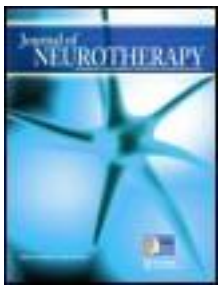

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Role of Quantitative Electroencephalography, Neurotherapy, and Neuroplasticity in Recovery from Neurological and Psychiatric Disorders

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NEW PERSPECTIVES

Role of Quantitative Electroencephalography, Neurotherapy, and Neuroplasticity in Recovery from Neurological and Psychiatric Disorders

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ABSTRACT. *Introduction*. Until recently, patients with brain injuries had poor prognosis for recovery, but new insights into neuroplasticity and neurorehabilitation have significantly improved outcomes. Neurotherapy or neurofeedback is one of those promising techniques for neurorehabilitation.

Methods. Neurofeedback or EEG biofeedback, as it is also called, uses operant conditioning to reinforce desirable self-regulated changes in EEG rhythms, changes that are believed to correspond to reorganization in neural networks, particularly in thalamocortical and corticothalamic circuits. Sensorimotor rhythm reinforcement has been effective in facilitating recovery in patients with traumatic brain injury, stroke, seizures, and certain sleep disorders.

Results. We describe the case of a 19-year-old man with severe, partial secondarily generalized seizures that did not respond to extensive conventional treatments including all antiepilepsy drugs.

Conclusion. He underwent two 3-week sessions of daily neurotherapy, which produced remarkable EEG and behavioral normalization.

KEYWORDS. Brain rehabilitation, neuroplasticity, neurotherapy, quantitative electroencephalography, seizures

INTRODUCTION

Knowledge about brain function, neuroplasticity, and rehabilitation along with advances in technology and brain imaging have provided us with new insights into psychiatric and neurological disorders. The efficacy of integrative approaches to diagnosis and treatment with improved outcomes in many disorders has promoted noninvasive

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strategies for treatment. One such noninvasive learning-based approach is neurorehabilitation by stimulating the visual cortex with neurodevelopmentally appropriate stimuli (Malkowicz, Myers, & Leisman, 2006). Another approach is the use of quantitative electroencephalographic (QEEG)-guided neurotherapy or neurofeedback. Both of these noninvasive strategies have in common the ability to reintegrate neural circuits and networks resulting in the return of associated function. Scientific studies behind EEG neurotherapy, a form of biofeedback, have been in progress since the late 1960s (e.g., Kamiya, Callaway, & Yeager, 1969; Wywricka & Sterman, 1968). Recent advances in technology and the recognition of brain neuroplasticity have presented us with the opportunity to use such methods for diagnosis and treatment of a number of neurological and psychiatric disorders (e.g., Hughes & John, 1999). Such approaches are in a state of evolution in terms of technology, techniques, and treatment protocols. In this article we discuss a specific approach to treating seizure, QEEG-guided neurotherapy, a technique that requires a keen understanding of neurophysiology, EEG interpretation, operant conditioning, and a working understanding of the principles of neurology and psychiatry. Multiple studies are in progress determining the type of approach and treatment protocols that would be most effective for specific disorders (e.g., Beauregard, 2009; Hoedlmoser et al., 2008), and we believe success requires cooperation among investigators working together on rigorous ongoing studies for this approach to be accepted by the various medical and scientific communities serving epileptic patients.

TECHNICAL AND CLINICAL CONSIDERATIONS

QEEG-guided neurofeedback is based on analysis of mathematically-processed digitized EEG data obtained from participants in awake and increasingly neurologically (cognitively) engaged states. The pretraining EEG is recorded and analyzed for brain activity and compared with individuals in the same maturational state (based on age) during similar challenges such as rest or silent reading. EEG voltages were acquired with a 19-channel digital EEG system with electrodes positioned according to the International 10–20 System with linked-ear reference electrodes and ground electrodes. Electrode impedances are matched and kept at an optimal range between 1–5 KOhms. For out study we used the Neuronavigator system (J & J Engineering Inc., Poulsbo, WA) for EEG acquisition, and data were recorded during eyes closed rest, eyes open rest, reading, math and other specific task states to analyze the EEG background. Each condition was approximately 3 min in length with any signs of drowsiness eliminated. Standard EEG activation procedures such as hyperventilation or photic stimulation were not performed in our example.

EEG recorded by scalp electrodes is generated by postsynaptic potentials. Excitatory postsynaptic potentials and inhibitory postsynaptic potentials, recorded from cortical pyramidal neurons are under the influence of input from oscillating thalamocortical circuits (e.g., Steriade, 2005; Valdes-Sosa et al., 2008). An action potential occurs when the sum of excitatory postsynaptic potentials and inhibitory postsynaptic potentials reach threshold. An exchange of positive and negative ions occurs across neuronal membrane ion channels. The neurotransmitter released at the synapse can be inhibitory or an excitatory, depending on the neuron. The neurotransmitter binds to postsynaptic receptors and causes an excitatory or inhibitory response. Changes in the ionic milieu around neurons and glia can affect firing potential. Medications or other substances can alter neurotransmitter formation, release, binding, uptake, or degradation and thereby alter neuronal firing. The ability to alter this environment and the critical interactions between neurons through biofeedback, albeit grossly, is very exciting for the fields of psychiatry and neurology.

Proper interpretation of the EEG relies on dipole localization. EEG recordings are the electrical potential difference between electrodes. Electrical dipoles (negative–positive charge units) which are radially oriented and closer to the surface are best represented

on the EEG. Activity occurring farther away from scalp electrodes may be detected poorly or not at all, which includes activity arising in deeper gray matter structures, within deeper sulci, fissures, or in mesial or basal areas of the brain. Even activity at the surface of the cortex is poorly seen, if the dipoles occur at unfavorable angles to the recording electrodes (Niedermeyer & Lopes da Silva, 2004). Also, artifacts can arise from physiologic sources such as eye movement, muscle movement, chewing swallowing, and even EKG or pulse activity, as well as from other outside nonphysiological electrical fields or interference. Nonphysiologic artifact occurs from electrical contamination of the EEG from other electrical fields in the environment.

Normal EEG contains a mixture of age-appropriate background activities at appropriate frequencies and voltages in all states and has normal variability and reactivity. There are no focal or generalized abnormalities. Abnormal features tend to disturb the background, emerge from abnormal areas, and persist in location and throughout states associated with other EEG or clinical findings. EEG abnormalities can be diffuse or focal. White matter injury tends to manifest with focal or diffuse slowing such as paroxysmal delta activity. Grey matter injury tends to manifest while loss or slowing of frequencies or reduction in their amplitude (e.g., Ebersole & Pedley, 2003).

Interpretation of the QEEG record requires knowledge of the age and state of the patient. Medication effects or withdrawal states influence EEG activity. Medications such as benzodiazepine, barbiturates, antiepilepsy medications, and psychiatric drugs can significantly alter EEG background or cause paroxysmal activity. Normal EEG looks different in individuals of different ages and in the same individual in different states. Abnormalities may be episodic and might not show up on the EEG (e.g., Malkowicz, 2006).

NEUROTHERAPY AND NEUROPLASTICITY

Many assumptions underlie the practice of neurofeedback. The brain is both structurally and functionally plastic, and conditioning and altering EEG rhythms facilitates changes in this plasticity, the neural circuitry. The ability to adapt, learn, and reinforce specific EEG rhythm under proper conditions can make lasting changes in the generation and regulation of selected brain neural circuits and thalamocortical loops underlying the activity that we record as EEG. Sterman (1976, 2000) focused on the appearance and learned control of an EEG rhythm over the sensorimotor cortex, which emerged above nonrhythmic low-voltage background activity. He and his colleagues named this the sensorimotor rhythm (SMR) and it was characterized by a rhythm of 12–19 Hz in cats, with a spectral peak around 12–14 Hz. In a series of experiments Sterman and colleagues investigated whether operant conditioning methods could work on the recorded EEG (e.g., Sterman, Howe, & Macdonald, 1970; Sterman, Wyrwicka, & Howe, 1969). They discovered that the experimental animal, the cat, could voluntarily produce SMR. This had an important clinical significance. Studies in which the cats that had taken part in SMR training were found to have a significantly elevated seizure thresholds, compared to the thresholds of untrained cats, when exposed to a highly epileptogenic fuel compound (Sterman, Fairchild, & McRae, 1972). After extensive study, it was noted that the SMR constitutes the dominant frequency of the integrated thalamocortical, somatosensory, and somatomotor pathways. The operant training of SMR resulted in improved control in overexcitation in this system with increased thresholds for excitation and is thought to underlie the clinical benefit of SMR training in epilepsy. This seems to be true in other disorders characterized by cortical and/or thalamocortical hyperexcitability, such as attention deficit hyperactivity disorder (e.g., Lubar & Bahler, 1976). Further more studies in children, with attention deficit hyperactivity disorder using functional Magnetic Resonance Imaging (fMRI), support the idea that those who improved significantly in cognitive tests, after neurofeedback training, have had an observed significant increase in metabolic activity in the striatum (Lévesque,

Beauregard, & Mensour, 2006). This result suggests that facilitation and/or regulation of SMR substrates alters motor output. This can set up reduced proprioceptive afferent input to the thalamus and suggests reorganization of motor and thalamic status accompanied by volleys of strong oscillating discharges to the cortex with each trained SMR response.

The neuroplastic changes associated with neurofeedback training include changes in oscillatory activity, which we know are influenced by neurotransmitter activity, such as cholinergic and monoaminergic neuromodulators, which affect excitability levels. Changes in neuromodulatory influences and cortical projections affect cellular depolarization and bursting patterns. Increased metabolic activity noted in specific areas of the brain, increased synaptic strength in relevant neural circuits, increased protein synthesis, and insertion of new excitatory transmitter channels at postsynaptic receptor sites result in long-term potentiation, increasing synaptic sensitivity, and the probability of future activity in neuronal circuits (e.g., Meador, 2007). Another example of EEG oscillation is the Post Reinforcement Synchronization seen after reward delivery. The physiological reward for meeting EEG criteria is referred to as Post Reinforcement Synchronization, which produces a brief burst of EEG activity (Marczynski, Harris, & Livezey, 1981). This burst of EEG activity results in drive reduction and feelings of satisfaction and achievement. This satisfies the requirements of neuroplasticity, resulting in changes in the EEG toward normalization. These changes have been seen in fMRI studies (Beauregard & Lévesque, 2006; Lévesque et al., 2006).

In addition to spectral magnitude or power, EEG connectivity parameters also exist such as coherence, comodulation, phase lag, and others factors. Connectivity is a property of neural circuits and their functional network. Properly integrated neural network would be expected to operate together appropriately under various conditions. Even the relationship of one electrode site can be compared to these around it in task conditions. Network parameters compare EEG signal characteristics between sites and represent a variety of ways to display and analyze comparative signal variation, and thereby reflects various functional relationships between neural networks.

Basis for QEEG-Guided Neurotherapy in Treatment of Refractory Epilepsy After Traumatic Brain Injury

A seizure is a paroxysmal, abnormal electrical discharge from cortical neurons under the influence of oscillating thalamocortical circuits. Depending on the location of the focus the seizure can result in a change in awareness or consciousness, motor, sensory, or ''psychic'' behavior, or a combination of these. These seizures can arise from a discrete focus (partial seizures) or generalized seizure activity involves the whole brain mediated by thalamocortical circuits. Regardless of the seizure type, the discharge alone involves thalamocortical areas and cortex circuits that can sustain abnormal activity. There is often post-ictal state during which the patient gradually returns to awareness and function as neurons recover. The corresponding EEG can show varying degrees of slowing and suppression during the recovery stage (Ebersole & Pedley, 2003).

Epilepsy is a condition characterized by recurrent, unprovoked seizures or a disorder of the brain with the tendency for recurrent unprovoked seizures. Treatment options include medications, epilepsy surgery, and vagal nerve stimulation, to name a few of the more common and traditional approaches.

Neurotherapy reinforces the desired EEG activity over time, as the patient generates the desired EEG frequency, maintaining it above a certain threshold for a certain period. The reward can also require that specific EEG sites are acting in common or simultaneously by training connectivity parameters. This training is thought to reintegrate thalamocortical circuits in the specific areas of training. The task can also be structured to have certain brain areas firing in a desired pattern. The goal is to

normalize the EEG by producing normal frequencies where there was a lack of normal activity, or an area of abnormal activity. It also allows the clinician to have areas of the brain work in proper synchrony. Reinforcing a frequency trains up the frequency making it more likely to occur and become permanent over time with multiple repetitions. The clinician can also decide to train down or inhibit abnormal EEG activity by setting parameters that rewarding the patient for reducing threshold magnitude of certain frequencies or eliminating abnormal activity. In most cases the clinician is monitoring various parameters occurring at the electrode training sites on a separate therapist screen, which can display EEG frequency bands, thresholds, and other factors in the ongoing EEG. This allows the clinician to adjust the parameters required to receive a reward during the session. For example, as the patient becomes more proficient at producing the desired frequency, duration, and threshold, the variables such as threshold can be adjusted to make it more challenging to receive the reward. This is called shaping, and it allows the patient to increase the ability to produce the desired responses over time, yielding a more effective session. Site by site and session by session the desired neural circuitry is built and reinforced until the changes in EEG background are permanent and the desired behavior is achieved.

METHOD

Participant

This is the case of a 19-year-old male participant who had a severe, partial secondarily generalized seizures that failed all antiepilepsy drugs and other conventional forms of therapy. The epileptic focus was thought to be left temporal lobe or multifocal in onset. He would remain in a somnolent post-ictal state for days following a single seizure, and he was more impaired in all areas of function. On average, he could sleep for only 2 hours a night.

Ten years ago he had closed head traumatic brain injury due to a motor vehicle accident, after which he was in a coma for 18 months. He had neurological deficits that did not completely resolve despite various types of rehabilitation programs. He still had significant impairment in neurological functions, relatively unchanged in the 4 years prior to entering the Neurotherapy Protocol for the Institute for the Achievement of Human Potential. He was unable to ambulate independently because of poor motor control of axial and lower extremity muscles, as well as having moderately severe incoordination and spasticity. He could not pronate or suppinate his hands or use his fingers, which were held in a spastic posture. He was unable to articulate due to a motor aphasia and had severe difficulty chewing and swallowing. Because of these deficits he was unable to be independent in any activity. In addition to this regularly occurring partial secondarily generalized seizures continued to create setbacks in neurological function.

He was 29 years old when he entered the Institute for the Achievement of Human Potential's QEEG-Neurotherapy Seizure Protocol, 10 years after traumatic brain injury and 4 years without significant improvements despite daily, intensive rehabilitation programs. Prior MRIs showed severe generalized cortical atrophy worse in the frontal-temporal areas with compensatory lateral ventricular dilatation and a left temporal horn dilatation greater than the right temporal horn. Prior EEGs and the pretraining QEEG showed prominent rhythms in the delta and theta range in all brain areas, especially the frontal-temporal central regions. There were no normal patterns such as a posterior dominant rhythm or SMR. No epileptiform activity was seen.

Materials and Procedure

During neurotherapy training, the patient sits in a comfortable chair, such as a recliner, and watches a video display. Scalp electrodes are placed at the sites to be trained, as determined by the QEEG analysis. Appropriate reference and ground electrodes are also used. All electrodes are isolated from any possible source of electricity. This

is accomplished using optical isolators. In the Use3 system by J&J Engineering it is possible to simultaneously train four separate cortical locations, each in three different frequency bands, if required by the individual's treatment protocol. The neurotherapy training system rewards the patient when their EEG activity meets the preset criteria simultaneously at all electrode locations being trained. To be effective, the visual and auditory rewards are contingent on the correct response and occur discretely and immediately following the desired response. A postreward pause in the program of about 2 sec follows, to allow the information to be optimally processed. The patient can be rewarded for training up certain desirable frequencies, called *reinforcement*. Reinforcement makes it more likely that the desired activity will be produced in the again. The patient can be rewarded for training down undesirable frequencies. This is called *inhibition*. There are typically between one and three groups of cortical locations that are trained during a session. The EEG criteria must be maintained for a period of 250 to 500 msec to receive a reward. The reward is in the form of a change in the video display and an auditory tone. The visual display is often in the form of a piece of a picture puzzle. Repeated rewards allow completion of the picture, and the process is then repeated. In this operant conditioning protocol, the visual and auditory events or "rewards" are contingent on the patient producing desired responses and this increases the likelihood of future successful responses.

During Session 1, after his initial clinical evaluation, he had a QEEG evaluation. The EEG for pretraining QEEG Session 1 was obtained using a 19-channel electrode cap. EEG samples were obtained in various states of progressive brain engagement. Two samples were obtained in the eyes closed condition and the eyes opened condition. Two samples were also obtained while performing mathematical tasks and reading. The QEEG was analyzed using Sterman-Kaiser Imaging Laboratory analysis with the EEG analyzed by Fast Fourier transformation and other mathematical transformations yielding records in referential bipolar Laplacian and Horth montages. The resultant data displayed in topometric anlysis, spectral plot, and brain map images were compared to age- and state-matched normative and statistical databases. Brain mapping included comodulation, coherence, and Brodmann's area mapping. The results showed predominantly delta frequencies in all brain areas, particularly frontal, temporal, central, and parietal regions with a moderate amount of theta in the same distribution (see Figures 1–3). This highly abnormal

FIGURE 1. Statistical deviation of 1-Hz bands from healthy adults during eyes closed baseline reveals excessive slow activity prior to neurotherapy training.

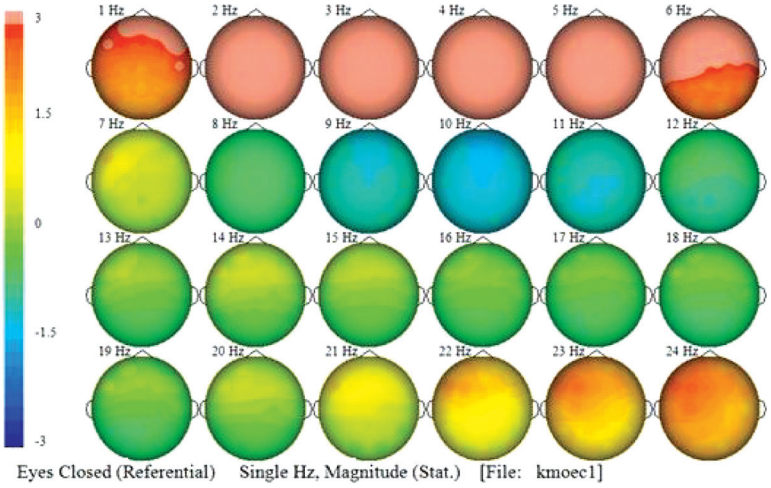
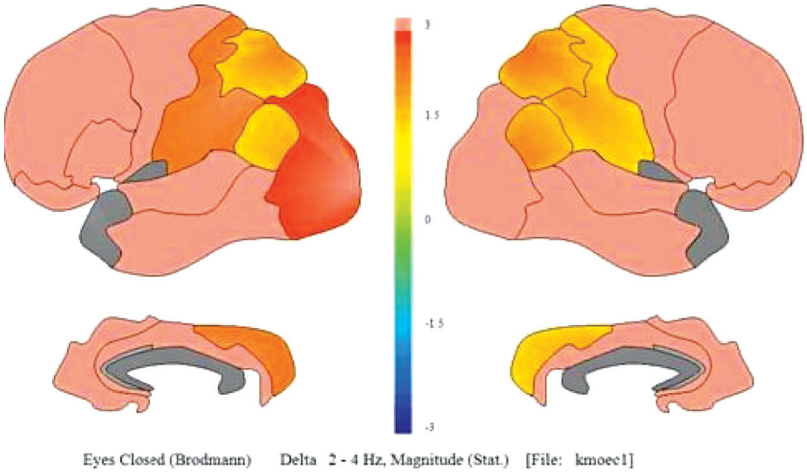


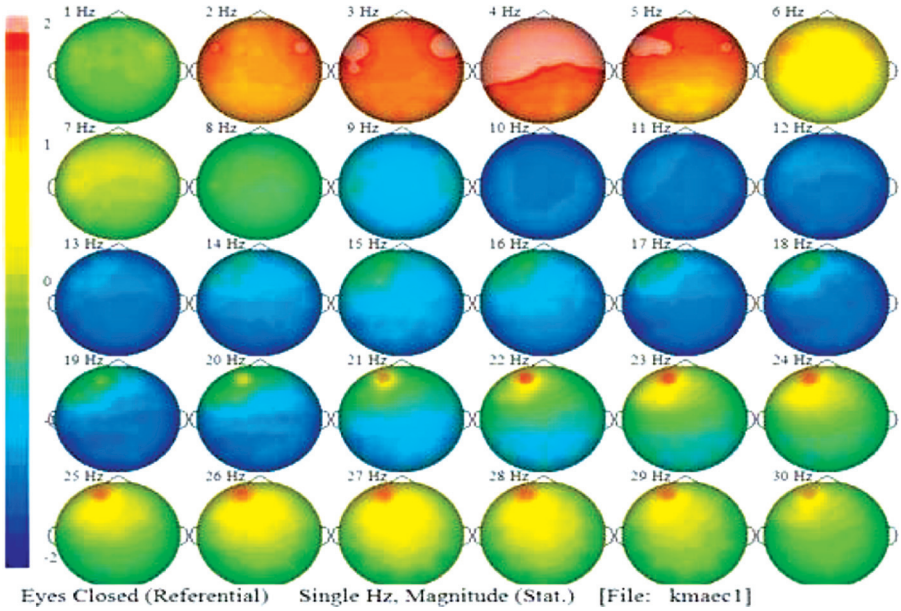
FIGURE 2. Statistical deviation of delta activity in a current source density map from healthy adults during eyes closed baseline reveals excessive delta activity in most Brodmann areas prior to neurotherapy training.



finding was consistent with severe brain injury involving white matter, gray matter, and the associated thalamocortical circuits. No normal frequencies or pattern such as SMR or posterior dominant rhythm (PDR) were seen.

After the Session 1 pretraining QEEG a neurotherapy (neurofeedback) program was designed based on his QEEG analysis. This program's goal was to reinforce SMR 12–15 Hz over central (C3 and C4) electrode sites, sensory motor area. As this was achieved, the program progressed to reinforcing 8.6–10.6 Hz alpha rhythms in the frontal, temporal, central, and parietal areas. This was reinforced with operant conditioning, a reward of a pleasant tone, and visual feedback was obtained when the desired

FIGURE 3. Excessive slow frequency after 3 weeks of daily neurotherapy has been attenuated to include fewer sites and fewer frequencies.



frequencies were maintained. In addition, the patient could see an ongoing readout of his EEG correlating to the areas of training and was instructed to maintain the desired frequencies above a certain threshold to reinforce the production of that rhythm.

The first neurofeedback program began with reinforcement of SMR 12–15 Hz at sites C3 and C4, gradually building up to approximately 1 hr per day. As success was achieved at that site other adjacent areas were added. These would include frontal, temporal, central, and parietal area electrodes. The sites that were first used for up training, or reinforcement, of 8.6 to 10.6 Hz rhythms were those surrounding C3 and C4 sites. The participant maintained at EEG frequencies at threshold for 0.5 sec. When this was achieved he received a neurofeedback reward. New electrode sites were added daily and the program expanded to train C3, C4, SMR, as well as frontal, temporal, central, and parietal areas at 8.6–10.6 Hz. As the participant achieved goals, the sessions were made more challenging as the clinicians were monitoring all electrode sites on a linked therapists screen on which the could monitor three frequency and threshold bands at four electrode site at one time. As the participant achieved a certain number of rewards the program was made more difficult, thus shaping and reinforcing his responses.

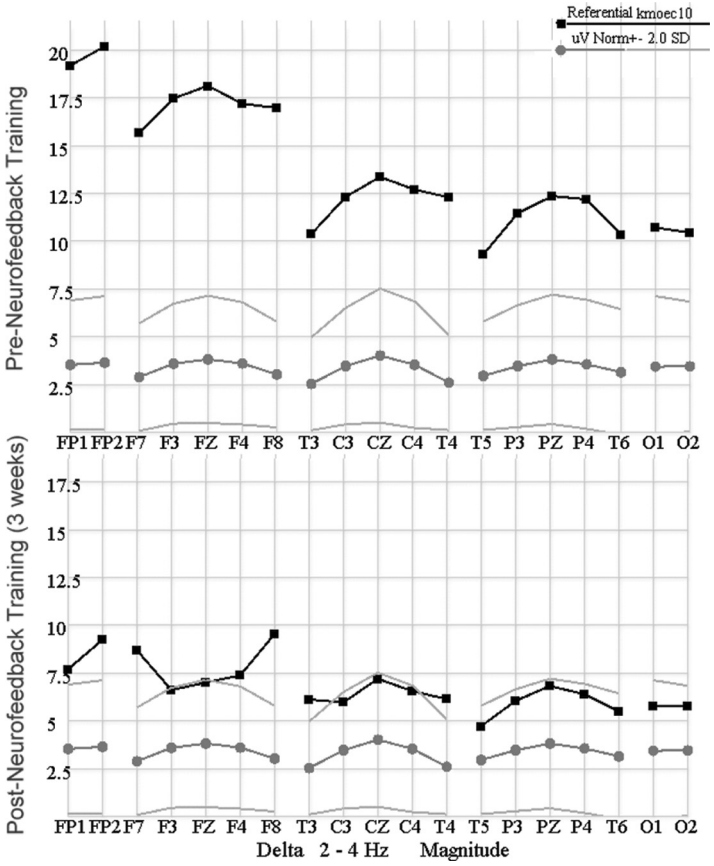
RESULTS

After 3 weeks of daily neurotherapy the patient could pronate and suppinate his hands and demonstrated fine motor control of his fingers. Axial stability and upper and lower extremity motor control and coordination improved significantly. He required less assistance in walking. His speech articulation improved 20 to 30% and his dysphasia improved by 50%. He previously slept only 2 hr per night and now slept for 8 hr uninterrupted every night. His seizures were less frequent and much less severe with return to pre-ictal baseline within minutes. Session 1 posttraining QEEG showed that the delta and theta activity decreased from as much as 10 standard deviations above normal values to 2 standard deviations in all areas as seen on the topometric analysis (see Figures 3 and 4). SMR and alpha were seen in the recording for the first time.

The patient returned home for 5 months, during which time he had no further QEEG neurotherapy or rehabilitation. On his return for Session 2 of neurotherapy it was noted that he maintained all of his neurological improvements including motor status and sleep pattern. He had a few, brief partial seizures without secondary generalization and no significant post-ictal sequela in that time. During QEEG-Neurotherapy Session 2, his pretraining QEEG showed the improvements noted on his posttraining Session 1 training QEEG. His second QEEG-Neurotherapy session consisted of 3 weeks of daily neurofeedback programs. The pretraining QEEG showed no loss of EEG function in the interim of 5 months. A similar neurotherapy program was designed emphasizing SMR 12–15 Hz reinforcement at C3 and C4. The 8.6 to 10.6 Hz activity was reinforced in the frontal, temporal, central, and parietal regions (electrode sites F3, F4, T3, T4, P3, P4, T5, T6, F7, F8). During Session 2, he continued to gain motor control and enhanced his fine motor skills and coordination. Additional improvements in articulation and swallowing were also noted. He had no seizures off of all antiepilepsy drugs. He could walk more independently, including climbing stairs. His second session posttraining QEEG showed that his delta and theta were at normal levels and that he had gained a significant amount of SMR and alpha in the appropriate brain areas (see Figures 5 and 6).

A QEEG-Neurotherapy intensive treatment program aimed at correcting deficits seen on the QEEG, such as lack of SMR and alpha and an overabundance of delta and theta, was successful in establishing and maintaining better motor control and coordination. Speech and swallowing improved significantly. The participant had fewer seizures, and they were much less severe. Nocturnal sleep was normalized in amount and improved in quality. The emphasis on SMR training 12–15 Hz reinforcement was

FIGURE 4. Statistical improvement of delta activity at all electrode sites during eyes closed resting baseline after 3 weeks of daily neurotherapy.



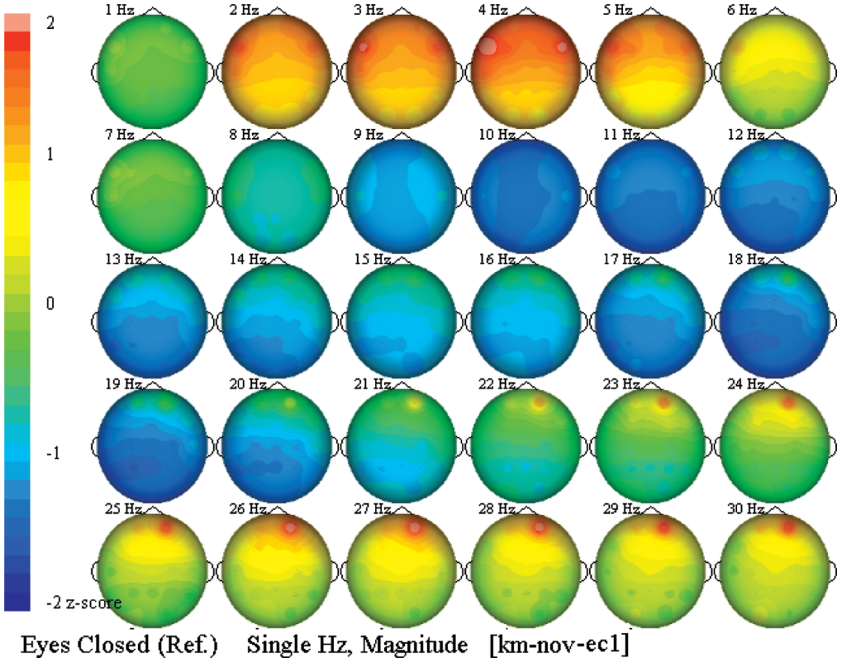
probably key to many of the improvements seen in Session 1, that is, motor control, reintegration of sleep, and control of seizures. This would be expected given the basic neurophysiology involved in SMR generation. The maintenance of gross and fine motor control and coordination between sessions without further training suggests that once reintegrated neurocircuits are in place, activity of daily living will continue to reinforce or, perhaps, improve those areas.

After the second session, he returned home without further QEEG-Neurotherapy or rehabilitation programs but continued to make gains in fine motor abilities, especially in his hands and fingers, which allowed him to type and play the guitar, walk, and function independently. On his next visit in 5 months it is hoped that he will show no loss and possibly some gains in motor function because of ongoing use of his motor skills. This is consistent with reintegration of thalamocortical circuits, especially in motor sensory areas that occur with neurotherapy training. Once the neurocircuitry exists, ability appears to be maintained. This seems to indicate that a significant amount of neuroplasticity exists, even 10 years after traumatic brain injury, and that functional recovery can be achieved with proper rehabilitation, in this case, a protocol using QEEG-Neurotherapy.

DISCUSSION

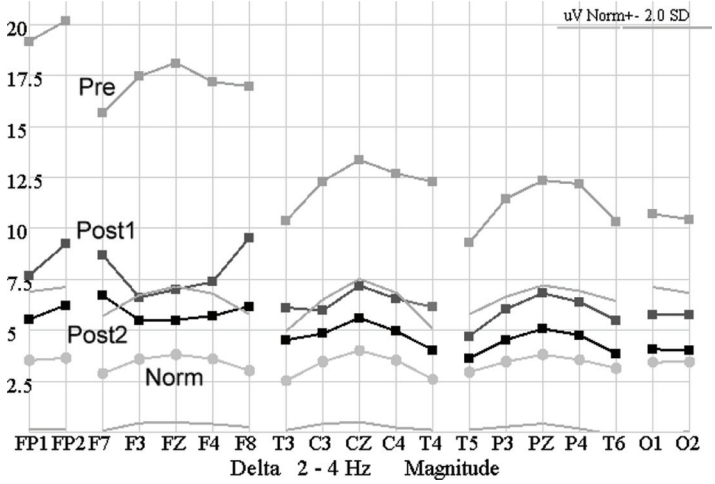
There is more than 40 years of research into EEG operant conditioning (Malkowicz, 2009; Sterman et al., 1969). In a series of experiments involving operant conditioning

FIGURE 5. Statistical improvement at all electrode sites during eyes closed resting baseline after 3 weeks of daily neurotherapy.



with a food reward, cats were trained to voluntarily control SMR. The EEG self-regulation and behavior associated with SMR production was one of corporal immobility, with SMR bursts regularly proceeded by a drop in muscle tone (Sterman et al., 1969; Wyricka & Sterman, 1968). Subsequently, Sterman's experiments on dose response to seizure of an epileptogenic compound demonstrated that cats having prior SMR conditioning training had significantly elevated epileptic thresholds

FIGURE 6. Statistical improvement of delta activity at all electrode sites during eyes closed resting baseline after 3 weeks of daily neurotherapy (Post 1) and further improvement after another period of 3 weeks of daily neurotherapy (Post 2).



compared with untrained cats. This suggested that SMR training had kept the SMR animal from experiencing seizures (Sterman et al., 1969). Further studies by Sterman and others showed that the SMR rhythm originates from the thalamus in the ventrobasal nuclei (nVB; Howe & Sterman, 1972). The nVB is concerned with conducting afferent somatosensory information. Further studies showed that during conditioned SMR production, nVB firing patterns shift from tonic, fast, and nonrhythmic discharges to systematic, rhythmic burst of discharge, which in turn are associated with suppression of somatosensory information passage and reduction of muscle tone (Howe & Sterman, 1973). It was demonstrated that upon reduction of afferent somatosensory input, the cells in the nVB thalamus hyperpolarize. Studies showed that instead of remaining at a stable level of inhibition, a gradual depolarization mediated by a slow calcium influx causes the neurons of the nVB to discharge a burst of spikes. These spikes are relayed to the sensorimotor cortex and to the neuron of the thalamic reticular nucleus. Subsequently, stimulation of the thalamic reticular nucleus leads to a GABAergic inhibition of nVB relay cells, thus returning them to a hyperpolarized state. This initiates a new cycle of slow depolarization, thus the interplay between neuronal populations in the thalamic nVB, thalamic reticular nucleus and the sensorimotor cortex results in rhythmic thalamocortical volleys and consequent cortical EEG oscillations (Sterman, 2008; Steriade & Morin, 1981). Several situations can affect SMR. Attenuation of efferent motor and afferent somatosensory activity can initiate SMR. The oscillatory activity is also largely influenced by nonspecific cholinergic and monoaminergic neuromodulation, which can affect excitability level both thalamic relay nuclei and in the cortical areas receiving the relay signals.

SMR constitutes the dominant frequency of the integrated thalamocortical, thalamosensory, and somatomotor pathway. Thus operant conditioning of SMR is assumed to result in improved control over excitation in this system. Increased thresholds for excitation in turn are thought to underlie the clinical benefits of SMR training in epilepsy (Sterman, 2008). Over the years, numerous studies have been conducted by various investigators into the use of QEEG-Neurofeedback and/or SMR training for the treatment of seizures. In many cases, refractory partial seizures were studied. In others studies patients with partial seizure and generalized seizures were investigated.

It is crucial to understand whether the EEG we acquire and analyze is an accurate reflection of the neurophysiology of normal and pathological conditions under consideration or whether some features are epiphenomenon, which are not relevant. This requires coordinated ongoing scientific and clinical research into normal and condition specific EEG databases. Work advancing QEEG analysis, such as, appropriate references for data collection and advanced mathematical analysis of data continue to progress (Coben & Hudspeth, 2008; Joffe, 2008; Kaiser, 2008, 2009). The various neurotherapy strategies for treating conditions noted on QEEG need to be further analyzed. A coordinated multidisciplinary approach to this research is an asset to help understand whether the changes seen on QEEG with neurotherapy are effectively altering the neurophysiology of the brain in appropriate ways, and how these changes are occurring, to optimize treatment of each underlying condition.

SUMMARY

Neuroplasticity is the intrinsic ability of the brain to change itself in response to input, resulting in learning and formation of related functional neural networks. In the case of brain injury or dysfunction, neuroplasticity allows for the reintegration and formation of neural networks. This process can take place with relevant stimulation and learning experiences over time. However, it appears that this can be significantly enhanced, in terms of extent of recovery and time to recovery, when the individual receives an appropriate neurorehabilitation program.

QEEG-neurotherapy applies operant conditioning principles, rewarding and reinforcing

desirable self-regulated EEG activity and inhibiting or suppressing undesirable activity. The changes in EEG toward normalcy appear to reflect changes in underlying neurophysiology with reintegration of neural circuits and networks. Clinically, this is linked to changes in neurological, psychiatric, or cognitive function.

Our protocol was different in that we used an intensive treatment model to more rapidly and thoroughly reinforce neurocircuits changes instead of intermittent session of 20 min, we used daily session progressively increasing the complexity and amount of time spend in the neurotherapy. We started with SMR reinforcement, which was the center piece of each day's neurotherapy, but we continued to add adjacent and functionally involve areas to the neurotherapy. As each area showed improvement or normalization we added its homologous or adjacent functional electrode area to the protocol. We did not try to train down slow frequencies but rather trained up faster frequencies in the slow areas, because his QEEG showed only slowing (delta and theta). These seem to accelerate the process of normalization within a single session at certain electrodes were monitored for delta, theta, and alpha but regarded for producing alpha. We see that change moment by moment in the EEG from that area decreasing delta and theta and increasing alpha. SMR continued to be trained at each session. Our approach differs from many done at that time because we emphasis training normal frequencies in to until the area was independent in producing normal frequency and continued daily to reinforce the circuits as they required and forming other functional circuits as well.

One of the features that these strategies have in common is that they appear to work through utilizing neuroplasticity, retraining the brain, building neural circuits and networks. Other similar features are that these therapies are self-directed, are noninvasive, and lack any adverse side effects. Some of the questions that remain to be answered are, How do these process take place, and how can we influence these processes to yield maximum benefit for recovery? Do intensive neurotherapy protocols, emphasizing SMR reinforcement, provide the opportunity for long term potentiation in thalamocortical circuits allowing for the rapid, robust and self-regenerating change demonstrated in this patients? This requires ongoing cooperative studies of population of participants with these particular disorders. Studies in QEEG-Neurotherapy need to be cared out by investigators who have expertise in EEG, neurofeedback, operant condition, neurology, neurophysiology, and psychiatry. Investigators in QEEG-Neurotherapy face the challenge of designing appropriate diagnostic and treatment protocols that can yield benefits. These studies can help standardize approaches to QEEG-Neurotherapy protocol for diagnosis and therapy. The future of these emerging strategies is in cooperative scientific research. The future of neurorehabilitation is to harness the brain's neuroplasticity to optimize recovery.

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