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Neurofeedback Training for a Patient With Thalamic and Cortical Infarctions

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One year after a left posterior and thalamic stroke, a 52-year-old male participant was treated with 14 weeks of theta reduction neurofeedback training. Imaging studies revealed left temporal, parietal, occipital, and bilateral thalamic infarctions along the distribution of the posterior cerebral artery. Neuropsychological testing demonstrated severe verbal memory, naming, visual tracking, and fine motor deficits. Additionally, alexia without agraphia was present. A pretraining quantitative electroencephalograph (QEEG) found alpha attenuation, lack of alpha reactivity to eye opening, and excessive theta activity from the left posterior head region. Neurofeedback training to inhibit 4–8 Hz theta activity was conducted for 42 sessions from left hemisphere sites. Over the course of the training, significant reductions in theta amplitude occurred from the training sites as assessed from the post-session baseline periods. Posttraining, a relative normalization of the QEEG was observed from the left posterior head region.

KEY WORDS: neurofeedback; neurotherapy; QEEG; stroke; CVA.

INTRODUCTION

The rationale for neurotherapy with patients who have suffered strokes is largely based on a correlation between decreased cerebral blood flow and increased theta activity. In a review of studies assessing patients with strokes, Hughes and John (1999) reported correlations of $>.7$ between decreased cerebral blood flow and increased EEG slow wave amplitude. Neurotherapy for patients with stroke seeks to promote normalization of brain function by reducing the amount of EEG slow wave activity and increasing faster rhythms in hypofunctional brain regions. To date, there are very few published peer-reviewed studies that address the use of neurofeedback therapy with patients with either stroke or posttraumatic head injury.

In a review of her 20 years of work with patients poststroke, Ayers (1999) reported improvements in muscle performance after neurofeedback from left sensorimotor (T3–C3)

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locations with small numbers of child and adult clinical cases. Hoffman, Stockdale, Hicks, and Schwaninger (1995) reported 80% of their mildly posttraumatic head-injured patients demonstrate improvements in self-reported symptoms and neuropsychological measures after an average of 40 sessions of neurofeedback training. Thornton (2000) created a normative quantitative electroencephalograph (QEEG) database that assessed correlates of effective memory functioning. He compared three patients' (two post-head-injury and one posthippocampal surgery) QEEGs during paragraph recall to the database and devised individualized treatment protocols to remediate their deficits. Thornton reported his three patients demonstrated 68–181% improvement in mean recall to paragraphs after amplitude and coherence neurofeedback training.

Rozelle and Budzynski (1995) published a case report detailing their use of audio/visual stimulation and neurofeedback therapy with a 55-year-old male who had suffered a left posterior temporal/parietal cerebrovascular accident (CVA) one year before training began. After 21 sessions of audio/visual stimulation, Rozelle and Budzynski conducted 48 sessions of neurofeedback training over the patient's most affected left hemisphere sites as delineated by an initial QEEG. Their patient was trained to decrease slower 4–7-Hz theta waves and increase faster 15–21-Hz activity over sensorimotor and speech areas. At the conclusion of training, Rozelle and Budzynski reported significant pre–posttraining reductions in eyes-open theta activity over the training sites as measured by the QEEG. Additionally, their patient's pretraining lack of alpha attenuation with eye opening was not evident after training. Their patient also demonstrated significant pre–posttraining changes on neuropsychological tests assessing aphasia, on a self-report inventory of psychological distress, and from an independent speech evaluation. Given that maximal neurological recovery from even initially severe stroke occurs within 4–5 months poststroke (Jorgensen et al., 1995), it is important to note that Rozelle and Budzynski's patient was already 1-year-poststroke before training was initiated. Therefore, the reported clinical improvements were unlikely to be due solely to the passage of time. Putman (2001) reported several clinical improvements after neurofeedback training with a 71-year-old patient that had recently suffered a stroke. However, Putman's patient was only 2-months poststroke when training began, so improvements may have been influenced by passage of time and concurrent treatment effects.

The present case study utilized a simple theta reduction neurofeedback protocol. No effort was made to enhance any higher frequency activity. It was hypothesized that (1) pre–posttraining QEEGs would demonstrate significant reductions in theta power and a general trend toward normalization, (2) reductions in theta across training sessions would occur, (3) a number of the pre–posttraining neuropsychological tests would show significant improvements, (4) there would be significant improvements in self-report measures of mood.

METHOD

Participant

The participant in this study was a 52-year-old male with no previous history of CVA. He experienced an abrupt onset of confusion and numbness and weakness of his right upper extremity while at work. CT and MRI scans revealed the patient had suffered an ischemic infarction with involvement of left hemisphere temporal, parietal, and occipital

head regions. The left temporal damage extended ventrally into the parahippocampal gyrus. Additionally, bilateral thalamic infarctions occurred with more damage to the left thalamus than the right. A Carotid Doppler study found stenosis of the left posterior cerebral artery and the patient's infarctions followed the distribution of the posterior cerebral artery.

On admission the patient demonstrated marked word retrieval problems, severe anomia (he recognized but was unable to name family members), and severe immediate and delayed verbal memory deficits. He also had visual tracking deficits, a right upper visual field deficit (right upper quadrantanopia), and an inability to read (alexia). However, he retained his ability to write. Alexia without agraphia is a very rare form of expressive aphasia that, in conjunction with verbal amnesia, has been associated with an extensive infarction of the posterior cerebral artery territory (Damasio & Damasio, 1983). One week after his stroke the patient was transferred to a rehabilitation hospital where he underwent 3 weeks of physical therapy. Because of mild to moderate depression, he was placed on 40 mg of Celexa. The patient was also placed on several other medications to help control his blood pressure while still in the hospital. They were Plavix (75 mg), Avapro (300 mg), and Hydrochlorthiazide (12.5 mg). After discharge from the rehabilitation hospital, his medications' dosages remained constant through his additional rehabilitation and the entire course of neurofeedback training. The patient subsequently entered an outpatient brain injury rehabilitation program for 10 months. His therapeutic regime consisted of group therapy that addressed the cognitive and affective sequelae of brain injury, and individual therapy that primarily consisted of remedial reading and short-term memory exercises.

Over the course of his rehabilitation therapy he was administered three extensive neuropsychological evaluations at 1-month, 6-month, and 1-year post-CVA. Comparison of the 6-month to 1-year post-CVA neuropsychological batteries revealed no significant improvements in functioning. The results of the 1-year post-CVA (pretraining) and post-training batteries are presented in detail in the Results section. At 1-year post-CVA, the neuropsychologist reported the patient continued to display severely impaired verbal memory, marked impairment in reading ability, a residual right upper field visual deficit, and severely impaired right upper extremity fine motor speed and dexterity. The patient was often unaware of repeating himself in conversation and had great difficulty learning new material. He continued to have severe difficulty remembering names, and would often forget his friends' and grandchildren's names. The patient had almost given up trying to read because of frustration about his extremely slow reading rate

The patient's pretraining QEEG (conducted 1-year post-CVA) revealed left hemisphere abnormalities consistent with his prior imaging studies and residual neuropsychological deficits. The eyes-closed posterior alpha rhythms were asymmetrical with alpha voltage suppression from the left posterior temporal (T5), parietal (P3), and occipital (O1) electrode sites compared to the homologous (T6, P4, O2) right hemisphere sites (left $M = 11.7\ \mu V^2$, right $M = 27.8\ \mu V^2$). Upon eye opening, the expected alpha attenuation occurred from the right posterior head region (59% reduction), versus only a slight attenuation (12% reduction) from the left posterior head region. Local voltage suppression and unilateral lack of alpha reactivity to eye opening are reliable signs of focal cerebral dysfunction (Niedermeyer, 1998; Sharbrough, 1998). Also when the patient's eyes were open, rare sharp-appearing transients and some 4–7 Hz slowing were occasionally observed from T5, P3, and O1.

Two months after his discharge from the rehabilitation program (1-year post-CVA), the patient elected to try neurotherapy and hospital ethics committee approval was obtained. All neurotherapy services were provided without charge.

Procedure

Pretraining and posttraining QEEGs were conducted with a Lexicor Neurosearch^{TM}-24 using a 19 channel linked-ears referential montage by an experienced Registered EEG Technologist (TSB). The pretraining QEEG was performed 3 days after the patient's 1-year post-CVA neuropsychological evaluation and 3 days before his first neurofeedback session. The posttraining QEEG was conducted 1 day after the conclusion of 14 weeks of neurofeedback training. Gold electrodes were applied with paste according to the 10–20 International System of electrode placement and electrode impedances were below 5 kΩ.

The 19 channels of EEG waveforms were digitized at a sampling rate of 128 samples/s in 2-s epochs with a gain of 32,000. The digitized waveforms were manually edited to remove epochs containing artifacts (such as eye movements and muscle activity). Fast-Fourier transforms (FFTs) were performed allowing spectral analysis of the EEG including power and amplitude measures, compressed spectral arrays, and topographic maps. Bandwidths were Delta: 1–4 Hz, Theta: 4–8 Hz, Alpha: 8–13 Hz, and Beta: 13–32 Hz.

Both QEEGs were performed in an identical manner with 10 min of eyes-closed recording, then 10 min of eyes-open recording (staring at a spot), followed by a recording while silently reading a passage designed to test reading speed from the Minnesota Test for the Differential Diagnosis of Aphasia (Schuell, 1965). Both QEEGs were conducted at 2:00 p.m. (to minimize possible diurnal effects) 1 h after the patient had eaten a light lunch (Cummings, Dane, Rhodes, Lynch, & Hughes, 2000).

Neurofeedback Training

The neurofeedback strategy was twofold. Based on the pretraining QEEG, it entailed training to reduce theta over the area of maximal dysfunction (P3) as evidenced by excessive theta activity and lack of alpha reactivity to eye opening. Additionally, an adjacent area over the left mid-temporal and left central sensorimotor strip (T3–C3) was trained to maximize the possibility of improving thalamocortical integration.

The neurofeedback training was conducted utilizing a Thought Technology Procomp+/Biograph^{TM} biofeedback system capable of recording and displaying two channels of EEG data. The analog EEG waveforms were digitized at a sampling rate of 256 samples/s and finite impulse response (FIR) filters were used for spectral analysis. The patient received visual feedback from a video monitor in the form of a green square (representing the amplitude of his theta band activity) that he attempted to keep within a box (which represented his preset 10-μV threshold). A counter advanced and a soft auditory tone occurred every time he kept his 4–8-Hz theta amplitude within the box for at least 250 ms. Additionally, a percent counter displayed the percent time the theta stayed below threshold. An adjacent bargraph display monitored EMG activity (as measured in the $\geq$45-Hz range from the EEG electrodes). The bargraph turned red and inhibited the other visual and auditory feedback modes whenever a preset threshold of 5 μV of EMG activity was exceeded.

Each neurofeedback session had two 2-min pre- and posttraining baselines during which the patient stared at a fixed location 5 ft away. Each session consisted of five 5-min training periods divided by approximately 4-min breaks during which the training period was discussed and statistical results were printed out.

The theta threshold was set at the initial session so that reward occurred 50–70% of the time. The first training phase of 18 sessions was conducted from P3 referenced to the left ear. The ground electrode was placed on the right ear. The second EEG channel monitored EEG activity at T5 to ascertain if some generalization was occurring from training, but it was not used for feedback purposes. The second training phase of sessions 19 through 30 was conducted from a bipolar T3–C3 placement as advocated by Ayers (1999) and Rozelle and Budzynski (1995). Simultaneously, the EEG was monitored from P3, but no feedback was provided from this location. The third training phase of sessions 31 through 42, utilized the original P3 placement with T5 monitoring. Training occurred at the same time each day (2.00 p.m.) over a 14 week period. It was initially conducted three times a week and then gradually reduced to two times a week.

Beginning with session 12, a 5-min reading training period was added each session after the final baseline to directly address the patient's alexia. The patient silently read from short children's stories while attempting to keep the audio feedback tone on as much as possible.

RESULTS

The posttraining QEEG demonstrated marked changes from the pretraining QEEG. These pre–post differences are most readily observed in the eyes-open and reading rate data. Figures 1 and 2 are compressed spectral arrays (CSAs) of the pre- and posttraining power from P3. These CSAs plot power versus frequency over time.

The pretraining eyes-open CSA (see Fig. 1) shows elevated levels of power across the frequency spectrum, but especially in the alpha band. Indeed the patient's eyes-open P3 alpha power (12.4 μV^2) was almost as high as his eyes-closed value (15.4 μV^2), which is not displayed. In contrast, the patient's posttraining eyes-open CSA demonstrates marked reductions in delta, theta, and alpha band activity indicative of a normalization of cortical regulation.

Likewise, the patient's pretraining reading CSA from P3 reflects an absence of the expected alpha attenuation (see Fig. 2). His posttraining reading CSA again demonstrates marked reductions in alpha, and to a slightly lesser extent, delta and theta band activity. Different numbers of epochs are present in the two reading CSAs, because the patient read the selection much faster posttraining than pretraining.

The α level of significance for the statistical comparisons of the pre–posttraining QEEG power values was set a priori at $p < .01$. Paired two-tailed t tests of mean power values per epoch support the interpretation of the CSAs described above and confirm that significant reductions in slow wave activity from the affected left parietal head region occurred after training across all experimental conditions (see Table I). Comparisons were made across all frequency bands for P3 in the eyes-open, eyes-closed, and reading conditions.

Significant eyes-open power decreases were found at P3 in the delta, $t(73) = 3.06$, $p < .01$, theta, $t(73) = 3.88$, $p < .001$, and alpha $t(73) = 3.38$, $p < .001$, frequency bands. Significant eyes-closed decreases were found at P3 in the delta, $t(89) = 6.01$, $p < .001$, and theta, $t(89) = 3.36$, $p < .001$, frequency bands. Significant reading decreases were found in the delta, $t(32) = 3.15$, $p < .01$, theta, $t(32) = 3.44$, $p < .01$, and alpha, $t(32) = 3.86$, $p < .001$, frequency bands.

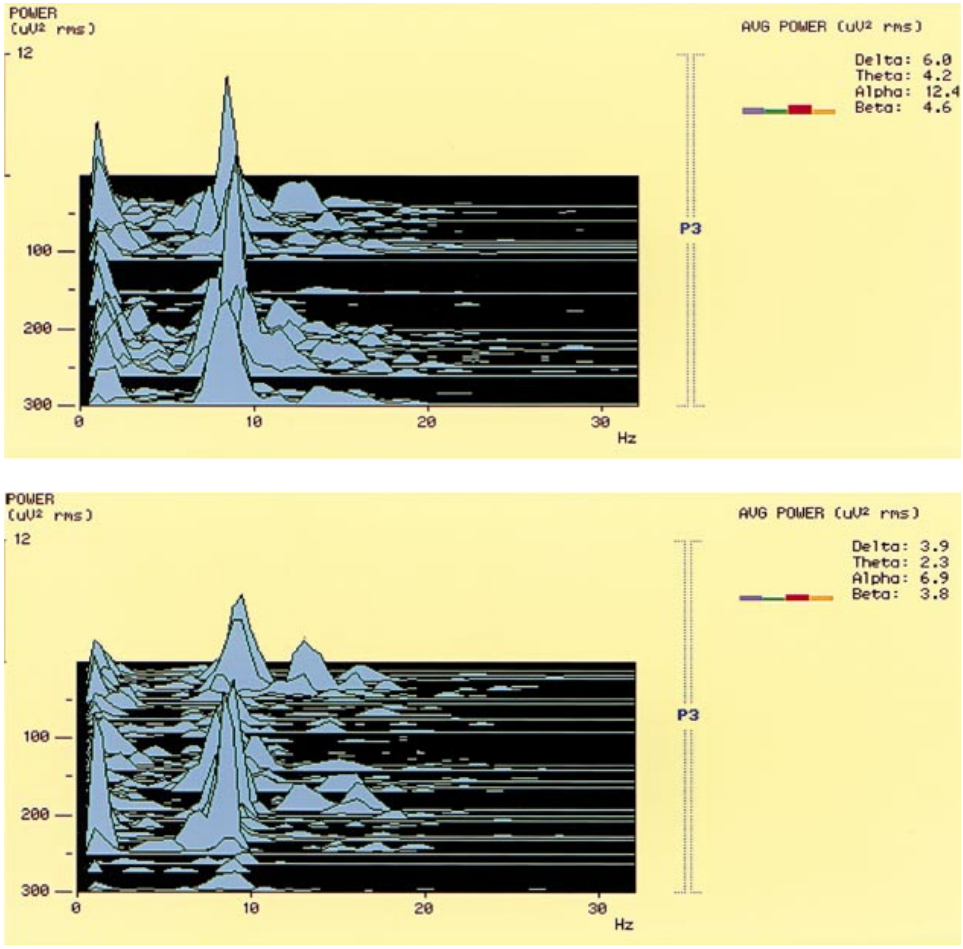

Fig. 1. Pretraining (A) and posttraining (B) eyes-open compressed spectral arrays of power from P3. The y-axis denotes time as measured across the 300 two-second epochs of the recording. The x-axis represents the frequency spectrum from 1 to 32 Hz. Power values are divided into the standard frequency bands and averaged across epochs. Improved alpha attenuation to eye-opening is evident on the posttraining CSA.

To reduce the number of t-test comparisons from the 19 possible electrode sites, 4 linear channel combinations, each composed of 3 electrode sites, were formed. t tests were only conducted between the eyes-open theta band means. A significant reduction in theta, $t(73) = 3.72, p < .001$, occurred from the left posterior quadrant combination (represented by P3, T5, and O1). This significant reduction of left posterior theta represents a localizing training effect because the homologous right posterior quadrant combination (represented by P4, T6, and O2) did not demonstrate a significant reduction in theta power, $t(73) = 1.17, ns$. A significant reduction in theta, $t\ (73) = 4.16, p < .001$, was also observed from both the left frontotemporal-central quadrant (represented by F7, T3, and C3), and the homologous right frontotemporal-central quadrant (represented by F8, T4, and C4), $t(73) = 3.45, p < .001$.

Statistical analyses were also performed to determine whether rms theta amplitude decreased both across and within training sessions. The α level of significance for these

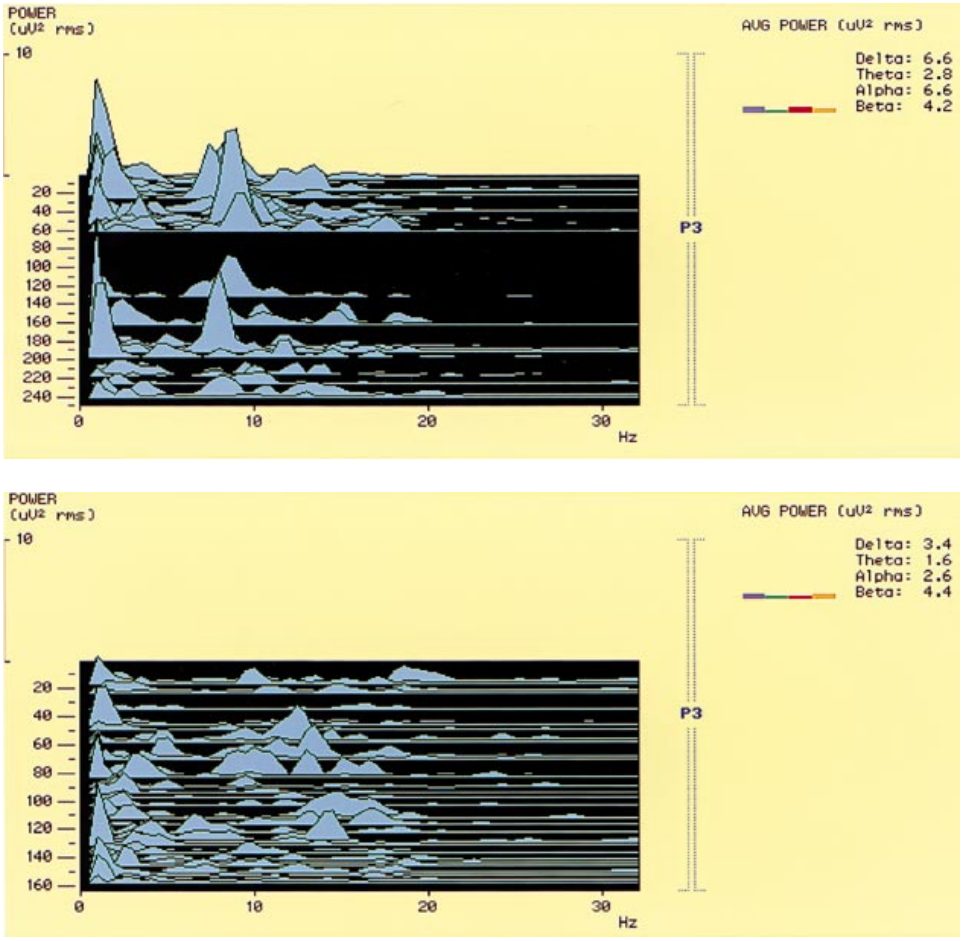

Fig. 2. Pretraining (A) and posttraining (B) reading compressed spectral arrays of power from P3. Improved alpha attenuation when reading is evident on the posttraining CSA.

statistical comparisons was set a priori at $p < .05$. In order to determine whether P3 rms theta amplitude decreased across sessions, a repeated-measures ANOVA was conducted using the posttraining baseline values from the three different training phases. The first training phase consisted of theta feedback from the P3 location, the second training phase consisted of theta feedback from the T3–C3 location, and third phase consisted of theta feedback from the P3 location again. Phase was entered as the within-subjects factor. The repeated measures ANOVAs revealed a significant within-subjects effect for phase, $F(2, 22) = 6.83$, $p < .01$. The mean P3 theta amplitudes for the three phases were 11.00 ($SD = 1.42$), 11.91 ($SD = 1.04$), and 10.17 ($SD = 1.18$), respectively. Post hoc testing revealed that the mean P3 theta amplitudes were significantly lower during the third training phase (the final 12 sessions) than both of the prior training phases and that the first two training phases did not differ from one another (see Fig. 3). In order to determine whether P3 theta amplitude decreased within sessions, a repeated-measures ANOVA was conducted on the three periods

Table I. Paired *t* Tests of Pre–Posttraining Mean RMS Power (μV^2)

Site	Band	Pretraining Mean	Pretraining *SD*	Posttraining Mean	Posttraining *SD*	*t*	*df*
Eyes-open							
P3	Delta	5.97	4.56	3.87	3.85	3.06*	73
	Theta	4.25	3.66	2.28	2.35	3.88*	73
	Alpha	12.39	11.37	6.89	8.50	3.38**	73
	Beta	4.62	3.75	3.78	3.43	1.40	73
Eyes-closed							
P3	Delta	9.09	6.32	4.26	3.36	6.01**	89
	Theta	4.64	3.19	3.35	2.22	3.36**	89
	Alpha	15.42	9.28	17.03	12.70	0.99	89
	Beta	4.21	2.45	4.79	3.96	1.18	89
Reading							
P3	Delta	6.63	4.83	3.43	2.28	3.15*	32
	Theta	2.84	1.82	1.55	1.32	3.44*	32
	Alpha	6.63	5.37	2.64	2.91	3.86**	32
	Beta	4.23	2.59	4.44	3.49	0.321	32
Linear channel combinations							
Eyes-open							
L. posterior	Theta	3.71	2.50	2.31	2.26	3.72**	73
R. posterior	Theta	2.02	2.04	1.59	2.23	1.17	73
L. frontotemporal	Theta	2.41	1.95	1.35	0.91	4.16**	73
R. frontotemporal	Theta	1.76	1.53	1.04	0.81	3.45**	73

Note. Left posterior combination = P3, T5, O1; right posterior = P4, T6, O2; left frontotemporal = F7, T3, C3; right frontotemporal = F8, T4, C4.
* $p < .01$. ** $p < .001$.

of each session from the third training phase (the final 12 sessions). The first period was the pretraining baseline, the second period was P3 feedback training, and the third period was the posttraining baseline. Period was entered as a within-subjects factor. The repeated measures ANOVAs revealed a significant within-subjects effect for period, $F(2, 22) = 3.26$, $p = .05$. The mean P3 theta amplitudes for the pretraining baseline, P3 feedback, and the posttraining baseline were 10.78 ($SD = 1.48$), 9.58 ($SD = 0.75$), and 10.17 ($SD = 1.18$), respectively. Post hoc testing revealed that the mean P3 theta levels were significantly lower during feedback than the pretraining baseline, and that the two baselines did not differ from one another (see Fig. 4).

The patient's reading speed was assessed by having him read short sentences (unrelated to the stories he read during training) from the Minnesota Test for the Differential Diagnosis of Aphasia (Schuell, 1965) and circling *yes* or *no* answers regarding their content. The pretraining reading duration was 8 min and 20 s, and the posttraining duration was 5 min and 28 s, which is a 34% increase in reading rate. However, both of these times were still within the extremely severely impaired range. Over the course of training, the patient reported his home reading progressed from initially reading first to second grade children's books to eventually reading short adult science fiction stories.

Table II displays the results of the pretraining (1-year post-CVA) and posttraining (16-months post-CVA) neuropsychological evaluations. The classification ranges are based on scales in Lezak (1983) and the Wechsler Adult Intelligence Scale—Revised (Wechsler, 1981). *T* scores should differ by at least 10 points (1 *SD*) to be considered significant (Rozelle & Budzynski, 1995). Significant pre–posttraining improvements in neuropsychological

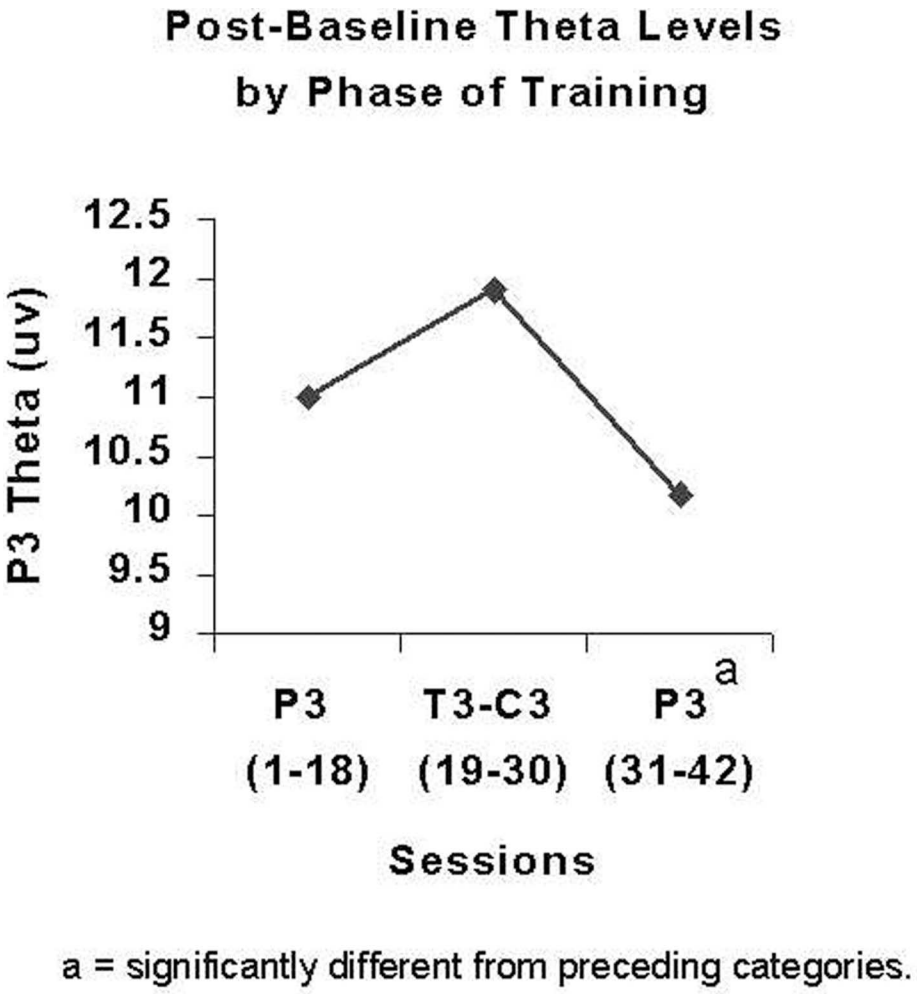

Fig. 3. Posttraining baselines of P3 theta amplitude in uv across training phases. Second phase training occurred from T3 to C3. Final phase differs significantly from previous two phases.

functioning occurred on the Trail Making A test (Reitan & Wolfson, 1993) and the Wechsler Memory Scale–Revised (WMS-R) Digit Span Forward and Backward tests (Wechsler, 1987). On the WMS-R Logical Memory test, immediate recall of the details of a story showed a significant decline. The Brief Symptom Inventory (Derogatis, 1993) depression scale score at pretraining was significantly elevated (T score of 71) and his posttraining score was no longer significantly elevated (T score of 61).

DISCUSSION

This clinical case study was an attempt to systematically examine the possible rehabilitative effects of neurofeedback training. The patient's infarction resulted in an unusual pattern of thalamic, left temporal, and left posterior cortical damage resulting in a number of

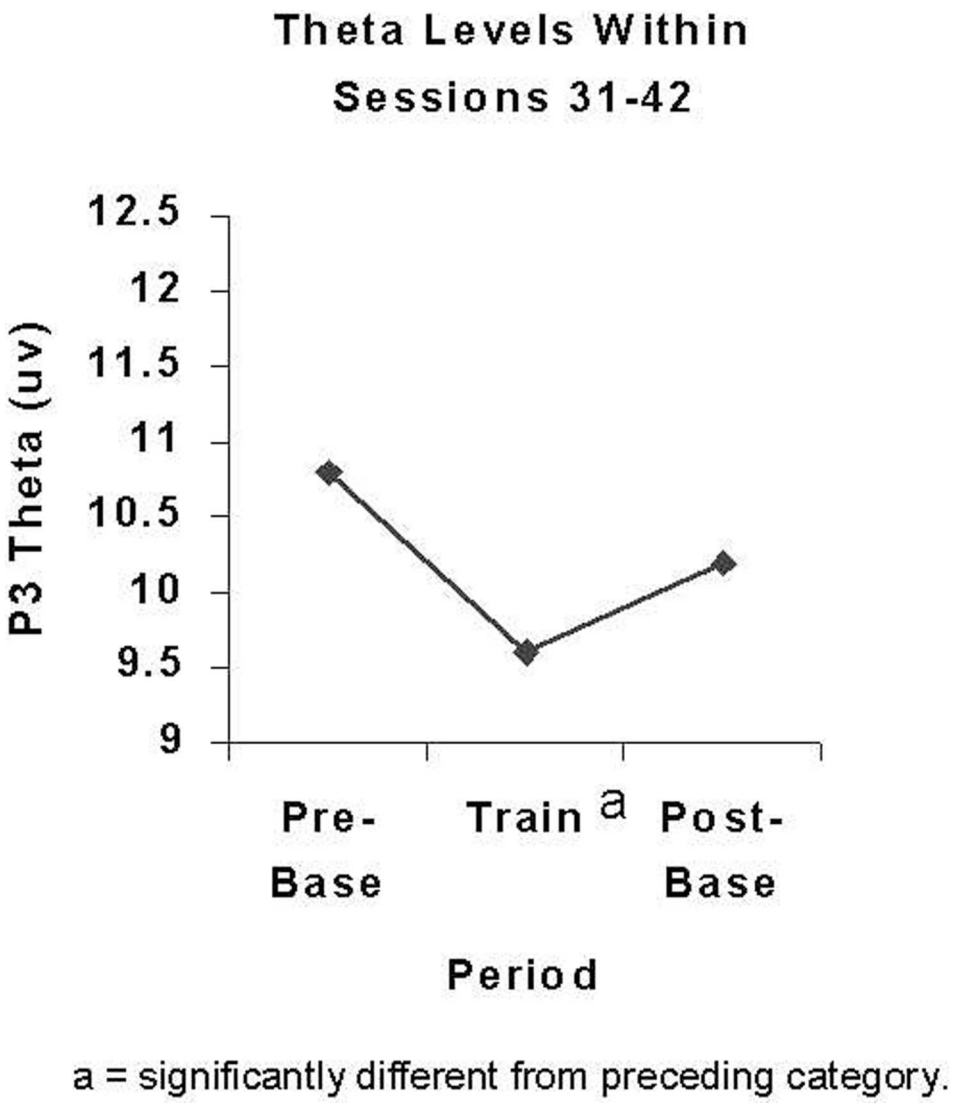

Fig. 4. Baseline and training P3 theta amplitudes within the final training phase. Mean training period amplitude differs significantly from the mean pretraining baseline.

neuropsychological deficits, including the very rare combination of alexia without agraphia. There are no published reports utilizing neurofeedback training with individuals demonstrating combined thalamic and cortical structural damage or with alexia without agraphia. Given that the thalamus is generally considered to be the generator source for most rhythmical cortical EEG activity (Cantor, 1999), it was felt neurofeedback training might be difficult but also possibly beneficial as well.

It was hypothesized that pre–posttraining QEEGs would demonstrate significant reductions in theta band activity and a general trend toward normalization. Clearly, there were significant P3 reductions in eyes-open and reading theta as well as delta and alpha band

Table II. Neuropsychological Evaluations With Results in *T* Scores and Range of Functioning

Test	Pretraining		Posttraining	
	T	Range score	*T*	Range score
Trail Making				
A	<14	V. Sev.	26	Mod.[a]
B	(unable to complete)		<13	V. Sev.
Grooved Pegboard				
Dominant Hand	22	Sev.	27	Mod.
Non-Dominant	30	Mild	34	Bord.
WMS-R				
Digit Span Forward	30	Mild	54	Ave.[b]
Digit Span Backward	38	Low Ave.	58	Hi. Ave.[b]
Logical Memory (Immediate)	51	Ave.	37	Low Ave.[a]
Logical Memory (Delayed)	39	Low Ave.	42	Low Ave.
WAIS -R: Block Design	43	Ave.	46	Ave.
Facial Recognition	>56	Hi. Ave.	66	Sup.
Rey Auditory Verbal Learning				
Trials 1–5	23	Sev.	28	Mod.
Trial 5	16	Sev.	23	Sev.
Short Delay Free Recall	18	Sev.	18	Sev.
Long Delay Free Recall	19	Sev.	19	Sev.

Note. Ranges of impairment include: very severe (V. Sev.), severe (Sev.), moderate (Mod.), mild (Mild), and borderline (Bord.). Other ranges include: low average (Low Ave.), average (Ave.), high average (Hi. Ave.), superior (Sup.), and very superior (V. Sup.).
[a] ≥1 *SD* change.
[b] ≥2 *SD* change.

activity. These reductions led to a normalization of the EEG as evidenced by the return of the alpha attenuation response to eye opening and reading. Interestingly, this same eye-opening normalization effect was observed by Rozelle and Budzynski (1995) after neurofeedback training with their patient who had suffered a left posterior CVA.

It was also hypothesized that reductions in theta amplitude would occur from the training sites across sessions. This hypothesis was partially confirmed by the significantly lower final training phase's P3 posttraining baseline amplitudes versus the two preceding phases' posttraining baselines. The lower amplitude of the final phase's posttraining baselines is suggestive of a generalization of training effects and is compatible with the lower amounts of theta activity found on the posttraining QEEG.

An additional hypothesis postulated that a number of posttraining neuropsychological tests would show significant improvements. In addition to a 34% increase in reading rate, three neuropsychological tests showed significant improvements. These tests encompassed measures of complex visual scanning with a motor component (Trail Making A) and immediate verbal recall (Digit Span Forward and Backward). Examination of *T* score changes on most of the other tests shows a trend toward improved neuropsychological functioning. These results are most likely not simply due to the passage of time, as there were no significant improvements between the patient's 6-month post-CVA neuropsychological tests and his pretraining (1-year post-CVA) tests.

The final hypothesis stated significant improvements in self-report measures of mood would occur. These improvements occurred as evidenced by a reduction of the patient's posttraining BSI Depression scale's *T* score to below clinically significant levels.

At the conclusion of training the patient demonstrated clinical improvements in verbal recall, reading, visual tracking, and emotional stability. A number of nonspecific factors could have influenced these improvements. The attention from a professional during the two to three training sessions each week and his sense of accomplishment at succeeding at a mental task, could have influenced his depression and possibly his neuropsychological test scores as well. However, these reward effects are not likely to be an explanation for his pre–posttraining-QEEG changes.

The pre–post-QEEG changes, especially in the eyes-open and reading conditions, are consistent with a trend toward normalization of electrocortical functioning. The functional significance of the relative normalization of the patient's posttraining reading QEEG is apparent from his concomitant 34% increase in reading rate. Increased normalization of the EEG in response to eye opening and reading is suggestive of an improvement in thalamocortical regulation. Improvement in subcortical to cortical regulation would be expected to result in not only cognitive, but positive affective changes as well (Othmer, Othmer, & Kaiser, 1999). In future studies, the use of multiple pretraining QEEGs over a time frame of several months would help differentiate possible neurofeedback training effects when significant differences are observed on the posttraining QEEG.

This case study adds to the extant literature supportive of the use of neurotherapy for rehabilitation of patients who have suffered CVAs. Additional neurofeedback studies with control groups, large numbers of neurotherapy patients, and long-term follow-up are necessary to fully ascertain the effectiveness of this therapy.

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