differences in the posttreatment eLORETA analysis showing decreases in all of the frequency bands analyzed with the exception of some occipital elevations of gamma, which we are certain had minimal or no contribution from EMG. Consistent with improvements demonstrated in the post-treatment interviews is the decreases of delta frequency in Brodmann's area 44. Delta is most commonly associated with decreased cortical activation and is reflective of a restorative state such as sleep. Brodmann's area 44 is the motor cortical area in the posterior part of the inferior frontal gyrus, more commonly known as Broca's area and is involved in the production of language.

The decreases in theta are consistent across both QEEG and eLORETA analysis. The maximal decrease in theta amplitude and current source density is in Brodmann area 11, which is the associational cortical area in the orbital-medial prefrontal region of the frontal lobe. This area is primarily involved in prefrontal cortical networks that regulate personal and social behavior, emotion, and decision making. Also not present in the QEEG comparison were the decreases in the alpha frequency band (Williams, White, & Mace, 2005). There were significant decreases in Brodmann's area 20, located in the inferior temporal gyrus and involved in the analysis of visual form and the representation of objects. Most unexpected were the decreases of beta in Brodmann's area 40, which is involved in spatial orientation and semantic representation (Williams et al., 2005). Although these findings were not present in the QEEG analysis, which alternatively indicated occipital increases, the beta decreases are hypothesized to be attributed to the increased efficiency of other areas involved in language production and increased connectivity resulting in a more efficient utilization of cortical activation and the relinquishing of the Brodmann's area 40 for these tasks.

Noticeably, four of the significant eLORETA findings were frontally related. We did not utilize any protocols that were specifically targeting frontal areas. However, the frequency band changes were involved in protocols implemented. This was a reminder to us of the complicated networks involved in brain functions. Although we may desire to target regions of the brain that are most correlative with decreased functioning or with the most significant aberrations, we should not forget that the brain is a system and we are never training only a local region.

Regardless of the specifics of location, frequency, amplitude, or statistical variability, the most important fact in this case is the remarkable improvement in quality of life and abilities for the individual. For this particular individual, neurofeedback therapy was the key to returning her pleasure in living.

REFERENCES

American Heart Association. (2009). *Heart disease and stroke statistics: 2009 update*. Dallas, TX: Author.

Bearden, T. S., Cassisi, J. E., & Pineda, M. (2003). Neurofeedback training for a patient with thalamic and cortical infarctions. *Applied Psychophysiology and Biofeedback*, *28*, 241–253.

Brett, M., Johnsrude, I., & Owen, A. M. (2002). The problem of functional localization in the human brain. *Nature Reviews Neuroscience*, *3*, 243–249.

Claassen, J., Hirsch, L. J., Kreiter, K. T., Du, E. Y., Connolly, E. S., Emerson, R. G., et al. (2004). Quantitative continuous EEG for detecting delayed cerebral ischemia in patients with poor-grade subarachnoid hemorrhage. *Clinical Neurophysiology: Official Journal of the International Federation of Clinical Neurophysiology*, *115*, 699–710.

Collins, D. L., Neelin, P., Peters, T., & Evans, A. (1994). Automatic 3D intersubject registration of MR volumetric data in standardized Talairach space. *Journal of Computer Assisted Tomography*, *18*, 192–205.

Congedo, M. (2005). *EureKa!* (Version 3.0). Mesa, AZ: Nova Tech EEG, Inc.

Frei, E., Gamma, A., Pascual-Marqui, R., Lehmann, D., Hell, D., & Vollenweifer, F. X. (2001). Localization of MDMA-induced brain activity in healthy volunteers using low-resolution brain electromagnetic tomography (LORETA). *Human Brain Mapping*, *14*, 152–165.

Hammond, D. C. (2006). *An introduction to neurofeedback. International Society for Neurofeedback & Research*. http://www.isnr.org/uploads/whatisnfb.pdf

Holmes, A. P., Blair, R. C., Watson, J. D. G., & Ford, I. (1996). Nonparametric analysis of statistic images

from functional mapping experiments. *Journal of Cerebral Blood Flow and Metabolism*, *16*, 7–22.

John, E. R., Prichep, L. S., & Easton, P. (1987). Normative data banks and neurometrics: Basic concepts, methods and results of norm construction. In A. Remond (Ed.), *Handbook of electroencephalography and clinical neurophysiology: Vol. III. Computer analysis of the EEG and other neurophysiological signals* (pp. 449–495). Amsterdam: Elsevier.

Jorgensen, H., Nakayama, H., Raaschou, H., Vive-Larsen, J., Stoier, M., & Olsen, T. (1995). Outcome and time course recovery in stroke. Part II: Time course of recovery. The Copenhagen study. *The Archives of Physical Medicine and Rehabilitation*, *76*, 406–412.

Lightstone, H. (2009). *EEG spectrum systems* (Version 4.2.2), Los Angeles, California.

Neau, J. P., Ingrand, P., Mouille-Brachet, C., Rosier, M. P., Couderq, C., Alvarez, A., et al. (1998). Functional recovery and social outcome after cerebral infarction in young adults. *Cerebrovascular Diseases*, *8*, 296–302.

Nelson, L. A. (2007). The role of biofeedback in stroke rehabilitation: Past and future directions. *Topics in Stroke Rehabilitation*, *14*(4), 59–66.

Nichols, T. E., & Holmes, A. P. (2002). Nonparametric permutation tests for functional neuroimaging: A primer with examples. *Human Brain Mapping*, *15*(1), 1–25.

Niedermeyer, E., & Lopes da Silva, F. (2004). *Electroencephalography: Basic principles, clinical applications, and related fields* (5th ed.). Philadelphia: Lippincott, Williams & Wilkins.

Nuwer, M. R., Jordan, S. E., & Ahn, S. S. (1987). Evaluation of stroke using EEG frequency analysis and topographic mapping. *Neurology*, *37*, 1153–1159.

Pascual Marqui, R. D. (2007). *Discrete, 3D distributed, linear imaging methods of electric neuronal activity. Part 1: exact, zero error localization*. arXiv:0710.3441v2 [math-ph]. Retrieved from http://arxiv.org/pdf/0710.3341

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*(1), 49–65.

Pascual Marqui, R. D., Pascual-Montano, A., Lehmann, D., Kochi, K., Esslen, M., Jancke, L., et al. (2006). Exact low resolution brain electromagnetic tomography (eLORETA). *Neuroimage*, *31*(Suppl. 1), S86.

Putman, J. A. (2001). EEG biofeedback on a female stroke patient with depression: A case study. *Journal of Neurotherapy*, *5*(3), 27–38.

Rozelle, G. R., & Budzynski, T. H. (1995). Neurotherapy for stroke rehabilitation: A single case study. *Biofeedback & Self-Regulation*, *20*, 211–228.

Sherlin, L. (2008). Diagnosing and treating brain function through the use of Low Resolution Electromagnetic Tomography (LORETA). In T. Budzynski, H. K. Budzynski, J. Evans, & A. Abarbanel (Eds.), *Introduction to quantitative EEG and neurofeedback, advanced theory and applications* (2nd ed.) (pp. 83–102). Amsterdam: Elsevier.

Sherlin, L., Budzynski, T., Budzynski, H. K., Congedo, M., Fischer, M. E., & Buchwald, D. (2007). Low-resolution electromagnetic brain tomography (LORETA) of monozygotic twins discordant for chronic fatigue syndrome. *NeuroImage*, *34*, 1438–1442.

Szelies, B., Mielke, R., Kessler, J., & Heiss, W. D. (2002). Prognostic relevance of quantitative topographical EEG in patients with poststroke aphasia. *Brain Language*, *82*(1), 87–94.

Talairach, J., & Tournoux, P. (1988). *Co-planar stereotaxic atlas of the human brain*. New York: Thieme.

Thatcher, R. W., Walker, R. A., Biver, C., North, D., & Curtin, R. (2003). Quantitative EEG normative databases: Validation and clinical correlation. *Journal of Neurotherapy*, *7*, 87–122.

Towle, V. L., Bolanos, 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.

Williams, S. M., White, L. E., & Mace, A. C. (2005). *SylviusVG: Visual glossary of human neuroanatomy*. Sunderland, MA: Puramis Studios, Inc.