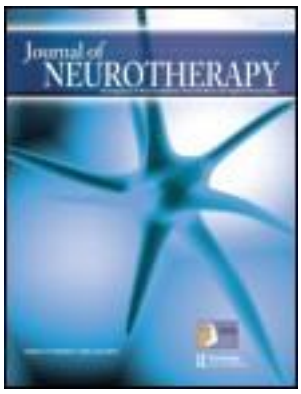

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QEEG Guided Neurofeedback to Treat Schizophrenia: A Case Study

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QEEG GUIDED NEUROFEEDBACK TO TREAT SCHIZOPHRENIA: A CASE STUDY

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The subject in this case study was a 21-year-old man who had to leave college at the beginning of his senior year after being diagnosed with adult onset schizophrenia, undifferentiated type. For 18 months, the only interventions the subject utilized were varying doses of aripiprazole and intensive neurofeedback training. Data derived from five serial quantitative EEGs identified statistically significant coherence abnormalities that appeared as the schizophrenia progressed. Neurofeedback training to enhance cortical stability and normalize neural connectivity was directed by the convergence of data from each quantitative EEG and clinical assessment. Results showed a dramatic increase in functional abilities and a decreased need for medication. The subject resumed his challenging college curriculum and graduated. However, he then stopped neurofeedback and, in spite of increased medication, became floridly psychotic. This study supports other research showing neurofeedback training may be a useful treatment in the management of schizophrenia.

INTRODUCTION

Presently there is no cure for schizophrenia, a disorder that typically yields chronic disability affecting an estimated 24 million people worldwide. Although medications help manage the disorder, antipsychotics are only one part of a comprehensive treatment regimen. Schizophrenia research is aggressively exploring the specific mechanisms involved in the onset of the disorder with a view toward prevention while also supporting studies that show efficacy in treating the symptoms of the disorder. Treatments that effectively reduce symptoms of schizophrenia may also hold clues to the specific molecular signaling process that begins the deterioration in brain connectivity during critical periods for high-risk individuals. With evidence accumulating that neurofeedback has the potential to be a beneficial intervention for schizophrenia, this study presents the results of the quantitative EEG (qEEG) directed neurofeedback treatment of a single subject in the early stages of schizophrenia.

Various neuroimaging techniques have supported the long-held hypothesis that symptoms of schizophrenia result from disconnection syndromes (Skelly et al., 2008). Using MRI data to measure cortical thickness, Zhang et al. (2012) found that the structural networks of schizophrenic patients had less optimal topological organization resulting in reduced capacity to integrate information across brain regions compared to normal controls. fMRI studies show that schizophrenics have connectivity deficits between right insular subregions and the central executive/default mode network (Moran et al., 2013). Lynall et al. (2010), based on fMRI studies, concluded that persons with schizophrenia tend to have a less strongly integrated, more diverse profile of brain functional connectivity that is associated with a less hub-dominated configuration of complex brain functional networks. Also using fMRI, a team of neuroscientists led by Vaubhav Diwadkar at Wayne State University School of Medicine found that children at risk for schizophrenia are characterized by reduced

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network communication and disordered network responses to emotional faces. Diffusion tensor imaging studies indicate that the symptoms of the disorder are associated with differential white matter fractional anisotropy values in specific association fibers. After a review of the literature, Ruiz, Birbaumer, and Sitaram (2013) concluded that neuroimaging consistently leads researchers to conclude that the key impairments associated with the onset and progression of schizophrenia result from a progressive failure of the brain to integrate activity in local and distributed neural circuits. This was termed the abnormal neural connectivity hypothesis of schizophrenia, a failure mode consistent with what Norman Geschwind (1965) called the ''disconnection syndrome.''

Neurofeedback training has shown efficacy in remediating disconnection syndromes (Schummer, 2008) through application of coherence training (Coben & Padolsky, 2007; Walker, Kozlowski, & Lawson, 2007), thus remediating a key neurophysiological impairment associated with schizophrenia. Angelo Bolea (2010) provided 130 neurofeedback sessions to adult schizophrenic inpatients over an 18-month period. Most were discharged into the community and continued to show improvements after 2 years. Ruiz et al. (2013), using fMRI neurofeedback, reported a reduction in symptoms for subjects with schizophrenia after they were trained to enhance brain connectivity via self-regulation of insula activity. The largest controlled study to date by Surmeli, Ertem, Eralp, and Kos (2012) reported that 47 outpatient schizophrenic subjects who completed individualized qEEG derived neurofeedback training showed significant improvement on measures as divergent as the Positive and Negative Syndrome Scale (considered a positive treatment response measure for pharmaceutical trials), the Minnesota Multiphasic Personality Inventory, and the Test of Variables of Attention. Forty subjects were followed for nearly 2 years and continued to show positive effects from the neurofeedback training; 19 of the subjects no longer qualified for a diagnosis of schizophrenia, 27 did not require medication, and the majority of those who required medication were maintained at a reduced dosage with polypharmacy nearly eliminated.

The subject in this case study was a 21-year-old male college student diagnosed with adult onset schizophrenia, undifferentiated type. Due to cognitive impairments related to the onset of this disorder, he had to take a leave of absence during his senior year in college. He was given a very poor prognosis by his psychiatrist and placed on aripiprazole (Abilify) with the dose varying between 5 mg and 20 mg. No other therapy or intervention was utilized during the course of this study other than neurofeedback training and medication.

METHOD

Neurofeedback training was guided by data derived from qEEG testing administered five times over an 18-month period. Low Resolution Brain Electromagnetic Tomography (Pascual-Marqui, Michel, & Lehmann, 1994), NxLink (John, Prichep, Fridman, & Easton, 1988) and NeuroGuide (Thatcher, 1998) databases were utilized to discern the type of neurofeedback to be applied, the cortical sites for sensor placement, and the appropriate frequency bands for training. The training utilized EEG data acquired with the ProComp Infinity amplifier (Thought Technology, Ltd.) with a sampling rate of 2,048 per second in conjunction with EEGer, a computer-based analysis and feedback software driving the amplitude, sum, or coherence modules (EEGer Software, LLC).

With each qEEG, areas that showed statistically significant amplitude and coherence abnormalities were identified for training. The subject engaged in intensive neurofeedback training with four to six 30-min sessions per week totaling 530 in-office sessions. During each session, the subject was provided with visual and auditory feedback when identified criteria (e.g., amplitude levels within particular frequencies, percentage coherence, etc.) were achieved. The convergence between qEEG abnormalities and clinically relevant functional

impairments determined the specifics of how the treatment plan was implemented.

There were two main types of neurofeedback training provided to the subject during this study. The first focused on normalizing and regulating paroxysmal brainwave activity, abnormal amplitude ratios, and other known patterns of EEG dysregulation. When the subject reached optimal ratio levels in critical frequency ranges and showed improved stability, as indicated by maintaining lower coefficients of variation, the subject was moved to the next protocol. The second type of neurofeedback training was two-channel sum and, when it became available, coherence neurofeedback. This second type of neurofeedback was used with cortical pairs that showed statistically significant *Z*-scored coherence abnormalities in the qEEG analysis. When the real-time measurement, derived after each session, indicated either a normal percentage coherence or that a point of maximum benefit had been reached, the subject was moved on to the next protocol.

Treatment began with 66 neurofeedback sessions provided predominantly at C3 and C4 (according to the 10–20 International electrode placement system) to improve cortical stability. Subsequently, the subject received 272 sessions to remediate statistically significant qEEG derived coherence (connectivity) impairments. Interspersed throughout this phase were an additional 192 sessions of C3 and C4 stabilization training administered before each qEEG and when clinically indicated.

RESULTS

The EEG of a normal individual exhibits much greater stability than was seen in this subject and is generally considered to be a highly reliable physiological measurement over time. The subject's serial QEEG recordings documented the spontaneous appearance of hypocoherence issues and amplitude abnormalities without a discernable pattern. Over the course of this subject's neurofeedback treatment, the qEEG analyses identified a total of 36 coherence abnormalities that converged with the subject's symptoms (see next) and were treated. Clinical results of the neurofeedback training showed that the treatment was effective in allowing the subject to experience major cognitive and emotional improvements. These improvements were also documented as each serial qEEG showed normalized coherence readings for those areas treated, with the exception of two, which then normalized after being treated a second time. As can be seen in Table 1, in spite of the fact that targeted coherence abnormalities normalized, with each new qEEG there appeared new coherence abnormalities, presumably caused by the encroaching schizophrenia.

Figure 1 shows the *z*-scored *absolute power* results from each qEEG. Figure 2 shows the *z*-scored *relative power* results from each qEEG. Beta in absolute power is not statistically significant at qEEG 5, whereas in relative power, beta shows $3 + SD$ in the parietal region. Focusing only on relative power, it appears that a shift occurred from essentially normal power on qEEG 1 to elevated beta power with emphasis in the parietal lobe on qEEG 5 over the course of treatment.

As a result of neurofeedback training, the subject not only experienced a significant reduction in symptoms but also was also able to reduce his need for aripiprazole (20 mg was reduced to 7.5 mg). This window of improved functioning allowed him to return to college where, in spite of a challenging course load, he successfully completed his senior year and graduated. Unfortunately, the subject discontinued neurofeedback treatment against the advice of his doctors and his family. Approximately 6 months after stopping neurofeedback and in spite of increased pharmacological intervention, the subject was markedly psychotic, exhibiting delusions of grandeur, auditory hallucinations, and severe paranoid ideation.

CONCLUSION

Adult onset schizophrenia beginning at the age of this subject normally has a poor

TABLE 1. Coherence Deviations Remediated with Neurofeedback

Coherence training based on qEEG 1

Electrode placement	Reward frequency	Training modality	QEEG 1 S.D	Statistically significant	QEEG 2 S.D.	Statistically significant
F7-F8	1–4	2-Chan Sum	−2.85	Y	−0.78	N
F7-F8	4–7	2-Chan Sum	−2.11	Y	−0.51	N
F7-F8	8–11	2-Chan Sum	−2.23	Y	−0.85	N
FP1-T3	8–11	2-Chan Sum	−3.11	Y	−1.26	N
FP1-F7	8–11	2-Chan Sum	−2.78	Y	0.23	N
C3-O1	1–4	2-Chan Sum	−2.32	Y	−0.59	N
C3-O1	8–11	2-Chan Sum	−2.69	Y	−1.45	N
C4-O2	8–11	2-Chan Sum	−2.38	Y	0.08	N
T3-O1	8–11	2-Chan Sum	−2.52	Y	−0.99	N
F4-P4	8–11	2-Chan Sum	−2.03	Y	−1.29	N
C4-O2	8–11	2-Chan Sum	−1.99	Y	−0.69	N
P3-F7	8–11	2-Chan Sum	−2.21	Y	−1.45	N
FP1-C3	8–11	2-Chan Sum	−2.1	Y	−0.86	N
F3-T3	8–11	2-Chan Sum	−2.05	Y	−0.7	N

Coherence training based on qEEG 2

Electrode placement	Reward frequency	Training modality	QEEG 2 S.D.	Statistically significant	QEEG 3 S.D.	Statistically significant
FP1-FP2	1–4	2-Chan Sum	−2.51	Y	−5.18	Y
PP1-FP2	4–7	2-Chan Sum	−3.62	Y	−4.3	Y
F4-P4	4–7	2-Chan Sum	−2.63	Y	0.35	N
F3-O1	4–7	2-Chan Sum	−2.22	Y	0.55	N
F4-O2	4–7	2-Chan Sum	−2.09	Y	0.4	N
FP2-P4	4–7	2-Chan Sum	−2	Y	−0.61	N
C4-O2	4–7	2-Chan Sum	−1.97	Y	0.78	N
FP2-P4	8–11	2-Chan Sum	−1.97	Y	−0.61	N

Coherence training based on qEEG 3

Electrode placement	Reward frequency	Training modality	QEEG 3 S.D.	Statistically significant	QEEG 4 S.D.	Statistically significant
FP1-FP2	0.5–3.5	Coherence	−5.18	Y	−0.99	N
FP1-FP2	4–7	Coherence	−4.3	Y	−0.19	N
FP1-FP2	8–11	Coherence	−4.51	Y	−0.18	N
FP1-FP2	15–18	Coherence	−2.62	Y	0.31	N
F3-F4	0.5–3.5	Coherence	−3.84	Y	−1.71	N
C3-C4	0.5–3.5	Coherence	−3.7	Y	−0.63	N
O1-O2	0.5–3.5	Coherence	−3.53	Y	−0.63	N
P3-P4	0.5–3.5	Coherence	−2.2	Y	0.32	N

Coherence training based on qEEG 4

Electrode placement	Reward frequency	Training modality	QEEG 4 S.D	Statistically significant	QEEG 5 S.D	Statistically significant
P4-O2	0.5–3.5	Coherence	−2.44	Y	−0.7	N
P4-O2	4–7	Coherence	−3.32	Y	−0.28	N

prognosis. However, for this individual, neurofeedback was a powerful intervention enabling him to reach a difficult scholastic milestone while on reduced levels of medication. Data from this study support the use of neurofeedback training to stabilize the amplitude dynamics of a subject's brain while reducing the rate at which abnormal cortical connections develop. In addition, several concerns emerged that warrant further investigation. For example, the subject's deterioration after stopping the neurofeedback training raises a

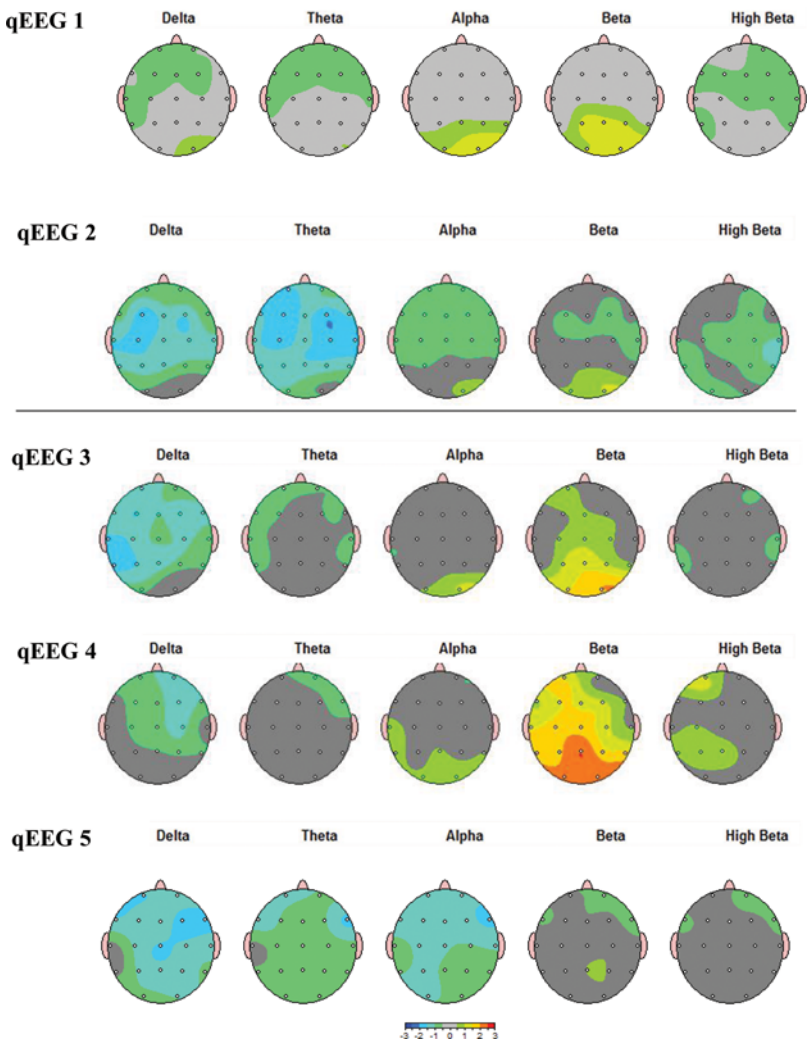

FIGURE 1. Topographic maps: *Z*-scored absolute power eyes closed (neuroguide). (Color figure available online.)

concern regarding the permanency of this intervention. As with similar disorders, schizophrenia has a genetically determined window of onset. This case study indicated that neurofeedback may prove helpful during this vulnerable time to minimize symptoms. The optimal duration of treatment including when it may safely be reduced or terminated remains to be studied. This study gives rise to speculation that qEEG beta absolute power may be a sensitive indicator of the progression of the disorder. It is unknown why normalization occurred in beta in the absolute power analysis in qEEG 5 but suggests that the subject may have reached a midpoint in the window of susceptibility sometime between qEEG 4 and qEEG 5. Perhaps if the subject had continued the training he may have resolved this window of vulnerability with a significantly milder constellation of schizophrenia symptoms. Other studies may also discover ways to decrease the number of sessions or make the training more efficient, thus allowing the treatment to be more feasible. This study supports research indicating the crucial role that the brain's integrated neural network plays in optimal functioning and provides evidence that, with further research, neurofeedback training may one day prove to be a useful tool to treat schizophrenia.

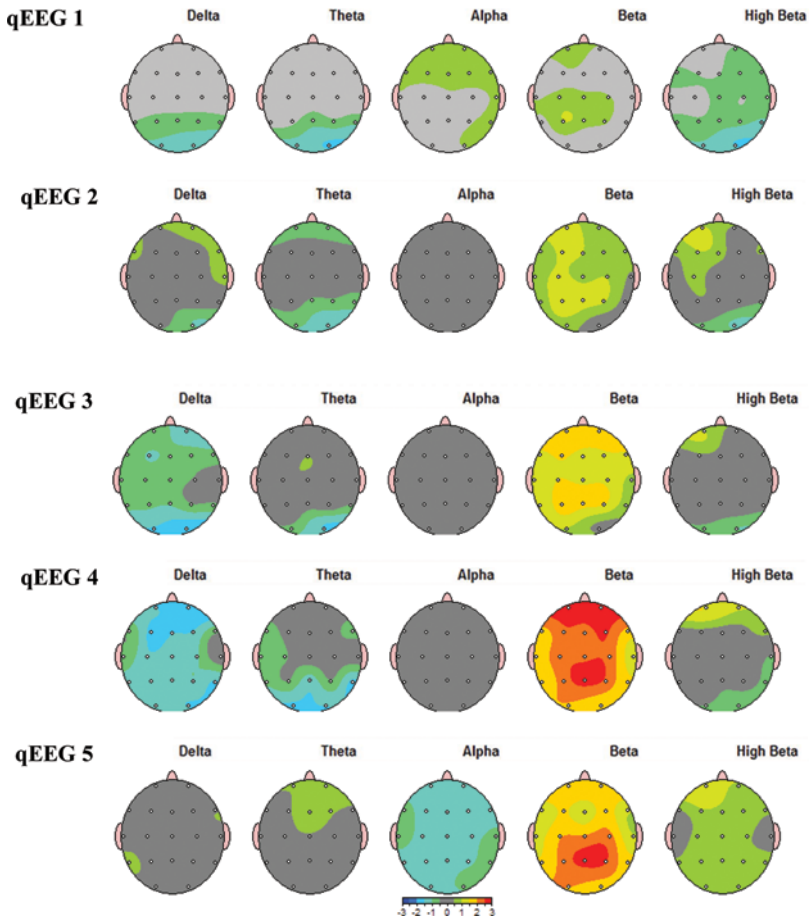

FIGURE 2. Topographic maps: Z-scored relative power eyes closed (neuroguide). (Color figure available online.)

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