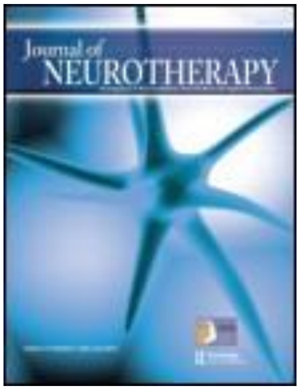

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EEG Biofeedback on a Female Stroke Patient with Depression: A Case Study

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ABSTRACT. *Background*. This single case concerns the treatment of a 71-year-old female stroke patient. The patient's MRI revealed that the location of the stroke was in the right side basal ganglia with damage extending into the anterior limb of the internal capsule. She presented with a virtual paralysis of the left side of her body (hemiplegia with immobilized left arm, contracted fist, minimal motor control over left leg, absence of muscle tonus in left side of face and slurred, monotonic speech).

Method. The client was provided with EEG biofeedback training on a one to two half-hour sessions per week schedule. Bipolar montages were used along with single site protocols. This was based largely on the idea of reciprocal communication loops between widely separated cortical generators. It was thought that encouraging communication between cortical sites would have a beneficial impact on impairments related to both functional and structural damage. EEG training protocols included SMR (12-15 Hz) enhancement at C4, C4-Pz and T3-T4 with theta suppression; beta (15-18 Hz) enhancement with theta suppression at C3, C3-Fpz and at C3-Fp1.

Results. Patient showed significant improvement in gross motor control and range of movement of left arm and leg. The most dramatic improvement was observed in speech (articulation, strength and tone). While substantial improvements were observed in motor ability, restoration of mood stability proved somewhat more elusive. Since she was receiving additional treatment (physical therapy and medication man-

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agement), it is impossible to attribute the improvement in functioning solely to the EEG training. However, the consensus among the attending medical personnel was that the improvements noted above took place with unusual expeditiousness.

Discussion. When performing EEG biofeedback it may be most practical to adopt an "exercise model" approach in which the regulatory mechanisms in the brain are challenged through the sequential use of multiple protocol configurations. In this case several different training protocols proved useful in her ongoing recovery. While improvements in functioning were a result of a concerted effort involving multiple therapeutic interventions, it is likely that neurofeedback played a vital synergistic role.

KEYWORDS. Stroke, post stroke depression, EEG biofeedback, bipolar protocols, EDA

BACKGROUND

Stroke is the third leading cause of mortality and morbidity in the United States with a yearly incidence of between 300,000 and 400,000 (Starkstein & Robinson, 1992). Investigators discovered early on that stroke often resulted in depression (Robinson & Starkstein, 1994). Although it is expected that depression will often follow a stroke due to the impairments in physical and cognitive functioning, post stroke depression (PSD) appears to be more than a reaction to loss of function. Robinson and Starkstein found that the severity of the depression did not show a consistent relationship to the degree of physical impairment. Additionally, there is a significantly increased rate of depression among stroke patients when compared to orthopedic patients with similar levels of impairment, suggesting that mood instability may be related to how the stroke impacts the brain rather than the level of disability. Research has also demonstrated that there is a three-fold greater mortality rate in depressed stroke patients independent of age, type of stroke, lesion location and size (Morris, Robinson, & Andrezejewski, 1993). Additionally, Starkstein and Robinson (1990) reported that patients with cortical lesions in the left basal ganglia had an increased rate of post stroke depression similar to those with left anterior frontal lobe lesions, where injury to the right side can result in manic symptoms.

EEG biofeedback has been used successfully with cerebral vascular accident (CVA) in improving speech fluency, word finding, balance, coordination, attention, depression and anxiety (Rozelle & Budzynski, 1995). EEG training has also proved effective in cases of pediatric stroke. A 1995 study demonstrated that the children receiving the training showed improved range of motion, dorsiflexion of foot, improved concentration, short term memory and mood stability where the control group, receiving only the standard post stroke treatment, showed considerably less improvement (Ayers, 1995).

METHOD

Subject. LM, a 71-year-old woman, was referred by her psychiatrist for EEG biofeedback for treatment of depression related to a stroke she had suffered two months earlier. The patient's MRI revealed that the location of the stroke was in the right side of the basal ganglia with damage extending into the anterior limb of the internal capsule and immediately lateral to the thalamus. The patient suffered an ischemic stroke, which later developed hemorrhagic features. Over 40 percent of ischemic strokes will become hemorrhagic within one to two weeks (Hornig, Dorndorf, & Angnoli, 1986). This is primarily due to the necrosis of the vasculature in the area affected by the obstruction (Welch & Levine, 1991). The basal ganglia are involved with integrating feelings, thoughts, movements and smoothing motor behavior. A malfunctioning or damaged basal ganglia can also play a role in anxiety, negative thinking and moodiness. Although research has indicated that right side strokes impacting the basal ganglia may result in manic symptoms (Starkstein & Robinson, 1990), the client evidenced no symptoms of mania. Additionally, hemorrhages tend to be more serious than infarctions (obstructions) due to the fact that the basic pH of the blood is very damaging to neural tissue. Upon examination, the client's EEG revealed a substantial degree of theta to beta ratio asymmetry between the right and left hemispheres (2.7 as measured at C4 and 1.6 as measured at C3). Although a quantitative EEG was requested, it was not performed due to a number of situational factors, among them, the client's very limited financial resources and the husband's inability to drive the long distance required to get to a suitably equipped clinic because of his degenerative eye condition. (Both resided in the high desert of California.) LM presented with a virtual paralysis of the left side of her body (immobilized left arm with contracted fist; minimal motor control over left leg; absence of muscle tonus in left side of face and slurred, monotonic speech). She required full time use of a wheel chair and a great deal of assistance when moving from the chair to her bed. Her medications included Remeron (a tetracyclic antidepressant), Ambien (a sedative for sleep) and Xanax (for anxiety) in addition to blood pressure and cholesterol reducing medications. LM had also been receiving physical therapy since shortly after her stroke. Upon initiation of treatment LM conveyed a profound sense of hopelessness and fear regarding the extent of recovery she would achieve. Despite these feelings, LM was very motivated to try EEG training and received a great deal of support and encouragement from her husband. Thus, LM's depression likely had multiple causes: reaction to the stroke and subsequent paralysis as well as disruption of basal ganglia functioning leading, perhaps, to an alteration in arousal threshold and mood stability.

Instrumentation. EEG biofeedback equipment manufactured by EEG Spectrum was used, consisting of two sets of instrumentation, employed at different points in the treatment. Initially, the BioIntegrator was used. The BioIntegrator is an eight-channel multimodality biofeedback system manufactured by the Bio Research Institute with two channels dedicated to the EEG measure. The system uses a battery-operated encoder (four AA 1.5 volt nickel-cadmium batteries) with a fiber optic cable for data transmission (the ProComp from Thought Technology). The EEG sampling rate was set at 200 per second. The raw EEG was fed through a sliding window with signal analysis performed using Fast Fourier Transforms (FFTs). FFTs were performed 10 times per second on the sampled data. Total bandwidth was 0.8-40 Hz with a frequency resolution of .8 Hz. The system provided both audio and visual feedback. The audio feedback varied in pitch directly with the magnitude of the reward band and inversely with the inhibit band. In order to reduce the possibility of confusion, the pitch of both the reward and inhibit frequencies were programmed to rise together when the desired EEG pattern was being produced (i.e., increased midrange frequency and decreased low frequency activity). Although the 1996 version of the BioIntegrator software does not provide for theta inhibition per se, it does have an audio feedback polarity inversion feature. As such, it was easy to configure a theta frequency band audio inhibit due to the fact that the feedback is analog and not binary (i.e., it is not contingent on meeting threshold criterion). Since theta inhibition is considered important in achieving a successful treatment outcome in cases of cortical disregulation and has been employed with success in treating stroke victims, augmenting the feedback with a theta inhibit was deemed necessary (Ayers, 1995;

Rozelle & Budzynski, 1995). The video portion of the feedback took the form of a multi-colored mandala. The intensity of the mandala varied directly with the amplitude of the reward band, which in this case was 12-15 Hz. Theta activity was not visually fed back to the client as the "mandala graph" can only display one frequency at a time, which reflected the reward band only. The scale was set to 0-30 microvolts where the screen turned dark at 0 µV and was completely filled at 30 µV. At approximately the 20th session a switch was made to Neurocybernetics instrumentation. This was due to both an equipment availability issue and Neurocybernetics' emphasis on the use of inhibits.

The Neurocybernetics system uses digital filtering for signal processing with roll off rates of 12 db per octave at the low end and 30 db per octave at the high end. Signal amplification was set at 10,000. Input impedance for each of the two channels was 100 meg-ohms. The sampling rate was 160 per second. The filtered EEG trace and the three filtered waveforms were displayed in a continually scrolling fashion for monitoring by the therapist. Amplitude inhibition was in both the high beta (22-30 Hz) and the theta (4-7 Hz) bands. The reward bands used were 12-15 Hz, 15-18 Hz and 11-14 Hz. Upon digital filtering in the PC, the signal was then mapped into a video game format through a second computer with a video screen for viewing by the client. The second video screen displayed a box lights-like game in which the size of each of the three boxes (representing each of the filtered frequency bands) varied directly with the maximum amplitude in its associated band. Auditory feedback was binary in that the client would hear a tone only when all threshold criterion were met.

PROCEDURE

The treatment choices in this case were based on the protocols developed by the Othmers through their extensive clinical experience (Othmer, 1999). Their work, in turn, is based on the research of Barry Sterman with his emphasis on training at the sensorimotor cortex due in part to the density of thalamocortical connections that exist there (Sterman, 2000). Accordingly, right side training at C4 was used primarily for the treatment of anxiety, agitation and anger. Left side training at C3 was used primarily for treating depression and a lack of motivation. Nearly all training protocols included one of these two sites. EEG training montages included SMR (12-15 Hz) enhancement at C4 and C4-PZ with theta suppression; beta (15-18 Hz) enhancement with theta suppression at C3, C3-Fpz and C3-Fp1. Note that beta suppression at 22-30 Hz was employed only after the 20th session when a switch was made to the Neurocybernetics system. (The '96 version of the BioIntegrator software does not feature inhibits.) T3-T4 with 12-15 Hz or 11-14 Hz reward and theta inhibition was also used. All training site locations were in accordance with the International 10-20 system. All protocols were grounded on the ear with reference on the proximal ear of the signal electrode for single site training. A protocol switch to the left side was made following a worsening of her depressive symptoms (increased feelings of hopelessness, sadness and early morning awakenings) sustained for three days or more that did not appear to be related to situational factors. Similarly, a training location switch to the right side was made following an increase in anxiety (generalized fear and worry inappropriate to her immediate situation along with poor sleep onset). For example, the client would become extremely anxious when anticipating a medical exam. In addition, it was suggested (by S. Othmer) that augmenting C3 by placing the reference sensor at Fp1 or Fpz could enhance the effect of left side training on depression. Likewise, moving the reference to Pz when training at C4 may prove more stabilizing when training down anxiety. T3-T4 was also suggested for increasing mood stability. The general rationale for using bipolar protocols stems, in part, from the intrinsic differences between long versus short-range neural connections as stated in the "two compartmental" model of coherence. In this model, originally proposed by Braitenberg (1978), there are both short distance and long distance neural connections in the brain. The short distance system typically involves connections on the order of millimeters to a few centimeters. The long distance system involves interactions that occur over several centimeters. The critical difference between these two systems is that the long distance communication tends to require reciprocal feedback loops while the short distance networks tend to transmit their signal by the process of diffusion (Thatcher, 1998; Thatcher, Krause, & Hrybyk, 1986; Pasqual-Marqui, Valdes-Sosa, & Alvarez-Amador, 1988; Braitenberg, 1978; Braitenberg & Schuz, 1991). Thus, when performing single site training, we are likely inducing a more localized form of coherence, whereas with bipolar training we are more likely facilitating communication between cortical centers via subcortical linkages requiring greater orchestration by the thalamic regulatory centers. This may result in a greater degree of neural differentiation of function in the long term, leading, possibly, to a more complete recovery. All protocol changes took place on an inter-session basis in that there were no changes made within a given session.

Additional measures were temperature and EDA (electrodermal activity) to monitor peripheral vasodilation and sympathetic nervous system arousal. Electromyography (EMG) was used to monitor neuromuscular activity. These measures were employed during the first 20 sessions only and were not fed back to the patient but were used exclusively for monitoring purposes by the practitioner. EMG sensors were placed on the extensor muscles of the paralyzed left forearm. The temperature thermistor was placed on the index finger of the right hand with the EDA sensors placed on the third and fourth finger of the right hand. (The EDA measure was abandoned early on.) EMG measures were used primarily for the purpose of monitoring changes in activity in the area with the greatest degree of motor impairment when the patient was asked to initiate movement at the end of the session. The exclusive focus of treatment was EEG training. The patient was provided with EEG biofeedback one to two times per week (less than what was recommended) with session duration varying between 30 and 35 minutes.

RESULTS

It is very difficult to separate the relative contributions of physical therapy, neurofeedback and medication management to the overall improvement in the patient's functioning. However, her recovery appeared to take place expeditiously according to the physical therapists and medical professionals who deal with stroke patients on a regular basis.

Initial observation of the peripheral measures (temperature and skin conductance) seemed to indicate a high degree of sympathetic nervous system arousal, which is more consistent with anxiety than depression. The characteristic signature of anxiety is decreasing temperature and increasing skin conductance (Gilbert, 1986). Within eight sessions of EEG biofeedback training, peripheral temperature was sustained at greater than 92 degrees for most of the session. Baseline temperatures for the first several sessions were between 88 and 89 degrees. Treatment was initiated using C4 due to the apparent degree of high sympathetic nervous system arousal and the client's overt symptoms of anxiety (excessive worry and fearfulness). While C3-Fpz was used with overall success, it proved inconsistent with respect to alleviating LM's depression. This was less true for C3-Fp1, which appeared to be more effective in lifting her mood. Although the primary goal of the EEG training was to alleviate her depression and stabilize her anxiety, there were substantial improvements in motor control as well. LM appeared to make the most significant gains in motor control with right side training and T3-T4 although it is difficult to attribute any of her gains to a specific protocol. It is likely that all played a role in improving her level of functioning.

0-15 sessions. LM's mobility improved significantly and she is able to climb (with assistance) the 13 steps to her upstairs bedroom for the first time since her stroke. By session 15, she is taking daily 10-minute walks in the park using a walker with support from her husband. She also began setting the table for dinner for the first time since her stroke. The protocols used were: C4 with 12-15 Hz reward and 3.5-7.4 Hz inhibit. The instrumentation was the BioIntegrator.

20-25 sessions. LM showed improvement in speech articulation and an increase in facial muscle tone. She demonstrated greater motor control as evidenced by a smoother gate and greater range of movement of the left arm as well as increased thumb pressure and pull strength in upper arm. The psychiatrist noted marked improvement in speech and motor control. In LM's case, C4 SMR appeared to have the greatest effect on speech recovery. The apparent reason for this is that LM's speech difficulties were due primarily to poor neuromuscular control of the mouth and tongue due to damage to the motor control centers of the right side basal ganglia rather than damage to the actual speech centers in the left temporal area of the brain. The protocols used were: C4, 12-15 Hz reward and 4-7 Hz inhibition; C3-Fpz, 15-18 Hz reward, and 4-7 Hz and 22-30 Hz inhibition. The instrumentation was Neurocybernetics.

25-30 sessions. She continued to increase strength and range of motion in left upper arm. Additionally, LM is more animated and reports her depression, while continuing to vary from week to week, is less severe. The physical therapist reported "very pleased" with her progress. On session 28 she walked, with assistance, into the office for the first time without using her wheelchair. The protocols used were: C3, C3-Fpz with 15-18 Hz reward and 4-7 Hz and 22-30 Hz inhibition.

30-35 sessions. LM eliminated sleep medication and reduced antianxiety medication by 50%. She was able to walk backwards (a much more complex motor task) with aid of parallel bars for the first time in physical therapy. Her speech was nearly back to normal with respect to articulation and tone (less monotonic). She showed marked increase in facial muscle tone on left side. The rehabilitation hospital newspaper voted her "Most Improved Stroke Patient." LM's physical therapist and psychiatrist confirmed all improvements. The protocols used were: C3 with 15-18 Hz reward and 4-7 Hz and 22-30 Hz inhibition.

40-65 sessions. She was able to stand without support. The protocols used were: C4-Pz with 12-15 Hz reward; C3-Fpz, C3-Fp1 with 15-18 Hz reward. All with 4-7 Hz and 22-30 Hz inhibition.

65-70 sessions. LM was able to walk (approximately 10 steps) under her own power without support and move toes for the first time. Her mood was generally more stable although situational factors continue to cause a re-emergence of depressive symptoms. She was having more days where she felt like her "old self." The protocols used were: T3-T4 with 12-15 Hz reward and 4-7 Hz and 22-30 Hz inhibition.

An examination of the EEG trends at sites C3, C4, C3-Fpz, C4-Pz, T3-T4 revealed no particular patterns with respect to individual band amplitude averages or theta/beta ratios although there appeared to be a decrease in the inter-session variability on the right side.

In May 2001, exactly three years from the time of her stroke, LM underwent a muscle joint function re-evaluation at the Center of Achievement for the Physically Disabled on the campus of California State University, Northridge. The purpose was to assess the degree of improvement in muscle strength and range of motion measured since her initial evaluation at the same facility five months following her stroke in 1998. Table 1 represents pre- and post-treatment values in different aspects of left side motor functioning.

DISCUSSION

While the patient has shown marked improvement in speech and many aspects of her motor functioning, stabilization of her depression has proven somewhat more elusive. Although she has days when she feels good and is optimistic about her future, her mood continues to be somewhat easily derailed by seemingly minor situational stressors. Restoring stability in this regard has been only modestly successful. This may be due to a number of factors including: ongoing changes in her medication regimen, life circumstances, and the relatively intractable nature of the depression that often follows a stroke (Robinson & Starkstein, 1994).

However, LM did show substantial improvement in the motor control and muscle strength of her left arm and hand, particularly in the thumb, wrist, elbow and shoulder. Marked gains were also noted in movement and strength of the left knee with more modest gains ob-

TABLE 1

FUNCTION (Left Side)	Strength Rating (pre/post)	Range of Motion
Finger Flexion–Extensors	1/3	
Thumb Flexion–Extensors	0/2	
Wrist Flexion–Extensors	0/2	
Wrist Extension–Flexors	0/2	N
Supination–Pronators	0/2	N
Elbow Flexion–Extensors	0/2	worse
Shoulder Adduction–Abductors	1−/2	
Shoulder Abduction–Adductors	1−/0	
Shoulder Horiz Flex–Extend	1+/2	
Shoulder Horiz Extend–Flex	1−/0	N
Shoulder Int.–Ext. Rotation	0/2	
Cervical Rotation–Rotators	4/5	
Hip Flexion–Extensors	1/2	N
Hip Extension–Flexors	2+/3	N
Hip Int.–Ext. Rotation	1/0	N
Hip Ext.–Int. Rotation	1/2	
Knee Flexion–Extensors	1/0	
Knee Extension–Flexors	2+/4	
Eversion–Invertors	0/0	N
Meta Phal Extension–Flexors	0/0	N

The Muscle Strength rating scale: (0) zero, (1) trace, (2) poor, (3) fair, (4) good, (5) normal. Gradations are further refined with minus (−) or plus (+).
Range of Motion: Range of motion that has improved and is no longer considered “limited” is indicated by an “N” (for within normal limits).

served in the rotational capability of the hip socket. It would be difficult to isolate the gains produced solely by the EEG training from those resulting from the other treatment interventions. It is likely, however, that brainwave training provided something of a catalytic “push” thereby making traditional interventions more productive as evidenced by not only the degree of improvement observed but also the expeditiousness with which this improvement took place according to the attending physical therapists and medical personnel.

At our present level of knowledge it is difficult to say in every case whether up training or down training a specific set of frequencies is clearly called for. It may therefore be most practical to adopt an "exercise model" approach in which the brain's regulatory systems are sequentially challenged through the utilization of different protocols thereby increasing flexibility and efficiency of function. In cases of head injury and stroke, our task as neurofeedback practitioners is to bring as many sectors of the neural network back online as possible. It is assumed that with any head trauma or cerebral vascular accident (CVA) involving structural damage to the brain, there is a fair amount of functional damage as well. Certainly any insult that is capable of producing structural damage will likely disrupt the regulatory centers that manage the sequencing and timing of neural communication. The field of EEG biofeedback is now moving into new territory, which will most certainly call for a methodological exploration of new training protocols. This new territory is being defined more by the people in need of help who are turning to us in desperation than any deliberate attempt by practitioners to widen the market place for EEG biofeedback. As such, the greater utilization of both single site and bipolar training protocols may be of significant benefit in addressing not only the attentional, cognitive processing irregularities and affect disturbances resulting from stroke or head injury, but motor impairments as well.

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