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## THE RELATION BETWEEN MEMORY IMPROVEMENT AND QEEG CHANGES IN THREE CLINICAL GROUPS AS A RESULT OF EEG BIOFEEDBACK TREATMENT

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It is important to understand the relation between changes in the quantitative EEG (QEEG) variables and memory changes as a result of the EEG biofeedback treatment. With this goal in mind, the senior author reviewed his clinical files from the last 5 years and examined the QEEG data addressing relative power and coherence changes and memory (auditory and reading) improvements. The groups involved included (a) normal individuals wanting to improve their cognitive functioning, (b) traumatic brain injured (TBI) subjects, and (c) + (d) subjects who can best be classified as having a specific learning disability (SLD). The SLD group was divided between those who are (c) older than 14 (adults) and those who are (d) younger than 14 (children) in order to reference the appropriate age-related normative group values. The analysis revealed significant improvements in auditory and reading memory across all groups as well as changes on the QEEG variables. All of the groups were performing above the normative reference group on measures of auditory and reading memory in terms of percentage differences (24-97%) and standard deviations (+1.28–1.85). The average auditory memory SD improvement was +1.52, whereas the average percentage change was 82%. For the reading task the average memory standard deviation improvement was 1.38, whereas the percentage improvement was 154%. The experimental group was performing 1.66 SD (68%) above the control group on auditory memory and .90 SD (52%) above the control group on reading memory measures. For the QEEG variables, the average raw value of the Spectral Correlation Coefficient (SCC) change for alpha was 6.1 points (2.09 SD), for SCC beta1 (13-32 Hz) 6.53 points (1.81 SD), and for beta2 (32-64 Hz) 7.5 points (1.77 SD). The changes on the relative power measures were less dramatic, albeit significant. These results underlie the importance of addressing the SCC values in EEG biofeedback treatment protocols.

## INTRODUCTION/THEORETICAL BACKGROUND

The relations between quantitative EEG (QEEG) variables and cognitive abilities require continued exploration. It is necessary for EEG biofeedback field to document the QEEG changes that are occurring as a result of intervention and specify the QEEG changes that are linked to the cognitive changes. There have been few research reports that address these relations other than the Thornton and Carroll (2010) article. In light of these goals, the senior author reviewed his clinical records during the past 5 years and entered all available QEEG and cognitive performance data into spreadsheets for analysis. The senior author employed spontaneous free recall of auditory presented material and visually presented reading text. The free recall task was chosen because it is a more difficult cognitive task than a recognition test and conceptually underlies much of cognitive functioning.

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The interventions employed the coordinated allocation of resource (CAR) model of brain functioning as originally discussed in a previous publication (Thornton & Carmody, 2009b). The model states that specific cognitive skills are a function of separate (albeit overlapping on occasion) resources of the brain's electrophysiology. The flashlight metaphor was first employed by Thornton (2002a) to organize the multiple Spectral Correlation Coefficients (SCC) relations. The metaphor states that each location is sending out a "flashlight" beam to all other brain locations, within a specific frequency. The interventions are guided by the data obtained during an activation QEEG evaluation. The activation QEEG evaluation engages the subject in difficult cognitive tasks that address auditory memory, reading memory, and problem solving as well as the standard eyes-closed visual and auditory attention tasks. The subject's values for the QEEG variables are compared to the normative database for deviations in general and specifically on those variables that have been documented to relate to cognitive performance in a normal population (Thornton, 2001). The variables that are most deficient and relevant are generally addressed initially. The SCC and phase (P) focus of the interventions has generally been directed toward posterior and T3 relative power issues, SCC and phase alpha to beta2 in the left hemisphere, right frontal areas and within the frontal lobes. Relative power interventions have focused on increasing beta1 or beta2 and inhibiting delta, theta, and alpha in the seven posterior locations. The results of the CAR approach have been reported previously in Thornton and Carmody (2005, 2008, 2009a, 2009b), Thornton (2000a, 2000b, 2002b), and Thornton and Carroll (2010). Empirical research reports that have provided a basis for the CAR approach have been reported in Thornton (2001, 2006a, 2006b, 2007) and Thornton and Carmody (2009a, 2010, 2012). The clinical data were organized around the clinical diagnostic categories of traumatic brain injury (TBI), adult specific learning disability (SLD), child SLD and normal participants.

## **QEEG MEASURES**

Over the years, research studies have generally defined the frequency ranges according to standard practice and have employed the scalp locations defined by the 10-20 system (Jasper, 1958). The frequency definition ranges have typically been as follows: delta: 0-4 Hz, theta: 4-8 Hz, alpha: 8-13 Hz, and beta: 13-25 Hz. The ranges have been dependent upon hardware and software definitions as well as the preferences of individual researchers. Some studies have examined frequencies above 32 Hz (Thornton, 2000, 2001, 2002a; von Stein & Sarnthein, 2000). This research employs the following frequency definitions: Delta (0-4 Hz), Theta (4-8 Hz), Alpha (8-13 Hz), Beta1 (13-32 Hz), and Beta2 (32-64 Hz).

There are two types of data available to QEEG analysis. The first involves the activity at a scalp location and examines the different frequencies in terms of measures such as magnitude (M), relative power (RP), peak frequency (PF), symmetry (Sym) and peak amplitude (PKA). The second measure quantifies the association between locations with concepts of P and SCC. This article employs these abbreviations to represent the variables employed for this analysis. This research examines the relative power values of the five frequencies and the SCC of the 8-64 Hz range (Alpha to Beta2) as these variables have proven, in the senior author's experience, to be the most useful in predicting and changing cognitive performance.

## **Activation Measures**

RP: Relative Magnitude/Microvolt or Relative Power: the relative magnitude of a band defined as the absolute microvolt of the particular band divided by the total microvolt generated at a particular location across all bands.

### **Connectivity Measures**

The algorithms employed in the Lexicor software generated the SCC and phase values obtained in this research. Different hardware and software companies have employed different algorithms in calculating these values. The results reported in this article for SCC relations using the Lexicor software are not necessarily the same results that would be obtained with algorithms provided by other equipment manufacturers. Frederick, Lubar, Rasey, Brim, and Blackburn (1999) presented an analysis of the different algorithms for the coherence measure and did not find consistent similarities between the approaches. The only published analysis between concurrent performance on cognitive activation tasks and the QEEG variables have been conducted with the Lexicor measures.

C: Coherence or SCC: the average similarity between the waveforms of a particular band in two locations over the epoch (1 s). The SCC variable is conceptualized as the strength or number of connections between two locations and is a correlation of the magnitudes. References employ a combination of letters. For example, CA refers to coherence (SCC) alpha, and RPA refers to relative power of alpha.

## **METHODS**

## **Participants**

Participants were patients (N = 59) at a general mental health clinic who had undergone an activation QEEG evaluation as part of the initial evaluation for attention deficit disorders, traumatic brain injury, learning disability, memory problems, and normal individuals seeking better cognitive performance. The mean age was 23.4, which ranged from 7.28 to 65 years. There were 21 female and 38 male participants, and 55 right-handed and four left-handed participants. Documentation regarding the diagnosis was typically not readily available (except with TBI), which renders accurate classification problematic. The TBI diagnosis was made by the senior author on the basis of reported history and neuropsychological and/or QEEG analysis. The classification category of SLD was based upon parental or self-report of the subject's history, psychoeducational reports (when available), and deviant QEEG values. The subject's QEEG data were examined for deviation from the normative

database to determine possible presence of the attention deficit disorder or attention deficit hyperactivity disorder pattern. The deviant QEEG value for attention deficit disorder/ attention deficit hyperactivity disorder was based upon the subject's relative power values of theta or alpha to be 1 standard deviation or greater than the normative reference group when averaged across all nine cognitive activation tasks: auditory and visual attention, auditory memory (input, immediate, delayed recall), reading memory (input, immediate, delayed recall), and problem solving (Raven's Matrices). There were 39 subjects (of 59) who demonstrated elevated delta, theta, alpha or combined theta and alpha elevations, or beta elevations. The average number of sessions for the participants was 45. This value is an underestimate of the actual number of sessions, as only the posterior relative power interventions and SCC and phase interventions (alpha to beta2) were employed for the estimate. Some additional interventions occurred only one to two times or addressed phase relations and were not included in the analysis. Table 1 presents the characteristics of the participants.

The Raven's Matrices are considered a viable test of intelligence (Snow, Kyllonen, & Marshalek, 1984) due to its psychometric properties. The correlation between the Raven's Matrices and initial auditory memory values was .24 (*ns*; n = 43), initial reading memory value +.12 (*ns*; n = 36). The Raven's measures did not correlate significantly with the percentage improvement on either the reading or auditory memory scores or the changes in the SCC values. The Raven's value was, however, positively correlated (.69; n = 13) with percentage change in relative power of beta2 during reading and with the percentage change (.94; n = 5) of relative power of beta1 during problem solving.

## **EEG Recording**

Brain activity was recorded using a 19-channel QEEG hardware device (Lexicor Medical Technology, Inc., Augusta, GA). Band-pass filters were set between 0.0 and 64 Hz (3 dB points).

TABLE 1. Participant Characteristics

Group	Ν	Male	Female	Avg. age	Hand	Ravens-avg.	*D	*T	*A	*B1	*B2	Avg. # ss
Normal	12	6	6	28.8	R = 11, L = 1	15.4	0	0	5	1	0	49
TBI	15	9	6	31.1	R = 15	10.6	1	2	7	2	2	48.3
Adult SLD	17	11	6	23.4	R = 15, L = 2	23.2	0	4	11	0	0	38.4
Child SLD	15	12	3	10.4	R = 14, L = 1	11.29	0	3	1	1	0	45.4
Total	59	38	21	23.4	R = 55, L = 4	15.1	1	9	24	4	2	45.3

Note. An asterisk indicates participants whose relative power values averaged across nine cognitive tasks were greater than 1 *SD* above the normative values and indicated frequency. Some participants were above the cutoff on two frequencies. Avg. age = average age calculated within and across groups; Hand = Handedness; R = right handed; L = left handed; Avg. = Average Raven's score (participant is administered up to 11 difficult Raven's Matrices problems and allowed 400 to 500 s to provide answers. Scoring employed the following method: 4 points if correct on first guess, 3 points if correct on second guess, 2 points if correct on third guess, 1 point if correct on fourth guess. The measure is generally considered a measure of nonverbal intelligence.); D = delta; T = theta; A = alpha; B1 = beta1; B2 = beta2; Avg. # ss = estimate of average no. of sessions, and # is an underestimate of actual number as sessions that addressed specific issues (other than CA, CB1, CB2, and posterior relative power values) were not included in the analysis; TBI = traumatic brain injury; SLD = specific learning disability.

The signals were analyzed with a Fast Fourier Transform, which uses Cosine-tapered windows and provides spectral magnitude in microvolts as a function of frequency. The sampling rate was set to 256 samples per second to allow for examination up to 64 Hertz. An Electro-Cap was fitted to the participant. The electrodes were positioned at 19 scalp locations according to the standard 10-20 system (Jasper, 1958) with linked ear references. The scalp was prepped with rubbing alcohol and Nuprep, and the 19 electrodes were filled with Electro-gel. The earlobes and forehead were prepped with rubbing alcohol and Nuprep. Impedances were maintained below 10 K Ohm at all locations. Gain was set to 32000, and the high-pass filter was set to off.

The measurements available through the software provided by Lexicor Medical provided the numeric values of the QEEG variables. The data were examined for artifact (eye movements and EMG activity) as well as other possible sources of contamination (Thornton, 1996) and marked for deletion in the analysis. The bandwidths were grouped according to the following divisions: Delta: 0.0–4 Hz, Theta: 4–8 Hz, Alpha: 8–13 Hz, Beta1: 13–32 Hz, Beta2: 32–64 Hz.

## RESULTS

The data were organized around four groups: (a) normal, (b) TBI, (c) SLD older than 14 (adult SLD), and (d) SLD younger than 14 (child SLD). The measure employed was spontaneous free recall, a more difficult neuropsychological measure for participants than recognition memory. Table 2 examines the auditory memory performance changes in terms of raw scores, percentage changes, standard deviation changes, and final performance level in comparison to the normative sample (SD and % differences). Table 3 examines the same values for the reading memory task. Some of the normal group might have been considered appropriate for the TBI of Adult SLD group. However, the TBI was considered a very old one, a minor one, debatable, or didn't show the typical SCC beta2 deficit pattern (Thornton, 1999). For the questionable adult SLD participants there was no psycho- educational data that would support the diagnosis.

The data were examined for effect size. Effect size quantifies the size of the difference between two groups (Coe, 2000). It quantifies the effectiveness of a particular intervention relative to some comparison and answers the question of how well does the intervention work. An effect size (ES) of zero means that the mean scores of two groups are identical, whereas an ES of 1 indicates that the mean scores of one group are superior to a second group by a value of 1 standard deviation. Some examples of other effect sizes show the overlap in the distributions of scores. An ES of 0.20 indicates that the treatment moved a subject from the 50th percentile to the 58th percentile,

Group	Pre tx M (SD) <sup>a</sup>	Post tx M (SD) <sup>b</sup>	p level M diff.	ES*	95% CI effect size*	Avg. # sess <sup>c</sup>	% change <sup>d</sup>	Norm value (SD) (sample size) <sup>e</sup>	Vs. norm SD diff./% diff. <sup>f</sup>
	19.5 (6.7) 11.7 (7.7) 12.8 (8.9) 11.33 (6.6) 14.45 <sup>7</sup> (7.93)	31 (6.7) 24 (5.8) 23.8 (6.0) 19.7 (6.1) 24.3 <sup><i>l</i></sup> (7.0)	<.001 <.001 <.001	1.75 1.42 1.28	0.73, 2.58 0.88, 2.62 0.66, 2.17 0.47, 2.09 0.90, .71	2.0 1.93 1.94 1.93 1.95 <sup>n</sup>	59% 105% 86% 74% 82% <sup>m</sup>	15.7 (5.12) (N = 47) 15.7 (5.12) 15.7 (5.12) 11.7 (6.55) (N = 35)	1.52/53% +1.46/52%

TABLE 2. Raw Score/Percentage/SD Improvement Values in Auditory Memory

Note. Asterisks indicates Hedge's unbiased estimate of effect size, with confidence intervals effect size. If values are above 0, then results are significant. ES = effect size; CI = confidence interval; TBI = traumatic brain injury; SLD = specific learning disability. <sup>a</sup>Initial average auditory memory value (immediate and delayed recall score) and standard deviation value (*SD*) of group.

<sup>b</sup>Posttreatment average auditory memory value and SD value of group.

<sup>c</sup>Average no. of evaluations employed to obtain posttreatment memory scores.

<sup>*d*</sup>The  $\frac{\sqrt{3}}{2}$  change from preevaluation values, that is, (post-pre)/pre.

<sup>e</sup>The average memory score of the control group.

<sup>f</sup>The SD of the posttreatment memory score compared to the normative database values as well as the % difference from the normative values.

 $^{g}N = 12.$ 

 ${}^{h}N = 14.$ 

 $^{i}N = 17.$ 

 $^{j}N = 14.$ 

kN = 57.

<sup>1</sup>Recalculated from the combined CSS Statistica file (Version 8).

<sup>m</sup>Weighted average of all groups, using N size and effect size.

<sup>n</sup>Employs the values (averaged) in the table.

Group	Pre tx M (SD) <sup>a</sup>	Post tx $M (SD)^b$	p level M diff.	ES*	95% CI ES*	Avg. sess <sup>c</sup>	Change <sup>d</sup>	Norm value (SD) (sample size) <sup>e</sup>	Vs. norm <i>SD</i> diff. <sup>f</sup>
Normal <sup>g</sup> TBI <sup>h</sup> Adult SLD <sup>i</sup> Child SLD <sup>j</sup> Total/avg <sup>1,m</sup>	3.34 (2.33) 2.23 (1.38) 1.72 (1.2) .94 (.95) 2.01 <sup>m</sup> (1.72)	6.72 (2.7) 5.41 (1.9) 5.48 (2.74) 3.06 (1.87) 5.10 <sup>m</sup> (2.62)	<.001 <.001 <.001 <.001 <.001	1.29 1.85 1.71 1.38 1.38 <sup>n</sup>	0.41, 2.17 0.94, 2.77 0.73, 2.69 0.56, 2.21 0.95, 1.82	1.6 1.69 1.54 1.67 1.62°	101% 143% 219% 225% <sup>k</sup> 154% <sup>n</sup>	3.61 (1.5) (N = 39) 3.61 (1.5) 3.61 (1.5) 2.47 (2.1) (N = 30)	1.47/86% 1.06/50% .88/52% .30/24% +.90/52% <sup>n</sup>

TABLE 3. Raw Score/%/SD Improvement Values – Reading Memory

Note. Asterisks indicate Hedge's unbiased estimate of effect size, with confidence intervals effect size. If values are above 0, then results are significant. ES = effect size; CI = confidence interval; TBI = traumatic brain injury; SLD = specific learning disability.

<sup>a</sup>Reading memory (immediate and delayed score combined) and *SD* scores of the original evaluation per 10s of reading (i.e., participant reads for 100s, if has a recall score of 10, then the 10-s reading memory value is 1).

<sup>b</sup>Mean and SD of posttreatment reading scores for 10s of reading.

<sup>c</sup>The average # of sessions employed to obtain posttreatment reading scores.

<sup>d</sup>The % change value (i.e., (post-pre)/pre).

<sup>e</sup>The normative values, *SD*, and *N* size for adults and children.

<sup>f</sup>The *SD* difference of the posttreatment measure compared to the normative database as well as the percentage difference.

 $^{g}N = 12.$ 

 $^{h}N = 13.$ 

 $^{i}N = 11.$ 

 $^{j}N = 14.$ 

<sup>k</sup>The high elevations for the CSLD group were due to two outliers whose respective % improvement was 1335% and 976%. The first participant improved from a .15 recall (per 10 s of reading time) to 2.15, whereas the second participant improved from .5 to 5.38 (per 10 s of reading time). These two brought up the overall average values as well.

 $^{\prime}N = 50.$ 

<sup>m</sup>Recalculated from the combined CSS Statistica file (Version 8).

<sup>n</sup>Weighted average of all groups, using total sample size.

<sup>o</sup>Employs the values (averaged) in the table.

whereas an ES of 0.50 means that the subject is now performing at the 69th percentile, and an ES of 0.80 means that the subject is now performing at the 79th percentile. Olejnik and Algina (2000) described the history of methods for calculating effects size. Although Cohen's (1969) effect size, d, was the first commonly recognized effect size representing mean differences in units of common population standard deviation, Glass, McGaw, and Smith (1981) proposed a modification of the Cohen d where the common standard deviation was replaced with the standard deviation of the control group. Further refinements were made by Hedges (1981), who suggested that a better estimate of ES would use the pooled variance and standard deviation rather than the standard deviation of one of the groups. Choice of the estimate of variance varies in the literature. Although some investigators use the variance of the control group, representing the population, others argue for a pooled estimate when there is no control group but rather two treatment groups and the population variance is unknown. As indicated by Coe (2000), use of the pooled standard deviation to calculate the effect size results in a value slightly larger than the true population value, which is corrected using a formula (Hedges & Olkin, 1985, p. 80). Although Cohen (1988, p. 25) warned that he arbitrarily chose values to classify the interpretation of size of the effect, many studies continue to interpret an effect size of .2 as a small effect, a .5 as a medium effect, and a .8 is a large effect (Coe, 2000). Improvements in interpretation are available using confidence intervals (CIs) that provide a range of values around the effect size to determine the likelihood of the effect size occurring due to chance. Greater accuracy of the effect size is more likely when based on a large sample rather than a small sample. Interpretation of the effect size is straightforward, namely, that if the CI includes the value of zero, then the effect size is statistically equivalent to no effect. In contract, if the CI does not include the value of zero, then the effect size is statistically significant.

Table 2 indicates significant improvement in all of the groups with an overall effect size change of +1.52 and a percentage change of 82%. The average standard deviation difference from the normative group after treatment was +1.66 or 68%. Thus all groups were performing above the control group by the end of treatment. This is confirmed by the effect size analyses showing that the 95% CIs do not contain the value of zero. A previously published article (Thornton & Carmody, 2008) reported that a control group of normal individuals (N = 15) with no history of a head injury showed no significant improvements in auditory memory when retested with 15 different stories.

Table 3 indicates significant improvement in all of the groups with an overall effect size of +1.38, percentage change of 154%, and average standard deviation difference from the normative group of +.90 and percentage difference of 52%. As with the auditory memory performance all the groups were above the control group at the end of treatment, confirmed by the effect size analyses.

Data (N=9) on reading improvement in previous reports on reading (Thornton & Carroll, 2010) were incorporated into this report, when data regarding reading time were available, due to a difference in calculation of memory improvement. The previous report (Thornton & Carroll, 2010) examined the total recall score without taking into account the amount of time employed in the reading tasks. The improved approach calculates how many elements are recalled in a 10-s period. As the original evaluation involved 100s of reading time and subsequent retesting time could involve 25 to 100 s, it was decided that a more appropriate reading memory score would be the number of recall elements recalled during a 10-s period. Thus if a subject spent 100s during the initial evaluation and recalled 10 elements, his score would be 1. If, on subsequent retesting, he recalled 10 elements in 20 s, his score would be 5, reflecting a significant improvement. There were three normal subjects, one TBI subject, two SLD subjects younger than 14, and one SLD subject older

Group	Auditory	Reading	r betw. AM%
	memory	memory	& RM%
	(N size) <sup>a</sup>	(N size) <sup>b</sup>	change (N size) <sup>c</sup>
Normal	.19 (12)	-39 (11)	.30 (11)
TBI	.64 Sig. (15)	-09 (13)	.27 (12)
Adult SLD	.20 (16)	.50 (11)	.53 (11)
Child SLD	-10 (14)	-08 (12)	.28 (11)
Avg. total	.19 (57) <sup>d</sup>	0.0 (47) <sup>d</sup>	.31 (Sig.) (45)

TABLE 4. Correlation Between Number of Sessions and % Memory Improvement

Note. TBI = traumatic brain injury; SLD = specific learning disability.

<sup>a</sup>Correlation in clinical sample between % auditory memory improvement and no. of sessions (sample size).

<sup>6</sup>Correlation in clinical sample between % reading memory improvement and no. of sessions (sample size).

<sup>c</sup>Correlation (r) between auditory memory % improvement and reading memory % improvement (sample size).

<sup>d</sup>Value recalculated for all groups combined.

than 14, for a total of seven subjects of the 59 subjects reported in this article.

Table 4 presents the correlations between the number of sessions employed and the percentage memory improvement (auditory and reading). Table 4 indicates a significant relation for the TBI group between the average amount of SCC alpha change and the number of sessions employed. Although the correlation between auditory and reading memory percentage change was significant (.31), the correlation is small, suggesting that these improvements are largely independent. This is consistent with the research (Thornton, 2000a, 2002a, 2006a) that documents a difference in

TABLE 5. Improvement in SCC Relations (Alpha to Beta2)

how these abilities function in terms of QEEG								
variables. Auditory memory is predominantly								
an issue of SCC alpha (left hemisphere and								
right frontal; Thornton, 2006a), whereas								
reading memory is predominantly an issue								
of F7 coherence and phase beta activity								

#### SCC Alpha to Beta2 Changes

(Thornton, 2002a).

Table 5 presents the average improvements in the SCC (alpha to beta2) relations during the interventions. Each SCC treatment session was analyzed for the amount of change from the evaluation baseline condition. For example, if the baseline SCC alpha was 70 and the session average was 75, then the session change value was 5. This value was averaged across all sessions addressing SCC alpha and the number of sessions addressing SCC alpha was calculated. Table 5 reflects these calculations.

Table 5 indicates that the SCC alpha change across the 4 groups was 6.1, for SCC beta1 the change was 6.53 and for SCC beta2 the change was 7.5. Employing the standard deviation of the normative reference group the change represents a standard deviation change of 2.09 for SCC alpha, 1.81 for SCC beta1 and 1.77 for SCC beta2. As the SCC values vary according to the distance between the locations, for both the normative and treatment sessions, the most reasonable way to compare the change (given the diversity of

Group	N	Avg. CA C	Avg. ss	N	Avg. CB1 C	Avg. ss	N	Avg. CB2 C	Avg. ss	CA <i>SD</i> change norm (SDC)	CB1 <i>SD</i> change norm (SDC)	CB2 <i>SD</i> change norm (SDC)
Normal	11	6.11	11.3	6	6.6	13.7	12	6.33	27.6	3.1 (1.97)	3.77 (1.75)	4.0 (1.58)
TBI	12	5.97	20.5	2	8.13	8	13	8.98	29.9	3.1 (1.93)	1.88 (2.16)	3.8 (2.24)
Adult SLD	13	5.97	15.9	4	6.74	5.5	13	7.65	21.9	3.1 (1.92)	3.76 (1.58)	4.0 (1.91)
Child SLD	13	6.4	19.4	6	6.04	10.7	11	6.83	18.9	2.5 (2.56)	3.5 (1.72)	5.02 (1.36)
Avg. total	49	6.1 <sup>a</sup>	16.8 <sup>b</sup>	18	6.53 <sup>a</sup>	9.47 <sup>b</sup>	49	7.50 <sup>a</sup>	$24.6^{b}$	$2.09^{b}$	1.81 <sup>b</sup>	1.77 <sup>b</sup>

Note. Avg. CA C = average Spectral Correlation Coefficient (SCC) alpha improvement across all sessions addressing SCC alpha; Avg. ss = average no. of sessions; Avg. CB1 = average SCC beta1 improvements across all sessions addressing SCC beta1; Avg. CB2 = average average SCC beta2 improvements averaged across all SCC beta2 sessions; CA SD change norm (SD change for clinical group) = SD change of SCC alpha of treatment group employing mean SD from normative group; CB1 SD change norm (SD change for clinical group) = SD change of SCC beta1 of treatment group employing mean SD from normative group; CB2 SD change norm (SD change for clinical group) = SD change of SCC beta2 of treatment group employing mean SD from normative group. TBI = traumatic brain injury; SLD = specific learning disability.

<sup>a</sup>Recalculated from the combined CSS Statistica file (Version 8).

<sup>b</sup>Employs the values (averaged) in the table.

Group	r between CA +AM % change (N) <sup>a</sup>	r between CB1 + AM % change (N) <sup>a</sup>	r between CB2 + AM % change (N) <sup>a</sup>	<i>r</i> between CA +RM % change ( <i>N</i> ) <sup>b</sup>	<i>r</i> between CB1 + RM % change ( <i>N</i> ) <sup>b</sup>	<i>r</i> between CB2 + RM % change ( <i>N</i> ) <sup>b</sup>
Normal	.18 (11)	.51 (6)	-18 (12)	-16 (10)	.21 (5)	-22 (11)
ТВІ	-19 (12)	<3 cases	-37 (13)	-01 (10)	<3 cases	-60 (Sig.) (12)
Adult SLD	.12 (13)	-75 (4)	-38 (13)	.65 (8)	-76 (4)	.16 (9)
Child SLD	-23 (12)	.32 (8)	.55 (11)	.46 (11)	.77 (Sig) (7)	.37 (10)
Avg.	$-05 (48)^{c}$	.22 (20) <sup>c</sup>	$-10 (49)^{c}$	.27 (39) <sup>c</sup>	.53 (Sig) (18) <sup>c</sup>	.12 (42) <sup>c</sup>

TABLE 6. Relations Between % Improvement in Auditory and Reading Memory and Raw SCC Changes

*Note*. SCC = Spectral Correlation Coefficient; TBI = traumatic brain injury; SLD = specific learning disability.

<sup>a</sup>Correlation (r) between SCC frequency changes and auditory memory (AM) percentage changes.

<sup>b</sup>Correlation (r) between SCC frequency changes and reading memory (RM) percentage changes.

<sup>c</sup>Employed entire Statistica spreadsheet to calculate.

the intervention locations) was to employ the overall averaged SCC value.

Table 6 presents the relation between raw score SCC changes and the percentage improvement in auditory and reading memory. Table 6 reports a significant negative relationship between raw score SCC beta2 average change and percentage reading memory improvement (-.60) in the TBI group. The other significant relationships involve SCC and percentage reading memory beta1 improvement (.77) for the child SLD and the overall average (.53) across all groups. It is of some interest to note that the SCC relations for the child SLD group were positive in five of six analyses. What is problematic, however, is why there are any negative correlations.

Table 7 presents the relations between the number of sessions directed toward the SCC relations and the average SCC change for the

 TABLE 7. Relations
 Between
 Number
 of
 Sessions
 Directed

 Toward SCC Relations and Average SCC Improvement
 Second
 Secon

		•	
Group	r between	r between	r between
	#CA ss	#CB1 ss	#CB2 ss
	and CA	and CB1	and CB2
	changes (N) <sup>a</sup>	changes (N) <sup>a</sup>	changes (N) <sup>a</sup>
Normal	.10 (11)	-50 (6)	.01 (12)
TBI	.42 (12)	<3 cases	-36 (13)
Adult SLD	.31 (13)	-45 (4)	-05 (13)
Child SLD	.86 (13) (Sig.)	-05 (9)	-62 (11) (Sig.)
All subjects	.47 (49) (Sig.)	-26 (21)	-21 (49)

Note. SCC = Spectral Correlation Coefficient; TBI = traumatic brain injury; SLD = specific learning disability.

<sup>a</sup>Correlation (*r*) between the number of SCC sessions (within a frequency) and the average SCC change.

three frequencies (alpha to beta2). Table 7 indicates a significant relation between SCC alpha changes and number of sessions directed toward SCC alpha in the child SLD group (.86) and the overall average (.47) across all groups and significant negative relations in the SCC beta2 frequency (-.62) for the child SLD group.

Table 8 addresses the relations between the number of sessions directed toward SCC relations and percentage improvement in auditory and reading memory for the four groups. Table 8 generally does not indicate a positive relationship between the number of sessions employed toward the SCC value of a specific frequency and the percentage improvement in either auditory or reading memory. The one exception to this is in the adult SLD group that had a .79 correlation between the number of SCC beta2 sessions and percentage reading memory improvement.

#### **Relative Power Changes**

Table 9 presents the initial and posttreatment relative power values for the posterior locations. The focus of most of these interventions was on the posterior locations (T5-P3-Pz-P4-T6-O1-O2) or specific locations within the posterior lobes, such as P3-Pz-P4. The data were included whenever any set of posterior locations was addressed.

Table 9 presents the changes in relative power values of the five frequencies in the posterior locations. The reason for this focus resides in the results of the original research (Thornton, 2001). The determination of the

Group	r betw. # ss SCC alpha & % AM change (N) <sup>a</sup>	r betw. # ss SCC beta1 & % AM change (N) <sup>a</sup>	r betw. # ss SCC beta2 & % AM change (N) <sup>a</sup>	r betw. # ss SCC alpha & % RM change (N) <sup>a</sup>	r betw. # ss SCC beta1 & % RM change (N) <sup>a</sup>	r betw. # ss SCC beta2 & % RM change (N) <sup>a</sup>
Normal	-41 (11)	.28 (6)	.35 (12)	-45 (10)	.22 (5)	.05 (11)
TBI	.44 (12)	<3 cases	-35 (13)	-01 (10)	<3 cases	.01 (12)
Adult SLD	.12 (13)	.53 (4)	.22 (13)	.23 (8)	.33 (4)	.79 (9) (Sig.)
Child SLD	-15 (12)	-43 (10)	-51 (11)	.12 (11)	-13 (9)	-25 (10)
Avg.	.13 (48)	-21 (22)	-09 (49)	.07 (39)	-13 (20)	-11 (42)

TABLE 8. Relation Between Number of Sessions of SCC Intervention and % Auditory and Reading Memory Improvements

Note. SCC = Spectral Correlation Coefficient; TBI = traumatic brain injury; SLD = specific learning disability.

<sup>a</sup>Correlation (r) between the number of sessions directed toward SCC values (alpha, beta1, beta2) and the percentage changes in auditory and reading memory.

specific frequency to be addressed was made based on the findings of the initial evaluation. Thus, if there was elevated theta in posterior locations during the listening task, the RP theta was inhibited during the treatment while, generally, the RP of beta2 was rewarded. A particular problem with the listening condition was that the subject's eyes were closed during the initial evaluation while the eyes were generally open during the training. Merely opening the eyes changes the RP values. To counter this problem, the first intervention session was employed as the baseline for the auditory changes. However, the first session

TABLE 9. Changes in Relative Power Values During Listening to Paragraphs as a Result of Treatment

Group	Avg. # ss	Listening	RP delta (SD)	RP theta (SD)	RP alpha (SD)	RP beta1 (SD)	RP beta2 (SD)
Normal	5.8	Pre-Tx	23 (2.7)	15.3 (2.9)	14.3 (4.3)	23.6 (2.4)	23.7 (6.1)
N = 5		Post-Tx	21.6 (6.7)	14.6 (3.2)	12.3 (2.6)	24.1 (3.8)	27.2 (4.3)
		Diff. Raw	-1.4	-63	-2.1	.52	3.6
		Diff. SD	-0.29	-2	-59	.16	.68
		Diff. %	-6%	-5%	-14%	2%	15%
TBI	2.67	Pre	27.9 (8.6)	15.1 (2.8)	15.4 (4.1)	22 (1.33)	18.6 (6.6)
N = 3		Post	21.1 (7.1)	14.4 (2.6)	15.1 (2.1)	28.5 (8.6)	18.8 (8)
		Diff. Raw	-6.8	-73	-26	5.5	.18
		Diff. SD	-87	-27	-08	1.11	.02
		Diff. %	-24%	-5%	-2%	+30%	1%
Adult SLD	3.25	Pre	26.7 (6)	15 (3.4)	14 (2.9)	24.9 (2.2)	24.7 (4.6)
N = 4		Post	28.5 (5.4)	15.5 (2.8)	13.8 (3.2)	24.4 (4.3)	23.8 (5.6)
		Diff. Raw	1.83	.47	-19	-55	-95
		Diff. SD	.32	.15	-06	-17	-18
		Diff. %	7%	3%	-1%	-2%	-4%
Child SLD	6.4	Pre	28 (8.2)	18.5 (3.1)	12.8 (2.7)	21.5 (3.6)	19 (6.8)
N = 8		Post	23.5 (9)	18.2 (2.8)	13 (3.6)	23 (2.8)	22.3 (4.3)
		Diff. Raw	-4.5	-31	.18	1.52	3.35
		Diff. SD	-52	-10	.06	.47	.60
		Diff. %	-16%	-1%	1%	7%	17%
Total avg.	4.53	Pre	26.5 (6.64)	16.5 (3.3)	13.8 (3.25)	22.3 (2.95)	21.25 (6.34)
N = 20		Post	23.69 (7.52)	16.21 (3.17)	13.29 (3.0)	24.39 (4.48)	23.32 (5.5)
		Diff. Raw	-2.8	-30	-53	1.46	2.07
		Diff. SD	-34	-10	-17	.39	.28
		Diff. %	-10%	-2%	-4%	9%	10%

Note. Avg. # ss = average no. of sessions addressing posterior locations; RP delta = average value of relative power of Delta (*SD*); RP theta = average value of relative power of Alpha (*SD*); RP beta1 = average value of relative power of Alpha (*SD*); RP beta1 = average value of relative power of Beta1 (*SD*); RP beta2 = average value of relative power of Beta2 (*SD*); Diff. Raw = raw score difference between post treatment and pre treatment value of the frequency; Diff. *SD* = standard deviation difference between post- and pretreatment (employing the *SD* of both pre- and posttreatment values); Diff. % = percentage change between the post- and pretreatment values of the frequency (i.e., (post-pre)/pre); SLD = specific learning disability.

#### **QEEG AND MEMORY**

was also a treatment session, presenting a problem in data interpretation. Therefore, the changes in relative power described here are a conservative estimate of the changes from baseline to the end of treatment.

The largest changes were in beta2; however, the effect size of .34 was not a significant change (p = .28), CI [-.28, 0.96]. Given that the CI includes the value of zero, the effect size is not significant, although those who use the Cohen classifications would classify it as mild. This is an important example for those who interpret effect sizes without the CI.

Table 10 presents the changes in relative power values during reading conditions as a result of treatment. The initial evaluation data were employed as the baseline data, which is a more valid approach than the baseline condition for the auditory memory. Change scores in relative power for the five bandwidths were obtained. A multivariate analysis of variance was applied to the change sores to determine

Reading

Avg # ss

Group

whether changes were significant and to identify the affected bandwidths. An overall significant finding, Wilks's  $\lambda$ , F(4, 13) = 5.83, p = .007,  $\eta^2 = .64$ , led to post hoc tests. The only bandwidth that changed significantly was beta2 (*M* change = 6.12, *SE* = 1.35), 95% CI [3.30, 9.03]. Figure 1 shows the mean and variance estimates of change in relative power by bandwidth.

The results indicate nonsignificant reductions in delta and alpha that are accompanied by the significant changes in beta2, a bandwidth measured by few research reports. The figure indicates significant changes in all groups except adults with specific learning disorder.

Table 11 presents the changes in relative power values during problem solving as a result of treatment. The initial evaluation data were employed as the baseline data. The change scores in relative power the five bandwidths were obtained by subtracting the baseline power from the posttreatment power.

RP beta1 (SD)

RP beta2 (SD)

TABLE 10. Changes in Relative Power Values During Reading Conditions as a Result of Treatment

RP delta (SD)

Normal	3.5	Pre	23.5 (5.6)	17 (3.3)	14 (6.3)	21.8 (2)	19.4 (7)
N = 2		Post	21.3 (5.73)	17.7 (1.24)	12.5 (1.6)	23.8 (5.4)	21.7 (7.2)
		Diff. Raw	-2.23	.71	-1.55	2.12	2.35
		Diff. SD	-39	.32	-39	.57	.33
		% Change	-9%	14%	-11%	9%	12%
ТВІ	4.5	Pre	24.47 (6.24)	13.7 (3.6)	15.2 (6.97)	24.65 (4.42)	17.64 (6.5)
N = 5		Post	21.83 (5.23)	14.7 (3.5)	15.08 (2.5)	27.2 (3.6)	26.7 (5.7)
		Diff. Raw	-2.64	1.03	-13	2.58	9.07
		Diff. SD	-46	.29	-02	.64	1.49
		% Change	-11%	7%	-0%	10%	51%
Adult SLD	3.67	Pre	23.7 (2)	15.8 (2.3)	17.3 (8.2)	24.3 (2.2)	18.9 (8.2)
N = 3		Post	26.8 (5.1)	16.9 (3.5)	13.4 (4)	25.6 (3)	23.8 (3.9)
		Diff. Raw	3.14	1.05	-3.9	1.33	4.89
		Diff. SD	.88	.36	-64	.52	.80
		% Change	13%	7%	-23%	5%	26%
Child SLD	3.85	Pre	25.5 (3.71	16.85 (2.66)	12.6 (2.5)	22.6 (1.03)	18.7 (2.6)
N = 7		Post	22.4 (7.9)	17.1 (2.63)	11.9 (1.52)	24 (3.6)	24.4 (4.95)
		Diff. Raw	-3	.23	-79	1.46	5.73
		Diff. SD	-52	.09	-39	.62	1.51
		% Change	-1%	1%	-6%	6%	30%
Total avg.	3.88	Pre	24.64 (4.24)	15.75 (3.05)	14.39 (5.3)	23.38 (2.73)	18.49 (4.99)
$N = 17^{-1}$		Post	22.91 (6.27)	16.4 (2.95)	13.16 (2.56)	25.23 (3.64)	24.65 (5)
		Diff. Raw	-1.72	.67	-1.24	1.84	6.17
		Diff. SD	-33	.22	-31	.58	1.23
		% Change	-7%	4%	-8%	8%	33%

RP theta (SD)

RP alpha (SD)

ing beta2 and decrease delta, theta, alpha; RP values = relative power values of the frequency with SD value; Diff. Raw = change in raw relative power value; Diff. SD = change in SD units employing the SD of initial and evaluation values; % change = change in % values, that is, (post-pre)/pre values; TBI = traumatic brain injury; SLD = specific learning disability.



**FIGURE 1.** Confidence intervals for relative power changes in reading memory tasks. *Note*. CI = confidence interval; Delta Dif = change in relative power of delta; Theta Dif = change in relative power of theta; Alpha Dif = change in relative power of alpha; Beta1 Dif = change in relative power of beta1; Beta2 Dif = change in relative power of beta2.

A multivariate analysis of variance was applied to the change sores to determine whether changes were significant and to identify the affected bandwidths. An overall analysis was not significant, Wilks's  $\lambda$ , F(1, 3) = 18.31, p = .170; however, the partial eta-squared was quite large (.98), leading to examination of the CIs for the bandwidths. The only bandwidth that changed significantly was beta2 (M change = 9.83, SE = 2.91), 95% CI [0.58, 19.10]. Caution is suggested; whereas the effect size is impressive at 1.33, the 95% CI [-0.20, 2.86], includes the value of zero and thus the small sample size does not adequately support the finding. By reducing the CI to 90%, the limits of effect size reduce to 0.05 to 2.62 and the value of zero is not included; therefore, under the 90% CI, the effect size is reliable. Larger samples would be required to confirm the finding.

Table 12 presents the correlation between the relative power changes and auditory memory percentage changes. Table 12 does not indicate any significant correlations between percentage changes in relative power and percentage auditory memory improvements. The initial evaluation eyes-closed data were not employed as the baseline/initial treatment session (eyes-open) was employed, thus possibly confounding the problem of measurement as the baseline involved the first treatment session.

Table 13 presents the correlations between the percentage relative power changes and percentage reading memory changes. Table 13 indicates significant correlations between the percentage relative power values of beta1 and percentage reading memory improvement in the SLD groups (adult and children).

Table 14 presents the relation between initial reading memory and relative power values during evaluation. Table 14 indicates that a significant relationship (1.0) between relative power of beta2 and reading memory during the initial reading evaluation in the adult SLD group.

## CONCLUSIONS/ANALYSIS OF THE INTERRELATIONS BETWEEN QEEG VARIABLES AND COGNITIVE PERFORMANCE

Statistical analysis of the relations between the QEEG and cognitive variables was undertaken with CSS Statistica (Version 8). The following conclusions were obtained with the analysis.

Group	Avg # ss	Problem solving	RP delta (SD)	RP theta (SD)	RP alpha (SD)	RP beta1 (SD)	RP beta2 (SD)
Normal N = 4	3.75	Pre Post Diff. Raw Diff. <i>SD</i> % change	25.2 (4.6) 18.9 (7.9) -6.3 -1.0 -25%	14 (3.6) 14.7 (2.5) .71 .23 5%	12.9 (3.7) 12.3 (1.1) -65 -26 -5%	21.3 (3.4) 25.62 (2.5) 4.3 1.4 20%	19.6 (5.6) 29.4 (7.1) 9.8 1.5 50%

**TABLE 11.** Confidence Intervals for Relative Power Changes in Reading Memory Tasks

Note. Avg # ss = average no. of sessions directed toward changing relative power values, generally interventions focused upon increasing beta2 and decrease delta, theta, alpha; RP values = relative power values of the frequency with *SD* value; Diff. Raw = change in raw relative power value; Diff. *SD* = change in standard deviation units employing the *SD* of initial and evaluation values; % change = change in % values (i.e., (post-pre)/pre values). TBI = traumatic brain injury; SLD = specific learning disability.

- 1. There were highly significant effects (Tables 2 and 3) of the intervention for both auditory (average effect size of 1.52) and reading memory (average effect size of 1.38) across all groups. Individual group effect sizes ranged from 1.28 to 1.85. The highest effect size values were obtained with the TBI group. All CI analysis indicated significant results. All the treated groups were significantly above the performance level of the control group by the end of the treatment period. The superiority to the control group ranged from .30 *SD* to 2.58 *SD*. Cohen (1988) considered any effect size greater than .80 as large.
- 2. To discern what could be the electrophysiological underpinnings of the gains, an analysis of the SCC relations and relative power changes (posterior locations) was undertaken from several points of view on the data. The following conclusions

**TABLE 12.** Relations Between % Relative Power Changes and %

 Auditory Memory Changes

Group	<i>r</i> between	r between	<i>r</i> between	<i>r</i> between	r between
	RPD %	RPT %	RPA %	RPB1%	RPB2%
	C +AM				
	% C <sup>a</sup>				
Normal <sup>b</sup>	-70	19	-22	+.68	+.09
TBI <sup>c</sup>	.58	.95	-50	-69	.46
Adult SLD <sup>d</sup>	-72	06	0.0	.49	.77
Child SLD <sup>e</sup>	.02	.03	.15	-09	-08
Total avg.	0.0	.15	.13	-16	.04

Note. TBI = traumatic brain injury; SLD = specific learning disability.

<sup>a</sup>Correlation (r) between percentage changes in relative power values of frequency and auditory memory percentage changes.

 $^{c}N = 3.$ 

 $^{d}N = 4.$ 

eN = 8.

can be stated from the data regarding the SCC relations.

- 3. There was a significant relationship (Table 4) between the number of sessions and percent auditory memory improvement in the TBI group (r = .64). There was an overall small significant correlation (r = .31) between the percentage auditory and percentage reading memory improvement, indicating that these abilities are largely independent.
- 4. There were (Table 5) significant gains in all the SCC (alpha to beta2) relations across all groups. Employing the control group *SD* values, the average gain for SCC alpha was 2.09, for beta1 1.81 *SD* and for beta2 1.77 *SD*, with an overall range of 1.36 *SD* to 2.56 *SD*.
- The relationship between the SCC changes and percent improvement in auditory and reading memory was examined in Table 6. There were significant correlations for the

**TABLE 13.** Relations Between Relative Power % Changes and Reading Memory % Changes

Group	r between RPD % C +RM % C <sup>a</sup>	r between RPT % C +RM % C <sup>a</sup>	r between RPA % C +RM % C <sup>a</sup>	r between RPB1% C +RM % C <sup>a</sup>	r between RPB2% C +RM % C <sup>a</sup>
Normal	<3 cases				
TBI (4)	-85	.75	.90	.66	.30
Adult SLD (3)	.90	.18	-84	1.0 (Sig.)	.90
Child SLD (6)	-71	-20	.15	.86 (Sig.)	.62

Note. TBI = traumatic brain injury; SLD = specific learning disability.

<sup>a</sup>Correlation (*r*) between percentage changes in relative power values of frequency and reading memory percentage changes.

 $<sup>{}^{</sup>b}N = 5.$ 

Group	r between IRPD +RMª	r between IRPT +RM	r between IRPA +RM	r between IRPB1 + RM	r between IRPB2 + RM
Normal	<3 cases				
TBI (4)	-43	.71	.94	-12	-83
Adult SLD (3)	-10	-95	-92	-79	1.00 (Sig.)
Child SLD (6)	-81	.66	.40	.17	.06

**TABLE 14.** Relation Between Initial Reading Memory and Relative Power Values During Evaluation

Note. TBI = traumatic brain injury; SLD = specific learning disability.

child SLD between percentage reading memory improvement and raw SCC beta1 changes (r = .77) as well as the relationship between changes in SCC beta1 and percentage reading memory improvement (r = .53) for all groups combined. There was also a significant negative relationship between SCC beta2 changes and percentage reading memory improvement in the TBI group (r = -.60).

- 6. The examination of the relation between the number of SCC alpha sessions and improvement in SCC alpha values (Table 7) was significant for the child SLD group (r = .86), negative related in the SCC beta2 frequency (r = -.62), and significant across all groups for the SCC alpha frequency (r = .47).
- 7. The examination of the relations between the number of sessions addressing SCC relations and percent improvement in auditory and reading memory (Table 8) revealed one significant relationship (r = .79) in the adult SLD group for the reading task.

The following conclusions from the data can be gleaned from the analysis of the relative power changes.

- 1. Significant relative power beta2 changes during the reading condition occurred across all groups and for all specific groups except adults with specific learning disorders (Table 10, Figures 1 & 2).
- 2. Significant relative beta2 values changes were evident in the problem solving task, when employing a confidence interval of 90% (Table 11).

- 3. There was no evidence of significant correlations between changes in relative power values and auditory memory improvements. However, the baseline condition was the initial treatment session because the initial evaluation involved an eyes-closed listening condition. This problem may have compromised the data (Table 12).
- 4. There were significant correlations between percentage change of the relative power values of beta1 and percentage reading memory in the adult and child SLD groups (Table 13).
- 5. There was a significant relationship between the relative power of beta2 and reading memory during the initial reading evaluation in the adult specific learning disorder group (Table 14).

An analysis was conducted that combined these research data with previously reported research (Thornton & Carmody, 2005, 2008) on auditory memory and reading memory. The combined sample size across these three studies was 86 with 38 TBI participants (for auditory memory measures), 19 child SLD participants, 17 adult specific learning disability, and 12 normal subjects (auditory memory measures). A total of 130 assessments (combining auditory and reading memory) were conducted. An average effect size value was obtained across all subjects and measures that employed a weighting method to control for sample size. The formula employed was (effect size \* sample size) for all results. All the comparisons were employed and summed and then divided by the total sample assessed. The average weighted SD effect size was 1.78.

An additional control group of normal individuals was obtained and reported in the



**FIGURE 2.** Mean change in beta2 bandwidth in reading memory after treatment by clinical group. *Note*. N = normal; TBI = traumatic brain injury; ASLD = adult specific learning disability; CSLD = child specific learning disability. (Color figure available online.)

Thornton and Carmody (2008) article. Fifteen normal subjects were administered 15 different auditory memory tasks. There was no significant effect of practice on the measure across the 15 trials, when the performance on the first eight stories was compared to the last seven stories.

### CONCLUSION

The need to document cognitive changes concomitant with QEEG changes was the focus of this research report. The data reported support the ability of the EEG biofeedback to significantly change memory performance and the QEEG values. The major focus of the interventions was on the SCC alpha to beta2 values and, secondarily (when appropriate), on the posterior relative power values. The posterior intervention focused on reducing delta, theta, and alpha and increasing beta2 relative power values when the initial evaluation indicated elevations in the three lower frequencies and/or deficient relative power beta2. This focus was substantiated by the reading findings (Figures 1 & 2, Table 10). The findings of significant changes in the SCC values must be presumed to underlie much of the improved cognitive functions, as the SCC values were the main focus of most of the interventions.

Although the CAR activation database EEG biofeedback approach appears to be a viable intervention, the combination of the EEG and tutoring approaches may offer the best alternative. The CAR activation database EEG biofeedback approach changes the ability of the brain to absorb information; it does not change the content of what the brain knows. Tutoring interventions provide the specific content that the brain needs to know in order to succeed in life. The combination of the approaches offers the best approach for the improvement of cognitive functioning. In addition, providing the relevant QEEG information regarding the brain's electrophysiological response during the tutoring interventions may provide the best information on the effectiveness of specific tutoring response interventions. The QEEG information can provide information on whether the tutoring intervention is changing the critical QEEG variables as outlined in the original Thornton (2001) publication.

This research reported the use of a control group for the normal subjects. The field of EEG biofeedback has often been criticized for lack of a sham or placebo control groups in the research that reports on changes in behavior (impulsivity, IQ scores, etc.) as a result of the intervention. However, this criticism is vacuous and untenable. The EEG biofeedback approach is operant conditioning (OC) of the EEG signal. Decades of research have documented the effectiveness of OC (in animals and humans). It is logically and scientifically unnecessary for the EEG biofeedback area to replicate this well-established finding. The criticism reflects a lack of awareness of the present status of the OC method and what is new, preferred evidence in the biofeedback arena.

Primarily, what is required is to show that (a) there are specific relations between the QEEG variables and specific cognitive abilities/behavior, (b) specific QEEG variables can be changed (via OC), and (c) changing specific QEEG variables results in improvements in specific cognitive abilities. This reported research has attempted to accomplish these more relevant and cogent goals.

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