Traumatic Brain Injury Rehabilitation: QEEG Biofeedback Treatment Protocols

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Abstract Interventions for improvement of cognitive problems in patients with traumatic brain injury (TBI) include electroencephalography biofeedback, also known as neurofeedback. Quantitative electroencephalography (QEEG) patterns are assessed in TBI patients and then compared to a database obtained from a normative population. Deviations in QEEG patterns from the normative group are the basis for an intervention plan. While QEEG patterns, obtained under an eyes closed, resting condition, provide information about deviations at rest, QEEG patterns obtained while the patient engages in cognitive tasks reflect specific deficiencies in brain functioning. This paper reviews and assesses QEEG patterns collected under both resting conditions as well as cognitive tasks. The article provides a theoretical and empirical base for QEEG interventions with TBI.

Keywords EEG biofeedback · Traumatic brain injury · Cognitive rehabilitation · Activation QEEG · Memory rehabilitation · Cognitive challenge

An estimated 5.3 million Americans (2% of the population) currently have disabilities resulting from a traumatic brain injury (TBI; Thornton and Carmody 2005). Typical symptoms include problems in memory, concentration, decision making, slowness, headache, fatigability, easily confused, mood vacillations, sleep difficulties, dizziness, balance problems, increased sensitivity to lights or sounds, vision problems, problems in smell or taste, nausea, and ringing in the ears (CDC 1999).

While some of these symptoms are subjective in nature, many have been objectively assessed with neuropsychological instruments. Traditional cognitive rehabilitation methods have not proven fruitful (Thornton and Carmody 2008, 2009; Duff 2004) while the electroencephalography (EEG) biofeedback intervention offers a treatment possibility (Duff 2004). Traditional EEG data collection involves the direct printing of the waveforms during the recording that is then read by the professional. The quantitative electroencephalography (QEEG) involves the digitization of the EEG signal, which is saved to the hard disk and subsequently analyzed mathematically. One study (Nuwer et al. 2005) asserts that there are no clear unique TBI, EEG or QEEG features, despite the research report (Thatcher et al. 1989) that validated a discriminant function equation across three independent samples of TBI patients and obtained a hit rate above 90% and the research reports of Thornton (1999, 2000, 2003) which reported similar results. QEEG differences were reported between concussed athletes (mean assessment time was 89 days post concussion) and control subjects under the postural task condition of standing up (Thompson et al. 2005). Concussed athletes showed decreases in EEG power in all frequencies. However, specific treatment protocols for the TBI patient need delineation and validation.

In this article, we discuss the coordinated allocation of resource (CAR) model of brain functioning, the QEEG patterns in the TBI patient, the relations of QEEG measures to cognition in TBI patients, and the effects of various EEG

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biofeedback interventions. We also discuss the relation of QEEG measures to brain structure and function obtained with neuroimaging techniques of diffusion tensor imaging (DTI), functional magnetic resonance imaging (fMRI), event related potentials (ERP) and the invalidity of the spontaneous cure concept.
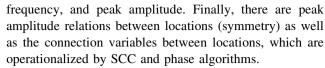
A Coordinated Allocation of Resource Model of Brain Functioning

A coordinated allocation of resources model (CAR) has been proposed to provide a conceptual understanding of brain processes (Thornton and Carmody 2008). The CAR model asserts that "cognitive effectiveness is a product of multiple specific activities in the brain, which vary according to the task" (Thornton and Carmody 2008). The multiple activities can involve different frequencies and different locations. The CAR model also involves a basic neuroanatomical and neuropsychological understanding of cortical location and function. The metaphor of a flashlight is employed, which states that a particular location generates a signal within a frequency range which is transmitted to all other 18 cortical locations. For example, in the case of reading in normal adults, memory performance is determined by SCC flashlight activity from F7 in the beta1 (13–32 Hz) and beta2 (32–64 Hz) frequency and by T5 SCC alpha relations to all other 18 cortical locations (Thornton 2002).

A clinically useful model of brain functioning should integrate the following criteria and research findings. Initially, the variables available for analysis require identification. The model should then ideally: (1) demonstrate one or more consistent patterns in TBI that are not present in other clinical or relevant conditions; (2) demonstrate that the patterns are specifically related to a relevant cognitive skill deficit in the TBI subject, such as memory, attention, or problem solving; (3) demonstrate that by changing the relevant QEEG variables then the related cognitive ability improves; and (4) demonstrate that the QEEG variable changes are long lasting.

QEEG Biofeedback Treatment Variables

There are many choices available to the practitioner using neurofeedback, which is the operant conditioning of the EEG signal. Among the choices are the 19 scalp locations in the 10–20 system (Jasper 1958) for measuring brain activity during baseline and treatment as well as the frequency bands (delta, 0–4 Hz; theta, 4–8 Hz; alpha, 8–13 Hz; beta1, 13–32 Hz; beta2, 32–64 Hz). In addition, there are several parameters of a frequency band that include microvolts (magnitudes), relative power, peak

frequency, and peak amplitude. Finally, there are peak amplitude relations between locations (symmetry) as well as the connection variables between locations, which are operationalized by SCC and phase algorithms.

After choosing the variables of location, frequency, parameters, and relations, there are choices as to the type of feedback to provide. These are to either inhibit or reward a single variable or simultaneously to reward and inhibit a set of variables. The number of possible reward only protocols is 2,945 when addressing data up to the 64-Hz, assuming the entire frequency range can be divided into five frequencies. If two variables are to be simultaneously addressed, such as rewarding one frequency band and inhibiting a second band, the number of possible protocols increases to almost 5,889. Any further divisions of these frequency ranges would only increase these numbers dramatically.

The conceptual and mathematical relations between the EEG waveforms in two or more different locations have been approached by difference theorists and EEG equipment manufacturers and have been termed coherence (Carter 1987; Collura 2008; Thatcher et al. 2005), co-modulation (SKIL 2008); and spectral correlation coefficient (SCC) as defined by Lexicor medical technologies (Joffe 1992). Some of the formulas have focused on data involving the entire epoch (period of time), segments within the epoch, timing issues between locations (phase), absolute or relative amplitude information, and wave form similarity and can involve the raw data, Fourier transformed, digitally filtered data, or involved complex demodulation. The SCC is a standard Pearson correlation of the amplitude data within a frequency band. The classical or "pure' coherence measure represents a generalization of the Pearson product correlation coefficient to variables expressed in the complex frequency domain and is calculated at the end of an epoch (Thatcher et al. 2005). The reader is referred to Collura (2008) for a more thorough discussion of these differences. This article focuses mainly on two specific formulations of these relations; classical or "pure" coherence as defined by Thatcher et al. (2001) and the SCC (Lexicor) algorithm, which was employed in the Thornton (1999, 2000, 2003) TBI research and is referred to as SCC in this article. The concept of phase employs the Lexicor definition and is sensitive to timing issues between the signals.

QEEG Patterns in TBI—Eyes Closed and Activation Patterns

The first criterion is to document a brain activation pattern under cognitive challenge conditions in the TBI subject that is not present in other clinical conditions or the normal population. The following discussion documents a QEEG

pattern in the TBI patient which is different from the normal population. However, there is no evidence to date indicating that the QEEG pattern in TBI does not exist in other clinical conditions.

Some of the related issues in this area concern the severity of the injury, how severity is defined and the nature of the data collected in determining differences; specifically, whether the data are collected under eyes closed, eyes open, or cognitive challenge activation conditions, as well as the frequencies examined (0–64 Hz or 0–22 Hz). Severity can be defined by the: (1) description of the subject immediately following the incident, (2) the physical damage as described by modern medical diagnostic tools, or (3) by the extent of the neuropsychological deficits (Thornton and Carmody 2008). There have been only two QEEG approaches, the eyes closed and the activation tasks, which have systematically examined the brain activity differences between the TBI patient and a normal population. Thatcher et al. (1989) employed an eyes closed normative database with TBI subjects and a 0.5–22 Hz frequency range while Thornton (2003) examined the differences between TBI subjects and a normal group under cognitive activation tasks with the frequency range of 0.0–64 Hz. Other available activation databases for reading and math conditions have been developed (SKIL 2008) but have not been employed in research with the TBI population.

Frequency Parameters

One study reported the presence of "decreased power differences between frontal and posterior regions and reduced alpha (relative power) in posterior locations" (Thatcher et al. 1989, p. 94) in the discriminant function analysis under eyes closed condition for TBI subjects within 1 year of injury. A study of the posterior activity of 18 subjects 3–10 days following a mild TBI (MTBI) found an increase in the mean power of the lower alpha range (8–10 Hz) and reduction in fast alpha (10.5–13.5 Hz) with an accompanying shift of the mean alpha frequency to lower values as well as a reduction in fast beta (20.5–36 Hz) activity (Tebano et al. 1988). Generalized, mild, nonspecific slowing of brain activity, with a focus in the temporal-frontal and temporal-occipital regions, appears in follow-up data ranging from 1 to 22 years post injury in TBI subjects (Hooshmand et al. 1989). In addition, the most common type of abnormality was in the asymmetry measure.

A review of the relations between TBI and brain activation obtained with eyes closed data concluded that "There is a broad consensus that increased focal or diffuse theta, decreased alpha… and increased asymmetry are common EEG indicators of the post-concussion syndrome" (Hughes and John 1999, p. 198). Specific asymmetry

findings are of limited clinical use, in Thornton's (2001) data, as there were limited findings of significance in the activation tasks. In addition the protocol is difficult to implement, at least as defined by the activation database as it involves the simultaneous relations between one location and 18 other locations in the 10–20 system. Thornton (1999) found increased theta relative power at locations O1–O2 under eyes closed condition. In summary, the increase in slow frequencies (delta, theta and slow alpha 8–10 Hz) and decrease in fast alpha (10.5–13.5 Hz) and beta levels appears to be the pattern in the eyes closed data in the TBI patient.

The activation database is composed of the QEEG values for microvolt, relative power, peak amplitude, peak frequency, symmetry, SCC and phase (means and SD) for a group ($N = 100$) of normal (no history of neurological diagnosis, learning disabilities, ADD, ADHD, etc.) individuals who have undergone a number of cognitive tasks (auditory and reading memory, problem solving, visual and auditory attention) while the values on their QEEG variables were simultaneously being collected (Thornton 2001). This group provides the normative database for the eyes-open resting condition as well as the activation database obtained from these subjects during cognitive challenges of attention and memory. The QEEG variables under eyes-closed resting and non-cognitive activation tasks which were positively correlated with subsequent memory performance were not the same as those variables that accounted for success during the memory task (Thornton and Carmody 2009). For example, the relative power of frontal and central theta under eyes closed condition was positively correlated with subsequent auditory memory ability. However, in contrast, the relative power of theta value from an eyes closed condition has shown a negative relation with academic performance (Harmony et al. 1990) in a group of normal and learning disabled children. Thus, the discrepancy between these results possibly resides in: (1) normal versus learning disabled and (2) eyes closed versus activation conditions.

Long term effects of TBI on brain functioning have been examined. For example, Randolph and Miller (1988) examined EEG of head injured and control subjects at 2–4 years post injury on several cognitive tasks while monitoring locations T3, T4, O1, and O2. The head injured group relative to controls had increased EEG amplitudes and amplitude variances particularly in the beta band, with no significant differences in relative power of the bands between the two groups. In other studies of long term effects, Thornton (1999, 2000, 2003) reported significantly lowered values in the TBI group (with a post injury time frame ranging from <1 to 43 years), compared to normal controls in SCC and phase beta2 (32–64 Hz) relations under eyes closed condition (1999), auditory and visual

attention (2000) and listening to paragraphs condition (2003), predominantly from frontal locations. Previous research (Thatcher et al. 1989) with the eyes closed condition had not examined these relations. In addition there were higher values of relative power of beta1 (13–32 Hz) and beta2 (32–64 Hz) with a frontal focus (all except F7), compared to controls, under the cognitive tasks of listening to paragraphs (8 of 9 frontal locations) and silent immediate recall (9 of 13 frontal locations) of the paragraphs. The delayed recall of paragraphs showed a greater right frontal, right hemisphere and posterior focus to higher relative power of beta2 (32–64 Hz) values compared to the normative group.

Thus, in the previous two cognitive activation studies cited (Randolph and Miller 1988; Thornton 2003), the TBI subject shows increased beta (amplitudes and/or relative power) compared to normals. The three eyes closed comparisons reported specific decreases in beta (20.5–36 Hz) and high alpha (10.5–13.5 Hz; Tebano et al. 1988), alpha (8–13 Hz; Thatcher et al. 1989), and increases in low alpha (8–10 Hz; Tebano et al. 1988) and theta (4–8 Hz; Thornton 1999). The pattern of increased beta activity under activation conditions in a TBI subject would strongly argue against protocols which increase beta activity. This argument would hold for both microvolts and relative power due to the positive inter-correlations between these measures (from +0.38 to +0.53; Thornton and Carmody 2009).

SCC, Phase and Coherence Relations

Due to the lack of equivalence of coherence algorithms in the field, interpretations of the research results with respect to coherence are problematic. The two main researchers (Thornton, Thatcher) who have addressed this population have not employed the same algorithm to measure coherence. As a result of these difference formulations, those investigators who are involved in TBI research studies have reported differences with the TBI group which appears contradictory. For example, while Hughes and John (1999) reported decreased coherences (frequency, locations and algorithms not specified) in the TBI subject in their review, others have reported increased theta coherence (Thatcher et al. 1989) and SCC theta values (Thornton 2000) in the left frontal region.

The Thatcher et al. (1989) discriminant function with eyes closed data indicated increased coherence and decreased phase in right frontal beta values (F3-F4, Fp2-F4) and increased left hemisphere beta coherences (T3-T5, P3-C3). The Thatcher et al. (2001) study found increased coherence values (eyes closed condition) (Delta—Fp1-C3; Theta—Fp1-Fp2; Alpha—O2-T6; Beta—P3-O1) and increased phase values (Theta—T3-T4; Alpha—F7-F8; Beta—T5-T6) and decreased coherences (Alpha—F7-O1)

and phase (Theta—Fp1-T3; Alpha—F7-O1) were related to a more severe injury within a TBI sample. Severity of injury was judged according to Glasgow coma score (GCS), duration of coma, and length of post traumatic amnesia information. Leon-Carrion et al. (2008) reported decreased beta (12–30 Hz) coherences in a TBI sample.

The Thornton studies indicated decreased SCC beta2 (32–64 Hz) and phase beta1 and beta2 values (Thornton 1999, 2000, 2003) with increased frontal SCC and phase theta values (Thornton 1999). The initial studies (Thornton 1999, 2000) reported the SCC problems predominantly within frontal locations while the later report (Thornton 2003) employed the flashlight metaphor and found all 18 cortical locations involved in the deficit frequency. The activation data results appear to present a more consistent pattern of deficits.

The conflicting results between the coherence and SCC values reside in the employment of the higher frequency (32–64 Hz) in the Thornton studies and non-equivalent algorithms to measure the relations.

Relations Between QEEG Variables and Cognition in the TBI Patient

The second criterion is to document that the QEEG deficits in TBI are related to specific cognitive skills. The research examining this linkage has employed both eyes closed and activation QEEG data. The eyes closed methodology involves collecting QEEG and neuropsychological data at different times and then correlating the variables. Decreased neuropsychological test performance was associated with increased delta amplitudes (0.5–3.5 Hz) and decreased alpha and beta (7–22 Hz) amplitudes in eyes closed QEEG data (Thatcher et al. 1998).

A discriminant function was developed to distinguish QEEG differences among mild, moderate and severe TBI groups (defined by Glascow coma scale, coma length, length of post-traumatic amnesia) using eyes closed data (Thatcher, et al. 2001). The QEEG discriminant function distinguished between good and poor performance on multiple neuropsychological measures with poorer performance associated with higher TBI severity, as defined by the discriminant function. The discriminant function variables were a mix of increased and decreased coherence and phase values.

Activation Data—Frequencies and SCC

The activation methodology involves collecting QEEG and neuropsychological data at the same time. Using activation data, head injured subjects show impaired performances when compared to control subjects. For example, head-injured subjects showed increased EEG amplitudes (T3,

T4, O1, and O2), amplitude variances in the beta band and impaired task performance (Randolph and Miller 1988). Thornton (2003) found that the relative power of beta1 (13–32 Hz) activity, obtained under three cognitive conditions, was consistently a negative predictor of cognitive performance. Thus, in two studies either increased beta activity or beta variances were negative correlates of performance.

Only one study has addressed SCC activity under activation conditions. Thornton (2003) determined that there were positive relations between auditory memory ability and SCC beta2 levels in a combined group of normals and TBI subjects. Thus the major QEEG deficit in the TBI subject, namely lowered SCC and phase beta2, had a deleterious effect on their memory performance. This finding relates to criteria 1 (consistent pattern) and 2 (related to specific cognitive skill). However, it has yet to be determined whether another group, such as learning disability, would exhibit the same pattern and thus the finding does not totally satisfy criterion 1.

In conclusion the effects of a TBI upon cognitive performance appear to consist of a negative influence of increased beta activity and decreased SCC beta activity under activation conditions. Specific QEEG variables related to specific cognitive tasks has yet to be accomplished in the QEEG TBI literature, except for auditory memory (Thornton 2003).

Intervention Effects: Changing QEEG Variables Results in Improved Cognition

Criterion 3 requires evidence that changing the relevant QEEG variables will improve the related cognitive measure. Two groups of protocols are described and their measures of their effectiveness are reviewed by assessing the associations between QEEG variables and cognitive skill with eyes closed and activation databases. First, we review protocols that inhibit theta frequencies and enhance beta frequencies and then we review coherence, SCC and phase interventions.

Protocol 1: Research on Beta Enhancement and Theta Inhibition

The general design of this protocol is to reduce the microvolts or relative power of the theta frequency and to increase the microvolts or relative power of the beta frequency. In a single case study, the subject was treated for 31 sessions with two intervention protocols (Byers 1995). The first was designed to suppress 4–7 Hz while enhancing 12–15 Hz and 15–18 Hz beta microvolt activity at top central location on head (Cz). The second was designed to increase 15–18 Hz at T3 and C3 while inhibiting theta

microvolts (4–7 Hz). Improvements were found when comparing pre- and post-administered cognitive problem solving measures (Defilippis and McCampbell 1979; Heaton 1981) Category test, Wisconsin card sorting test (WCST) and other cognitive measures including verbal fluency and IQ scores. Theta microvolts decreased an average of approximately 37% across the three locations, T3, C3, and Cz on post testing. However, beta microvolts also decreased an average of about 41%, reflecting the problem of the interrelationships between the frequencies. This problem will be addressed later in this section.

In a group study (Tinius and Tinius 2000), 16 MTBI patients were treated on the basis of QEEG normative reference group data (Thatcher et al. 1989) by reducing theta activity (20 sessions) at location Cz (then C3 and C4, if necessary) if theta microvolt value was above the normative reference value; the amount of elevation was not reported. For the patients with low theta values (in comparison to the database), the intervention goal was to increase SMR microvolt activity (12–15 Hz). Additional interventions addressed coherence training. The locations and frequencies were defined by comparing each patient to the database. The protocols selected were designed to increase the patient's coherence values (frequency not specified) when database comparison indicated low values and to decrease coherence values (frequency not specified) when the database comparison indicated higher than normative values. A comparison to the control group showed that TBI patients improved their attention and problem-solving abilities.

Patients with moderate TBI receiving EEG biofeedback were compared to a matched control group receiving standard cognitive rehabilitation attention training (Keller 2001). The EEG protocol for 10 treatment sessions was to increase beta microvolts (13–20 Hz range) at location Fz. Eight of the twelve TBI patients increased their beta microvolt levels and sustained the level for longer periods of time, while the remaining four, who started with high beta levels, showed a decrease in beta microvolt levels. QEEG measures were made on the control group at the beginning and at the end of the study, which allowed for a comparison of brain activity changes with the experimental group. The control group neither increased their beta microvolt levels nor their performance on the post treatment attentional measures of letter cancellation, simple choice reaction task and a sustained attention task. The conclusion from these data is that microvolt measures "may not be the most important factor in cognitive change" (Keller 2001, p. 26).

Stephens (2006) employed the Fz, Cz, P4 and C4 locations and inhibited theta microvolts (4–7 Hz) in some patients and alpha (7–13 Hz) in others while rewarding SMR microvolts (12–14 Hz) at those locations. As the
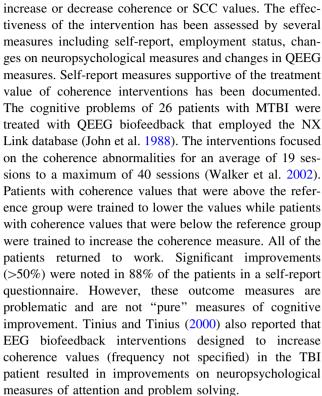
intervention protocols were not consistently tied to the QEEG database analysis, this research was treated as a standard QEEG method due to its consistency in inhibiting theta and rewarding low beta (12–14 Hz) microvolts. Across the six subjects on whom a post QEEG was conducted the consistent finding was an increase in beta (12.5–25 Hz) microvolts at the F7 location. No significant improvements in cognitive function were obtained.

The Flexyx system (now called LENS), a QEEG biofeedback program that provides extremely low energy electromagnetic stimulation based on the dominant EEG amplitude, is designed to reduce EEG microvolts (Schoenberger et al. 2001). Wait list controls were compared to subjects who received 25 sessions of treatment. The treatment group significantly improved their performance on attention measures.

The conflicting results of these five studies prevent drawing a firm conclusion. This is due to the different variables in the design of the interventions, as well as in the outcome measures. Specifically, two of three reports reduced theta and obtained improved cognitive scores; three reports attempted to raise beta microvolts and obtained either lower or no change in beta values, while two of the three reports showed no cognitive changes. The theta/beta interventions have proven to be useful in the attention deficit and learning disability research. However, in these TBI reports it is not always clear exactly what is occurring for several reasons: (1) There is a positive correlation (+0.62) between theta (4–8 Hz) microvolts and beta1 (13–32 Hz) microvolts which includes part of the SMR frequency (12–15 Hz) for the eyes closed condition (sample size = 40). The relative power values for these respective frequencies, however, are negatively correlated (−0.39; Thornton 2007); (2) only one study employed a database, thus it is not known, in the other studies, whether the subject's values were below or above norm on these values. It is, therefore, difficult to know if the interventions are normalizing these values or increasing them to above or below normal levels. (3) The relation between the specific cognitive measure employed and the QEEG variables addressed has not been specifically delineated in previous research; (4) effective variables for normal adults predominantly involve SCC values, not beta microvolts or relative power values (Thornton 2001); and (5) beta1 (13–32 Hz) relative power values are negatively related to auditory memory performance in the TBI population (Thornton 2003).

Protocol 2: Coherence, SCC and Phase Interventions

The general design of this protocol is to modify coherence or SCC values to be within a range of database norms. Therefore, depending on the values obtained in the activation conditions, interventions would be designed to increase or decrease coherence or SCC values. The effectiveness of the intervention has been assessed by several measures including self-report, employment status, changes on neuropsychological measures and changes in QEEG measures. Self-report measures supportive of the treatment value of coherence interventions has been documented. The cognitive problems of 26 patients with MTBI were treated with QEEG biofeedback that employed the NX Link database (John et al. 1988). The interventions focused on the coherence abnormalities for an average of 19 sessions to a maximum of 40 sessions (Walker et al. 2002). Patients with coherence values that were above the reference group were trained to lower the values while patients with coherence values that were below the reference group were trained to increase the coherence measure. All of the patients returned to work. Significant improvements (>50%) were noted in 88% of the patients in a self-report questionnaire. However, these outcome measures are problematic and are not "pure" measures of cognitive improvement. Tinius and Tinius (2000) also reported that EEG biofeedback interventions designed to increase coherence values (frequency not specified) in the TBI patient resulted in improvements on neuropsychological measures of attention and problem solving.

The QEEG activation database was employed in 19 cases of mild to moderate TBI to identify specific deficits in functioning for treatment intervention (Thornton and Carmody 2005). The main variable that was identified was the deviation in the phase and SCC measures from the normative database. The predominant focus was the beta2 SCC value, although beta1 and alpha SCC values were also addressed. Delta and theta SCC values were never addressed. The deviations were addressed, one by one, with appropriate treatment protocols. The protocols employed the CAR model. Interventions were conducted until the subject's values were at the normative value or above. Interventions were not pursued which would lower the SCC value. The activation database approach obtained an effect size (SD) change of +2.62 in auditory memory functioning on measures similar to the Wechsler logical memory task (Thornton and Carmody 2008). This evidence satisfies criterion 3 which states that by changing the relevant QEEG variables (SCC and phase) the related cognitive ability improves (auditory memory).

Case Examples

Some specific case examples may be useful with respect to criterion number 3, changing a specific QEEG variables and improving a specific cognitive skills. A subject (case study #1) was a middle age stunt actress who had experienced multiple mild-moderate TBI injuries during her career. The activation evaluation revealed deficits in SCC

beta2 (32–64 Hz) projection activity from the right frontal (F4) to left posterior locations (T5-P3-O1) during reading recall conditions. This relation has been shown to be critically involved in reading recall ability (Thornton 2002). The QEEG interventions were directed towards this specific problem. In five sessions she improved the SCC values 6.32 SD and her reading memory improved some 91%, from a total (immediate and delay) score of 25, obtained in 100 s of reading time to a score of 44, obtained in 42 s of reading time. Therefore, she improved her memory as well as her reading speed.

Long Lasting Effects of Interventions

Criterion 4 indicates that the protocols need to produce long lasting QEEG and cognitive changes. The data presented involve post treatment data, which does not satisfy what is typically meant by long term follow-up. Only Stephens (2006) (p. 182) has systematically obtained data in this relevant area. She noted that "EEG biofeedback was more effective than cognitive rehabilitation in achieving the normalization of disregulated cerebral EEG… More sites showed a significant shift away from normalization following cognitive rehabilitation". The subjects were reexamined after the end of their treatment. Although improvements were obtained on post QEEG evaluations, there were no significant changes in cognitive abilities.

The case studies of two additional patients will illustrate the value of QEEG activation database guided interventions. The patients received only activation database guided EEG biofeedback and both improved on subsequent cognitive retesting. For the second case study (case study #2) (Thornton and Carmody 2005), the main protocol intervention involved 110 sessions focusing on Fz SCC beta2 (32–64 Hz) to all other 18 cortical locations, due to the subject's values being below the normative values (Thornton and Carmody 2005). The improvements were evident in the post QEEG eyes closed (administered during the end of the treatment period) which documented a global (across all connections) average flashlight gain (compared to pre QEEG eyes closed) of +3.66 SD in beta2 SCC values, +1.84 SD in SCC beta1 values and +1.29 SD in SCC alpha values (eyes closed values). Similar improvements were seen in phase beta2 (+2.46 SD), phase beta1 (+0.62) (13–32 Hz) and phase alpha (+2.30 SD) (eyes closed). The improvements also held under the listening to paragraphs condition (pre vs. post evaluations) with increased SCC beta2 values (+1.66 SD), increased SCC beta1 (+2.39 SD) and increased SCC alpha values (+0.92 SD). Global (all locations) relative power changes showed an increase in delta (+1.33 SD), decrease in theta (−1.81 SD), no change in alpha (−0.02 SD), a decrease in beta1 (−1.04 SD), and a small increase in beta2

(+0.34 SD). The findings reflect the intervention's capacity to generalize to other frequencies not specifically addressed. Cognitive changes were noted in terms of decreased errors (from 10 to 2) on the continuous performance test, Shipley IQ changes (from 101 to 123), paragraph recall raw score improvement (from 20.2 to 33.7), total recall score (from 47 to 61) on the California verbal learning test (CVLT) as well as other improvements and an increase in errors (from 64 to 81) on the category test (Thornton and Carmody 2005).

In the third case study patient, multiple protocols were employed, guided by the original evaluation, to reduce delta at T5 and P3 and increase SCC flashlight activity (alpha, beta1 (13–32 Hz), beta2 (32–64 Hz). The total number of treatment sessions was 167, and the cognitive focus of the treatment was on auditory and reading recall. A re-evaluation, conducted towards the end of the treatment period, revealed changes in the global averaged QEEG measures across all locations and nine cognitive tasks. Specifically, the changes were reductions in delta relative power measures (−0.49 SD) and increases in SCC flashlight values (alpha, +0.47; beta1, +1.13 SD; beta2, +1.19 SD). The subject's cognitive improvement included a decrease in errors on the Category test (from 77 to 56), Shipley IQ increase (from 99 to 113), improved total recall score on the CVLT (from 43 to 53) or standard score increase (from 25 to 41), decreased time (from 10 to 7 min) and errors (from 13 to 5) on a visual scanning test developed by Thornton, improvement in paragraph recall raw score value (from 22 to 36), improvement in raw reading recall scores from 5.5 to 18, no significant change on the CALCAP reaction time test or Michigan serial recall test of working memory, and poorer performance on the WCST.

While these three case subjects were not examined for long term follow-up post treatment, the results do indicate significant changes in QEEG functioning with concomitant changes in cognitive abilities. The main focus of treatment for Subjects #2 and #3 was auditory and reading memory. Although the CAR model offers a useful structure to the interventions, there are questions raised by the results of the interventions which do not fit into this model. For example, with the first subject, the treatment was predominantly directed at the SCC beta2 activity which was clearly deficient in the initial evaluation. However, in a normal population SCC beta2 is not a correlate of auditory memory performance. SCC beta2, however, is a positive correlate of auditory memory when combining the traumatic brain injured population with normals (Thornton 2003). The CAR model does not address this problem. In addition, it has been observed clinically that addressing a particularly deficient QEEG variable can improve functioning even though that variable is not related to memory performance in a normal population.

Integration of QEEG with Medical Imaging Research

The TBI patient has been under investigation with other medical imaging technologies, such as positron emission tomography (PET), DTI, fMRI and ERP. DTI examines the alignment of water molecules in determining white matter tracks. Factional anisotropy (FA) is a measure of the directional diffusivity of water made using diffusion tensor imaging. A low FA reflects low white matter organization. The DTI measure is particularly well suited to examine the white matter effects of a TBI. A striking research finding was reported by Rutgers et al. (2008) who employed DTI technology and found that MTBI cases (5.5 months post accident) had multiple white matter regions with reduced FA, predominantly involving cerebral lobar white matter, cingulum, and corpus callosum. These white matter bundles mostly involved the supratentorial projection fiber bundles, callosal fibers, and fronto-temporo-occipital association fiber bundles. Of all of the involved fiber bundles, the report indicated that 19.3% (of 249 white matter fiber bundles) had discontinuity on fiber tracking. This is a considerable and unexpected effect in the MTBI cases. Reduced FA is associated with learning and memory in moderate and severe TBI (Salmond et al. 2006). The overall white matter load, which is a measure of the total number of regions with reduced FA, was more strongly related to the domains of executive and memory function than individual locations, reflecting the diffuse, integrative nature of cognition and relations of white matter to cognition.

In one study, there was no significant relation between the time interval after injury and the DTI fiber tracking findings (Rutgers et al. 2008). This last finding implies that the brain does not "spontaneously" cure itself. There are six cross sectional design studies involving DTI (Bendlin et al. 2008; Rutgers et al. 2008), QEEG studies (Thornton 2000; Thatcher et al. 1997), ERP (Lavoie et al. 2004) as well as high-resolution T1-weighted imaging and DTI (Bendlin et al. 2008) supporting the interpretation that the brain does not "spontaneously" cure itself.

A further line of evidence from the medical imaging literature involves the "greater effort" that the TBI subject experiences when engaged in cognitive tasks, despite performance equal to the control group in some cases. This "greater effort" has implicated the frontal lobes in terms of fMRI measures (Scheibel et al. 2007; Braver et al. 1997; Turner and Levine 2008). The QEEG research findings of SCC deficits and increased beta activations, in particular frontal regions (Thornton 2003) overlap meaningfully with findings from other neuroscience studies in the TBI area.

Spontaneous Changes in the TBI Brain Over Time

If the TBI brain does not spontaneously repair itself, then what is the effect of time and what is the brain doing to heal itself? The time since injury is a variable which is useful in understanding how the brain does or does not reorganize or repair itself. Thornton (1999) examined the effect of the passage of time since injury on QEEG variables in TBI subjects. A discriminant function using an eyes closed database returned a 0.90 hit rate in distinguishing the two groups, which were composed of subjects under 1 year post accident (group a) and over 1 year post accident (group b). The results indicated that group b had decreased beta1 at frontal and left posterior locations (relative power, peak amplitude, magnitude) and in beta2 (32–64 Hz) at left posterior locations (magnitude, peak amplitude) compared to group a. Group b also had increased SCC alpha relations (posterior, T5 in particular) and increased posterior phase beta1 (T5 in particular) and frontal phase theta (F7) compared to group a. Thus, time increases SCC relations and decreases beta activation levels. It would appear that the brain's self-healing involves lowering the beta hyperactivation response pattern and returning to the use of SCC relations. However, particularly noteworthy, was the lack of spontaneous change in the SCC and phase beta2 patterns in the TBI subject, supportive of the conclusion that "time does not heal" (Thornton 2000). The SCC beta2 deficits represent the main effect of a TBI on the QEEG variables, although there was evidence of effects in the lower frequencies as well. Perhaps, then, a more apt description would be that "time does not heal, it adjusts."

Conclusions

Given the results of the preceding analysis, there is tentative evidence that the CAR model (coordinated allocation of resources) of brain functioning, when employed with QEEG biofeedback, provides an alternative to present intervention approaches. Although clinically useful in the attention deficit disorder and learning disabled population, interventions directed towards increasing beta activity in the TBI population do not appear desirable. Interventions should focus on increasing SCC values in the 8–64 Hz range. Further independent research initiatives are required with larger sample sizes, control groups and sham treatment groups to further explore QEEG biofeedback approach in the remediation of the cognitive problems of the TBI patient.

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