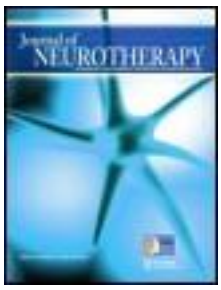

Journal of Neurotherapy: Investigations in Neuromodulation, Neurofeedback and Applied Neuroscience

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Published online: 26 Feb 2010.

To cite this article: Angelo S. Bolea PhD (2010) Neurofeedback Treatment of Chronic Inpatient Schizophrenia, Journal of Neurotherapy: Investigations in Neuromodulation, Neurofeedback and Applied Neuroscience, 14:1, 47-54, DOI: 10.1080/10874200903543971

To link to this article: http://dx.doi.org/10.1080/10874200903543971

Journal of Neurotherapy, 14:47–54, 2010

ISSN: 1087-4208 print/1530-017X online
DOI: 10.1080/10874200903543971

Neurofeedback Treatment of Chronic Inpatient Schizophrenia

Angelo S. Bolea, PhD

ABSTRACT. This is a study on the effect of neurofeedback on chronic inpatient complex paranoid schizophrenics. The purpose of this research was twofold: first, to determine the effects of the application of neurofeedback to very chronic cases of schizophrenia that had been resistant to years of inpatient medical and psychological treatment and second, to propose a connection paradigm of schizophrenia. The author obtained progress using neurofeedback with more than 70 hospital inpatients with chronic schizophrenia. Improvements were seen in the EEG patterns and in cognitive, affective and behavioral patterns that often resulted in successful release from the hospital to live in the community. A 2-year follow up found that positive changes were sustained. It is the author's impression that reinforcement of right parietal alpha and inhibiting frontal delta and fast beta activity obtained the best results.

KEYWORDS. Biofeedback, EEG, inpatient hospital, neurofeedback, schizophrenia

INTRODUCTION

Although much progress has been made in the treatment of schizophrenia in recent years, many cases persist that are considered relatively treatment resistant. This article describes the author's experience in an inpatient psychiatric hospital applying neurofeedback to severe and chronic schizophrenic patients who were deemed completely treatment resistant.

At this time, more than 70 chronic cases have been treated with neurofeedback. One severe case is detailed in this article to illustrate the common treatment process. This patient was typical of the 70 patients that I have treated thus far neurofeedback. He was a 40-year-old male who had been hospitalized for 20 years with paranoid schizophrenia. He was referred to Neuropsychology Lab because of explosive violent behavior. Several treatment modalities had been employed through the years, including individual psychotherapy by five different psychotherapists with no change in behavior. Other therapeutic interventions had included behavior and cognitive therapies, group psychotherapy, multiple medications, and milieu therapy on the hospital ward. Nothing had been effective in controlling his paranoid ideation or violence. He, like the more than 70 other inpatients I have treated with neurofeedback, were considered "hopeless."

The newly formed Hospital Neuropsychological Lab had as a part of its objective to develop innovative interventions for such treatment resistant conditions. Just prior to his initiating neurofeedback, a special neurobehavioral plan of treatment was conducted weekly in individual, 1-hr sessions for 1 year. The neurobehavioral intervention also failed

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to change the patient's paranoia, and therefore neurofeedback was initiated.

Because this author was the therapist for both the neurobehavioral and neurofeedback therapy, this case may be considered quasi-controlled—comparing two treatment interventions, neurobehavioral and neurofeedback, while keeping the therapist the same.

Much thought and preparation was given at the hospital Neuropsychology Lab regarding chronic psychiatric conditions and the application of neurofeedback. After reviewing numerous therapies for schizophrenia, it was felt that a paradigm shift was needed to a brain-based neuroconnection hypothesis (David & Cutting, 1994). A new paradigm was proposed consisting of two dimensions.

First, these conditions can be viewed as some form of neuro-disconnection (Davis, 2003). Rather than emphasizing thought disorder, emphasis was put on a competence cognitive modal approach. Neurofeedback was considered as developing neuronal connections, bridging disconnection, and thus developing a neuro-cognitive integrative thought process. It was believed that patients with improved neuroconnectivity would be able to express thoughts and feelings in understandable, meaningful shared communication. Trust was emphasized and developed to replace previous the paranoid emphasis. The object as the author conceptualizes it is not so much to remove the paranoia but to create trust through building neuronal bridges of improved neuro-cognitive functioning.

Medication treatment paradigms are based on neurochemistry, viewing schizophrenia and chronic psychiatric conditions as representing a chemical imbalance. All 70 patients treated by this author had been treated in accordance with this conceptual paradigm, receiving multiple medications. Other conceptual treatment models include an emphasis on cognitive, behavioral and psychological processes, and the therapies associated with these treatment models had also been tried without much improvement.

Studies over the past 30 plus years have consistently shown that electrophysiological patterns can be changed by neurofeedback (e.g., Hammond, 2006; Levesque, Beauregard, & Mensour, 2006; Monastra, Monastra, & George, 2002; Sterman, 2000). Therefore, it was decided that treatment at the lab would utilize neurofeedback, combining a neuro-disconnection and neuro-electrical paradigm for decision making and the application of neurofeedback. The lab did not have the capacity to do quantitative EEG evaluations. Therefore, it was simply hypothesized that a neuronal disconnection existed between the frontotemporal and parietal lobes that could result in islands of isolated, disconnected thought, relationships, and emotion. Disturbed electrophysiological patterns have been reported involving elevated frontal delta activity (Itil, 1977), and both hypercoherence and hypocoherence problems have also been found in schizophrenia (John et al., 2007, John et al., 1994; John et al., 2007; Pascual-Marqui, 2000). Such information was helpful in applying neurofeedback with schizophrenic patients.

METHODOLOGY

Computerized EEG biofeedback was used for treatment. A 40-year-old single man was invited to be hooked up to the computer to do neurofeedback to help him feel better, build his confidence, and improve the likelihood of discharge from the hospital. After demonstration of the equipment and process, he was willing to allow a brief session of about 5 min, which subsequently was gradually increased to 1-hr sessions twice per week for 18 months (130 individual sessions total).

Equipment

CapScan single-channel neurofeedback equipment attached to a laptop computer was used. Visual feedback on a screen consisted of three columns showing which brainwave frequencies were targeted. A green light with simultaneous audio feedback (a single discrete sound) occurred when the brainwave pattern changed toward a balance.

The patient was asked to make the sound and green light come on as often as he could. The operant conditioning process was described to him with the analogy that it was like "learning to ride a two-wheel bike." A score was kept for the number of times he received this feedback and he was encouraged to increase his score as sessions progressed. Over the course of several sessions, through trial and error, the patient sensed what was required to produce positive feedback. As sessions progressed, changes were noted in both the patient's EEG and behavior.

Outcome Assessment and Background History

Neuropsychological testing, EEG Spectral Analysis, and an individualized behavioral checklist were done pre- and posttreatment. Continuous weekly monitoring of patient behavior on the ward was recorded. Regular communication occurred face-to-face with the inpatient hospital staff and by telephone. Sessions were conducted in the Neuropsychology Lab primarily, with occasional sessions conducted on the ward when behavioral episodes made it unsafe to transport the patient across campus to the Neuropsychology Lab. Such restrictions were frequent in the earlier part of treatment. The total number of sessions was 130 over an 18-month period. Length of sessions gradually increased from 5 min to 1 hr in length.

The Wechsler Adult Intelligence Scale was administered and his IQ was in the low average range with a Verbal IQ of 78, Performance IQ of 87, and a Full Scale IQ of 81. Several of the Neuropsychological Tests were challenging. Although the Trails A test was passed, Trails B was failed despite three attempts. Tactual Performance, and Aphasic and Perceptual Screening tests were all failed. Aggressive behavior was frequent, requiring restriction to the ward and placement in seclusion and with physical restraints. Paranoid delusions were persistent, accompanied by complete social isolation and withdrawal.

His background EEG showed disorganization, and EEG Spectral Analysis showed elevated bifrontal delta activity, a low amplitude of posterior alpha, and generalized elevated EEG amplitudes in the left hemisphere while the right hemisphere was very low in amplitude.

The patient in this case example had been hospitalized at an inpatient psychiatric hospital for 20 years. He had a diagnosis of severe, chronic paranoid schizophrenia and was considered treatment resistant. He was a short, stocky, tough-looking man, about 5 foot 7, 220 lb, with a strong body odor. He had to be escorted to the therapy sessions. When he appeared for sessions he often pounded forcefully on the door and loudly burst into the office with an intimidating appearance. He always wore a torn coat, dark green T-shirt, and black pants, which were never changed. When his clothes had to be washed, he waited until they were dry and wore the same clothes. Prior to inpatient treatment, behavioral problems, temper outbursts, and social isolation were present. Problems escalated after his father died when the patient was in his teens. During his early 20s, violent episodes were common between him and his only sibling, a younger brother. During the first 5 years of hospitalization, his mother developed severe Alzheimer's and was placed in a nursing facility. She no longer recognized him. His brother left the area, having no further contact with the patient.

Being completely cut off from family, the patient spent most of his day sitting in the corner of the hospital ward, often sleeping and then being unable to sleep at night. All attempts of therapeutic intervention on the ward were either refused or ended in explosive episodes. Numerous unsuccessful attempts were made for community placement. Each episode of planning for discharge generated an increase in uncontrollable behavior with violent threats and paranoia.

Referral was made to the Neuropsychology Lab out of pure desperation with the intent of knowing if neurofeedback could be of assistance. Referral for neurofeedback for all of the 70 patients was often made after

numerous unsuccessful attempts to deal with symptoms of chronic schizophrenia.

Treatment Intervention

Electrodes were placed in strategic areas on the scalp to record the EEG. A criterion level measured in microvolts was set a level to achieve 80% reward. When the criterion level was achieved, the computer provided immediate visual and auditory feedback.

For the first 40 sessions this patient resisted any verbal suggestions. He repeatedly and emphatically stated, with hesitant speech, that the treatment would not work. He continued treatment because it got him off the ward. Sessions were often interrupted by resistance and continuous incessant repetitive statements of negativity. Neurofeedback treatment occurred in three phases.

Phase 1: Right parietal treatment phase. This phase focused on reinforcement of alpha activity (8–12 Hz) while inhibiting fast beta frequency (21–30 Hz) activity. This continued for 40 sessions. The electrode was placed at electrode side P4 alternating with PO4 with a reference electrode at A1 and a ground electrode on the other ear.

Phase 2: Frontal treatment phase. This phase focused on periodic, very brief (3 to 5 min) treatment with an electrode at FC3, inhibiting delta (2–5 Hz activity while reinforcing low beta (13–15 Hz). Following these few minutes of treatment, we returned to right parietal enhancement of alpha activity (Phase 1).

Phase 3: The connection and integration phase. This phase first consisted of bipolar (sequential) neurofeedback training (PO4-CP4) focused on reinforcing alpha activity (while inhibiting fast beta activity) for about 10 min. Second, bipolar training was done in the left frontal area (FC3-Fp1) as in phase 1, inhibiting 2–5 Hz while reinforcing 13–15 Hz for up to 10 min. As before, treatment always returned to the right parietal area (PO4-CP4) for calming for approximately 20 min.

Therapist feedback was provided at the end of each session, pointing out the amount of time and success. Points on the computer, for time on target, were continuously displayed during treatment sessions. The number of points earned in the sessions (number of times criterion was met) were reinforced as good when he reached 1,000 points and as very good when he reached 5,000 points. Behavioral observations were also recorded after each session, without verbal comment, as even the most positive comments about behavior resulted in negativity and agitation.

RESULTS

Critical Behavioral Observations at the Outset of Treatment

There were frequent furtive aggressive stares and glances by the patient in the early part of treatment. He was unable to carry on a conversation. If therapist comments were made, he reacted with an unusual incessant request to repeat, that is, "say it again . . . again . . . again . . . again." The sessions proceeded with very little verbalization other than hostile complaints, negativity, and statements like, "They are killing me," "I'm dying," and "Nobody cares." These statements continued for 6 months but were gradually decreasing in intensity and frequency. It took 1 year of treatment before a single positive statement was made by the patient ("Can you help me?").

Table 1 shows behaviors associated with cognitive, affective, and self-regulation behaviors before and after neurofeedback treatment.

Table 2 displays the number of sessions that occurred before critical behaviors began to change for the first time.

Table 3 displays shows pre- and posttreatment results.

Figure 1 presents pre- and posttreatment EEG spectral maps at the left and right temporal lobes reflecting change toward more healthy brainwave patterns.

Table 4 shows 2-year follow-up results.

DISCUSSION

Schizophrenia, typified by a severe case of treatment resistant and chronic schizophrenia,

TABLE 1. Changes in cognitive, affective, and self-regulation of chronic schizophrenia treated with neurofeedback.

Behaviors	Pre-Treatment Weekly Occurances	Post-Treatment Weekly Occurances*	Direction of Change
1. Violent outbursts	3	O	+
2. Physical altercations	4	0	+
3. In restraint room	4	0	+
4. Repetitive demands	more than 100	2	+
5. Loud screaming Screams, howling	10	1	+
6. Self grooming	0	5	+
7. Bilateral hand tremor	constant	1	+
8. Positive statements about self and others	0	3	+
9. Conversational language	0	2	+
10. Helping someone else	0	2	+

TABLE 2. First-time positive behavior was observed during neurofeedback treatment of chronic schizophrenia.*

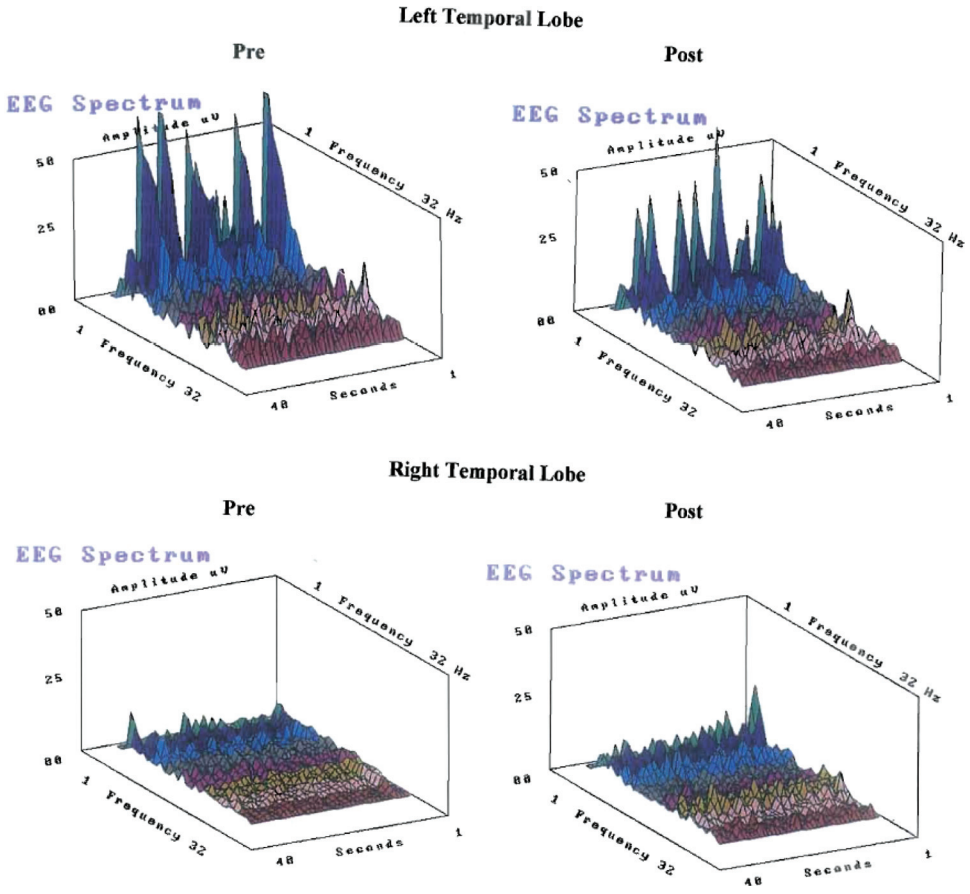
Behavior Observed	Number of Sessions Required
1. Acceptable Sensor Hook up	5
2. Gentle eye contact	15
3. Gentle (non-demanding) request	20
4. Show of gratitude, "thank you"	50
5. Tried to suppress curse outburst	67
6. Held back compulsive repetitions	72
7. Hand steadiness (loss of tremor)	80
8. Discharge to Community Living	130

*It was encouraging to the therapist to keep a "first time observed" record.

TABLE 3. Pre- and posttest results of chronic schizophrenia.

Test Name	Pre-Test	Post-Test	Direction of Change
EEG Spectral	Left Hemisphere		
Brain Map	Delta 45 mV	Delta 25 mV	+
	Fast Beta 12 mV	Fast Beta 5 mV	+
	SMR 5 mV	SMR 6 mV	+
	Right Hemisphere		
	Generalized Low mV Alpha 2 mV	Generalized Increase mV Alpha 6 mV	+
Neuropsychological Tests			
VIQ	78	96	+
PIQ	87	102	+
FSIQ	81	99	+
Trails A	ok	ok	+
B	Failed × 2 (stopped)	Slow − 3 min. passed	+
Tactual Performance Test	Failed × 2 (stopped)	Passed: slow Dom: 11 min. Non-Dom: 9 min. Both: 5 min.	+
Aphasic and Perceptual Screening	Failed	Passed	+
Projective Drawing	Failed (unable to construct Tree Drawing)	Passed (30%) Space Utilization)	+

FIGURE 1. EEG spectral maps showing positive changes.



responded positively to neurofeedback as evidenced not only by success in positive changes in behavioral self-regulation, cognitive, and affective changes but also by being able to resume living within the community. A 2-year follow-up documented continued satisfactory adjustment with stabilization on less than one half the number and dosage of medication in comparison with the onset of treatment.

This successful case outcome soon led to additional referrals of more than 70 other chronic treatment resistant schizophrenic patients, all of whom showed significant progress. Most were discharged to community

TABLE 4. Two-year follow-up: Posthospital discharge.

1. Living in Group Home satisfactorily
2. Attends Individual and Group Therapy
3. Attends Socialization Activities
4. No Explosive Episodes: Manages Temper
5. No Verbal Threats, but does share such thoughts with Therapist
6. No Pushing or Shoving but grumbles about people
7. Verbal Request for Repetition stopped but some Dysphasia: “I can’t find the word I want to say”
8. Cautious Trust: made friends with two residents, three staff.
9. Tremor Resolved
10. Medication Reduced in number and with ½ prior doseage

living. One of these patients had delusions of numerous pet animals around her at all times. After several sessions of right parietal alpha enhancement, these delusions transformed into an insight of the importance of interpersonal relationships. Another patient believed that she was pregnant, a delusionthat persisted for years until after neurofeedback treatment. This delusion also evolved into a profound question: "Do you think it is possible for a person my age to get born and start life over?" After such statements of insight, the delusions no longer surfaced. Remarkable positive behaviors were reported in all 70 patients.

What have I learned from clinical experience in treating inpatient schizophrenics? Reinforcement of right parietal alpha seemed to work the best with chronic schizophrenics but did require patience and persistent of up to more than 100 sessions. Relentless reduction of fast beta (21–30 Hz) and enhancement of alpha (8–12 Hz) at P4, PO4, CP4 seemed very important. Only later was it wise to reduce 2–5 Hz activity frontally, starting at PC3, AF3, and FP1, but returning always to the right parietal area to produce the quickest and most certain sense of calming.

Midline and interhemispheric placements were tried and resulted in exacerbated symptoms. Perhaps later, after more stabilizing time or with less chronic and severe conditions, other sensor placements would be appropriate.

Although circumstances did not permit continued neurofeedback after discharge, follow-up outpatient neurofeedback it is recommended after hospital discharge. Until more is learned about the long-term outcomes of neurofeedback with schizophrenics, periodic neurofeedback reinforcement sessions may be needed for maintenance of progress.

Suggestions and Cautions

1. Avoid staying at one electrode site for very long. Short sessions worked better.
2. Take frequent breaks during the session.
3. Careful therapist language selection: The therapist needs to be positive and consistent with the outcome desired from working in specific areas of the brain (e.g., "parietal talk" about calming and relationships).
4. Expect negativity and failed appointments. Avoid midline, interhemispheric, and frontal lobe placements until work in the parietal and temporal areas has produced more stability.
5. Ask less "how a client feels" and emphasize more about what is being worked on, such as "building trust" and "things will get better."
6. When unwanted thoughts surface, refer to them as "glitches" in the brain, nothing more. "We'll take care of it. . . . It's only a glitch."
7. Monitor medication as neurofeedback progresses. Less medication or a different type of medication may be needed, as patients become more responsive to medications as treatment progresses.
8. Change protocol according to pre-testing and the individual patient. "One size protocol does not fit all patients."
9. Remain solidly positive and keep in close communication with knowledgeable supportive professionals.

CONCLUSIONS

The rationale for neurofeedback treatment of schizophrenia is based on the fact that there is abundant evidence of disturbed electrophysiological functioning in this population. However, as demonstrated in previous studies such as Levesque et al. (2006), neurofeedback can take advantage of brain plasticity and successfully improve neuronal activity. Results of my neurofeedback treatment of schizophrenics have commonly shown an enhancement of alpha activity in the right parietal area, a decrease in 2–5 Hz activity in the left frontal area, and reduction in fast beta activity at all sites where training occurred. The outcomes in these chronic schizophrenic patients demonstrate what Gruzelier (2000) anticipated 10 years ago might be possible in the

future—that when neurofeedback is appropriately, persistently, and patiently applied, it can produce breakthroughs in previously treatment resistant chronic schizophrenics.

Further randomized controlled research is needed with schizophrenics utilizing standardized outcome measures and with either pre- and posttreatment QEEGs or functional MRIs, and long-term follow-ups comparing neurofeedback to standard psychiatric (medication) treatment. Until such studies have been completed, careful application of neurofeedback by experienced practitioners may find that emphasizing right parietal alpha enhancement may be a very important component in neurofeedback treatment for schizophrenia.

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