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Child Adolesc Psychiatric Clin N Am
14 (2005) 137–162

CHILD AND
ADOLESCENT
PSYCHIATRIC CLINICS
OF NORTH AMERICA

Electroencephalogram biofeedback for reading disability and traumatic brain injury

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Reading disabilities

Prevalence and costs

Reading disabilities present major challenges to the educational system. The estimated prevalence rate for learning disabilities is 15% of the student population [1], with 6.5 million children requiring special education in 2002 [2]. Approximately 63% of these special education children have specific learning disabilities or speech and language problems without a concomitant physical disability. Between 28% and 43% of inmates in adult correctional facilities require special education (versus 5% in normal population), and 82% of prison inmates in the United States are school dropouts [3]. Large financial and social costs are associated with programs to address learning disabilities. The federal government spent \$350 billion over a 20-year period on special education programs [4], and New York City spends \$55,300 per year for each incarcerated youth [3].

Neuroscience of reading disability

The underlying physical basis of reading disability condition is confirmed in studies that examined the activity of neurotransmitters, magnetic fields, blood

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flow, and deviant response patterns on physical measures and neuropsychological instruments. Galaburda et al [5] conducted postmortem examinations on four dyslexic subjects and observed abnormal neuronal development (dysplasias, extra large neurons) along the left hemisphere superior temporal lobes (Peri-Sylvian regions) and frontal lobes.

Neuroimaging studies using positron emission tomography, magnetic electroencephalography, and functional MRI have identified differences in the functional organization between dyslexic and typical readers. Temple et al [6] and McCandliss and Noble [7] reviewed the literature on functional neuroimaging of dyslexia in adult and pediatric samples. A summary of the two reviews serves to identify the brain regions associated with dyslexia.

Dyslexic adults show dysfunction in the left temporoparietal cortex during phonologic processing of visual stimuli as evidenced by positron emission tomography studies [8]. Specifically, the dysfunction is located in the superior temporal gyrus and inferior parietal cortex, particularly in the left hemisphere [9,10]. Functional MRI studies confirmed this finding in adults who showed decreased activity in temporoparietal regions, including superior temporal gyrus and angular gyrus, during phonologic processing of letters and pseudoword rhyme [11]. Dyslexic children aged 8 to 12 years who underwent functional MRI showed reduced temporoparietal activity during phonologic tasks, which suggested that the disruption is fundamental to the disorder and is not a compensation effect that occurs with maturation.

In addition to identifying the areas of activation, magnetic electroencephalography provides data about the temporal course of activation. A regular progression of activation was found for normal and dyslexic readers from occipital, to basal temporal region, to the temporoparietal areas, which consists of the posterior portions of superior and middle temporal gyri and the angular and supramarginal gyri [12]. Dyslexic readers had onset latencies similar to normal readers in the activation of all areas except left temporoparietal, an area known to be involved in word recognition and phonologic analysis. These findings indicated that the left temporoparietal area is slow to respond and responds with less activation in dyslexic readers than in nonimpaired readers.

A magnetic electroencephalographic study was conducted on a sample of 45 children (5–7 years old) at the beginning of their reading experience who were either at risk for reading difficulties or not [13]. The imaging scans showed that children at risk had greater right hemisphere activity, whereas children not at risk had greater activity in left posterior superior temporal gyrus. These findings suggested that the dysfunction occurs early in development.

Several functional MRI neuroimaging studies have compared cortical activation patterns under reading-related tasks in readers with dyslexia and control groups of nonimpaired readers [11,14,15]. This series of studies showed that nonimpaired adults increased their activation in posterior superior temporal gyrus, angular gyrus, and supramarginal gyrus as the task demands increased from orthographic comparisons to phonologic comparisons [11]. In contrast, adults with dyslexia showed overactivation in response to increasing task

demands in anterior regions, including the inferior frontal gyrus. Whereas non-impaired readers showed activation of a widely distributed system for reading, the readers with dyslexia had disrupted activity in the posterior cortex, which involves traditional attentional, visual, and language areas.

The anatomic correlates of the dysfunction in left temporoparietal regions can be visualized by diffusion tensor imaging, which identifies white matter tracts [16]. Using diffusion tensor imaging, Klingberg et al [17] showed that reading ability is directly related to the degree of anisotropy (water diffusion and the direction of diffusion within each voxel) of white matter in left temporoparietal regions for readers with dyslexia and nonimpaired readers. There are functional equivalents of the structural connectivity. For example, in a positron emission tomographic study of adults, the nonimpaired readers—but not the readers with dyslexia—showed correlated activation between angular gyrus and lingual and fusiform gyri and the left superior temporal gyrus and left inferior frontal area [18].

In summary, the left temporoparietal region is disrupted in developmental dyslexia. The magnitude of activation is low, and there is decreased coordination of activity between the left superior temporal gyrus and left frontal areas. The evidence indicates that the disruption is in place before children learn to read, is related to difficulties with phonologic processing, and is related to underdevelopment of white matter fibers in the region.

Efficacy research on intervention programs for students with learning disabilities

Despite the enormity of the social and educational problem, the interventions currently used largely have been unsuccessful in obtaining significant and meaningful results. In 1988, Lyon and Moats [19] concluded that “It is difficult, if not impossible, to find any evidence beyond testimonials and anecdotal reports that support the assumptions, treatment methods, and stated outcomes associated with medical and psycho educational models . . . [T]here is overwhelming empirical and clinical data indicating that medical and psycho educational models, as they are presently conceived and used, are inadequate for determining what and how to teach learning disabled students.” More recently, Birsh [20] concluded that “despite the widespread inclusion of multisensory techniques in remedial programs for dyslexic students and a strong belief among practitioners using these techniques that they work, there was little empirical evidence to support the techniques’ theoretical premises.”

A comparison of the research results with current popular approaches indicates an average improvement of +0.34 standard deviations (SD) (standardized testing, 350 intervention hours, $n = 48$, control group) for the Orton-Gillingham program [21], an average improvement of 13% (standardized testing, 125 intervention hours, $n = 171$, no control group) for the Lindamood-Bell program [22], and an average +0.40 SD improvement (standardized testing, 100 intervention sessions, $n = 130$, control group) for the Fast ForWord program [23]. The Orton-Gillingham videotape obtained the same results as individual tutoring with

this method. Increases in reading abilities can be accompanied by magnetic electroencephalographic images, however, showing that after an intervention of 80 hours of one-on-one instruction in phonologic structure, children with dyslexia increased their activations in left posterior superior temporal gyrus, left supramarginal gyrus, and angular gyrus [24].

Traumatic brain injury

Prevalence and costs

An estimated 5.3 million Americans (2% of the population) currently live with disabilities that resulted from traumatic brain injury (TBI). Each year, 1.5 million Americans sustain a TBI, with a new case added every 21 seconds, which leads to 80,000 new cases of long-term disability and 50,000 deaths. Although the causes of TBI are many, the leading causes are car accidents (44%) and falls (26%), which involve adolescent, young adult, and elderly populations [25]. The costs of TBI in the United States are estimated at \$48.3 billion a year, with hospitalization costs of \$31.7 billion and fatality costs of \$16.6 billion.

Neuroscience of traumatic brain injury

Most studies on the biomechanical effects of closed head injury have concluded that three force vectors contribute to the injury: a rotational vector, a shear vector, and a centripetal force vector, which is maximal at the outer cortex with a gradient to the subcortex and brain stem. The geometrical summation of these forces results in maximum injury in that part of the brain that is in contact with the skull (eg, the gray matter of the frontal and temporal lobes), which largely occurs independent of the direction of impact to the skull. Two other invariant consequences of blunt force injuries to the skull are (1) sheer forces that are maximal at the boundaries between different densities of tissue (eg, gray versus white matter) and (2) a percussion shock wave that travels from the point of impact and makes contact with the opposite side of the skull in less than 100 milliseconds, which results in a “coup-contra-coup” injury. All these forces are capable of seriously disrupting the molecular integrity and function of cortical neurons and glia [26].

The theoretical interpretations of biomechanical effects of TBI have found support in modern neurodiagnostic testing and their correlates with cognitive function. For example, patients with TBI have increased delta amplitudes and increased white matter signal on T2 MRI indicating dysfunction, and there are associations of decreased alpha and beta amplitudes with increased gray matter T2 MRI relaxation times [27]. Although increases in both relaxation times were associated with cognitive dysfunction, decreased alpha and beta amplitudes also were associated with decreased cognitive function.

Commonly reported cognitive and psychological consequences of TBI include difficulties with “orientation/concentration, overload-breakdown of comprehension, reasoning and problem solving, organizational skills, rate of processing, rate of performance, perseveration (a tendency to repeat a response or activity after it has proven ineffective), staying on task/topic, initiation/motivation, generalization, agitation, fatigue, stress and memory (possibly the most common residual effect of brain injury and one that families generally find the most troubling)” [25].

Efficacy research on intervention programs for traumatic brain injury

The research literature on memory improvement in patients with brain injury generally has found minimal to mixed results for several intervention approaches. One of the initial reviews in this area concluded that “findings regarding the effectiveness of memory remediation interventions have been inconsistent,” adding that methodologic inadequacies have hindered the identification of specific treatment effects [28]. Memory is not improved by simple, repetitive practice [29] or by repetitive recall drills [30]. Specific techniques, such as visualization, method of loci, and cognitive strategies, have shown different degrees of effectiveness. Researchers generally agree that the subject does not continue the use of the strategy after treatment ends [31]. Significant improvements from internal memory aids, such as imagery instructions, are used less than external memory aids, but patients on their own generally use neither.

More recent reviews of the literature report similar mixed to negative conclusions on the efficacy of cognitive rehabilitation therapy for memory and other areas of cognition and behavior [32–34]. In their exhaustive review, Carney et al [32] concluded that “specific forms of cognitive rehabilitation reduce memory failures (notebook training/electronic cueing devices—results didn’t hold 6 months post treatment) and anxiety, and improve self-concept and interpersonal relationships for persons with TBI.” A recent Defense and Veteran’s Head Injury program study did not find any significant improvement on their measures as a result of cognitive rehabilitation (compared with control group) in patients with moderate to severe TBI [34]. In conclusion, no definitive scientific evidence indicates that cognitive rehabilitation leads to sustained improvements in memory.

Electroencephalogram and neurofeedback

What is the quantitative electroencephalogram?

The quantitative electroencephalogram (QEEG) is a digitization of the traditional analog EEG signal. Instead of the EEG oscilloscope tracings being printed directly onto paper, the computer obtains information on the waveform being generated, displays the signal on a computer screen, and saves that

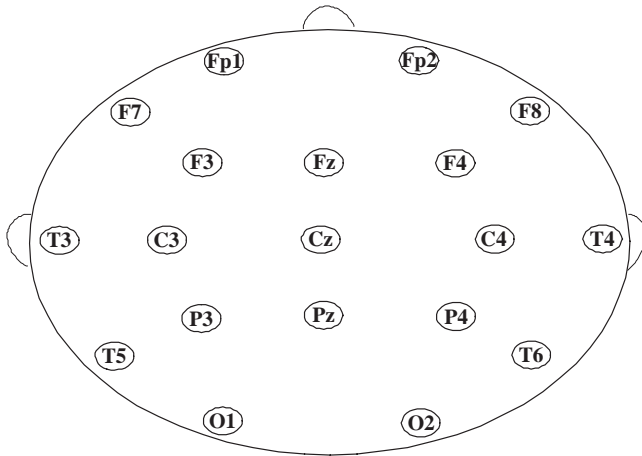


Fig. 1. Standard 10-20 system and nomenclature for locations.

information. This process makes it possible to recreate the waveform at a later time for computer display and statistical analysis. With this new capability for storage and quantitative analysis, the EEG of an individual can be compared with a database of individuals without any known neurologically based disorder, which allows for the analysis of the background activity to reveal patterns not apparent in the visual inspection of the routine EEG. For a review of the literature in this area, see the article by Chabot et al elsewhere in this issue.

The waveforms generated by the 3-mm cortical gray matter just below the scalp are measured based on the number of times per second that the waveform goes from one peak to the next (cycles per second or Hz). The entire range of EEG frequencies is conventionally divided into four standard frequency bands and designated as follows: delta (0–4 Hz), theta (4–8 Hz), alpha (8–13 Hz), beta (13 or more Hz), and gamma (40 Hz). Not all investigators use the same frequency definitions, however, which leads to difficulties in interpreting across studies.

The locations of the 19 electrodes follow the standardized 10-20 system. Fig. 1 shows the standardized locations of the electrodes.

There are two general classes of quantitative EEG (QEEG) measures. The first class examines the type of activity at each of the 19 locations in reference to a specific frequency. The value is usually correlated for a period of time or epoch that can vary according to how the evaluator collects the data. Examples of such quantitative measurements include the following:

- Magnitude: the average strength in absolute microvolts of the signal of a band during an epoch
- Relative power: the microvolts of the particular band divided by the total microvolts generated by all bands at a location

- Peak amplitude: the peak value in microvolts of a frequency band during an epoch
- Peak frequency: the highest frequency obtained during an epoch within a frequency range
- Symmetry: the peak amplitude symmetry between two locations (A and B) in a particular bandwidth (ie, defined as $(A - B)/(A + B)$). This measure analyzes the amplitude relationships that do not necessarily depend on connection activity but reflect differences in activity levels between different locations.
- Spectral power: the square of the microvolts of a frequency during an epoch

The second major class of variables addresses the issue of the connectivity patterns between locations. These variables are assumed to reflect the activity that occurs in the long myelinated fibers that connect the different regions and are known as the white matter of the brain. The variables are (1) coherence, which is the average amplitude similarity between the waveforms of a particular band in two locations over an epoch, and (2) phase, which is the time lag between two locations of a particular band as defined by how soon after the beginning of an epoch a particular waveform at location #1 is matched in amplitude at location #2.

Relation between quantitative electroencephalographic variables and cognition in reading disabilities and traumatic brain injury

Much of the original work on the relationship between the QEEG signal and cognition collected EEG data under eyes-closed conditions and then correlated those values with well-known cognitive measures, such as the IQ test. Different investigators reported the results with terms such as level or activity. These references can refer to magnitudes and relative power. These different measures are empirically highly intercorrelated.

Thatcher et al [35] sought to discriminate between normal subjects and subjects with TBI under the eyes-closed condition and obtained discriminate values at or above 0.90 across three independent samples. The predominant finding was decreased posterior alpha and increased posterior beta activity, frontal connection abnormalities, and some long cortico-cortico connection deviations in the group with TBI compared with the controls.

Additional studies generally have obtained consistent findings. Randolph and Miller [36] found variability of the EEG to be a critical component in discriminating patients with head injury from normals. Tabano et al [37] found higher mean power values in the lower alpha range (8–10 Hz), less power in fast alpha range (10.5–13.5 Hz), and lower mean alpha frequency in subjects with TBI compared with normal controls. They also reported a reduction in fast beta (20.5–26 Hz) activity. Trudeau et al [38] demonstrated high discriminant accuracy of qEEG for the evaluation of combat veterans with a history of blast injury.

Summarizing this body of research, Thatcher [26] concluded that “EEG coherence has been shown to be the most sensitive EEG measure of TBI.” He also concluded that “the standard or routine EEG and conventional MRI are essentially useless for the detection of TBI because of their low sensitivity and low reliability in detecting mild to moderate TBI (eg, <20% accuracy in routine visual EEG and visual MRI).”

These studies have focused on frequency ranges below the 32-Hz range and have not investigated EEG activity under task conditions. Collectively, the studies have indicated elevated beta levels after the trauma and decreased alpha in posterior locations, connection abnormalities, decreased alpha and beta amplitudes in frontal location, increased variance, and nonspecific generalized slowing. Some of the studies seem to have conflicting results (ie, increased posterior beta, reduction in fast beta) possibly because of definitions of the frequency ranges studied or differences in length of time since injury. Hughes and John [39] concluded that “there is a broad consensus that increased focal or diffuse theta, decreased alpha, decreased coherence and increased asymmetry are common EEG indicators of the post concussion syndrome.”

High-frequency electroencephalographic activity in the patient with traumatic brain injury and learning disability

The 40-Hz rhythm (gamma band) in animals has been found to be associated with the acquisition of learning. Basar-Eroglu et al [40] indicated that the 40-Hz rhythm exists spontaneously and can be evoked in the human brain, and they suggested that it may have multiple functions in sensory and cognitive processing. Forty-Hertz activity also has been found during problem solving in children [41] and adults [42]. Miltner et al [43] found increases in gamma band activity and gamma coherence between areas of the brain that undergo an associative learning procedure. Although more research is needed to clarify the role of 40-Hz activity in brain function, these early findings suggest the possibility that this frequency may be an important missing element in the understanding of patients with TBI and learning disabilities.

Activation conditions and the patient with traumatic brain injury

McEvoy et al [44] demonstrated that the test-retest reliability of the qEEG signal is greatly enhanced under task or activation conditions, because it requires the subject to focus on specific tasks, whereas the subject’s state during the eyes-closed condition may be expected to differ (because of vigilance, anxiety, cognitive processing variations). Seven-day test-retest reliabilities were higher for the activation condition (mean of 0.93) versus the eyes-closed condition (mean of 0.84). Even within a single EEG acquisition session, reliability varied more

during the resting condition (0.74–0.97) than the activation condition (0.92–0.99) when analyzing particular frequency bands (eg, theta, alpha).

In two studies, Thornton [45,46] compared subjects with TBI ($n = 32$) and normal controls ($n = 52$) under eyes-closed resting and activation conditions. The activation conditions were an auditory attention task, a visual attention task, and a listening-to-paragraphs task. In addition to measuring the traditional brain frequencies (1–32 Hz), this study measured higher frequencies in the 32- to 64-Hz range. An analysis of the EEG data collected in the eyes-closed condition led to correct classification of 100% of subjects as belonging to the TBI or normal control group (for accidents that occurred within 1 year of evaluation) and 93% (for all subjects regardless of time since accident). Separate analysis based on each of the activation measures yielded respective percent correct hit rates of 95% (auditory attention task: 79 of 84 subjects completing the tasks), 91% (visual attention task: 79 of 84 subjects completing the task), and 88% (listening to paragraphs: $n = 84$).

The listening-to-paragraphs task analysis required the least number of variables to discriminate. The variables that were involved most often in successful discrimination were high frequency (32–64 Hz) connectivity variables that emanated from the frontal lobes, which supported Thatcher’s emphasis on the effect on the frontal lobes in TBI cases. A separate analysis indicated that the length of time that had elapsed since the accident did not correlate positively with these connectivity values, which indicated that time does not result in improvement in these values.

The coherence and phase relationships between locations can be conceptualized in terms of a generator emanating from a particular location. This generator can be visualized as a “flashlight” effect, in which the origin of the beam comes from one location and sends the beam to all other 18 locations in a particular frequency. Fig. 2 expresses this relationship.

A correlational analysis was conducted to determine the EEG parameters that correlate with successful auditory recall for patients with TBI and normal controls [47]. The TBI group had significantly lower values than the control group for the beta 2 frequency (32–64 Hz) coherence and phase values involving frontal lobe

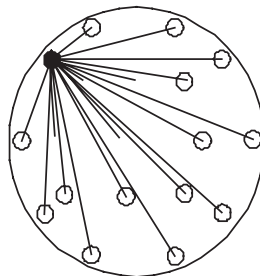


Fig. 2. The “flashlight” effect. One location sends out a signal in a particular frequency range to all other locations.

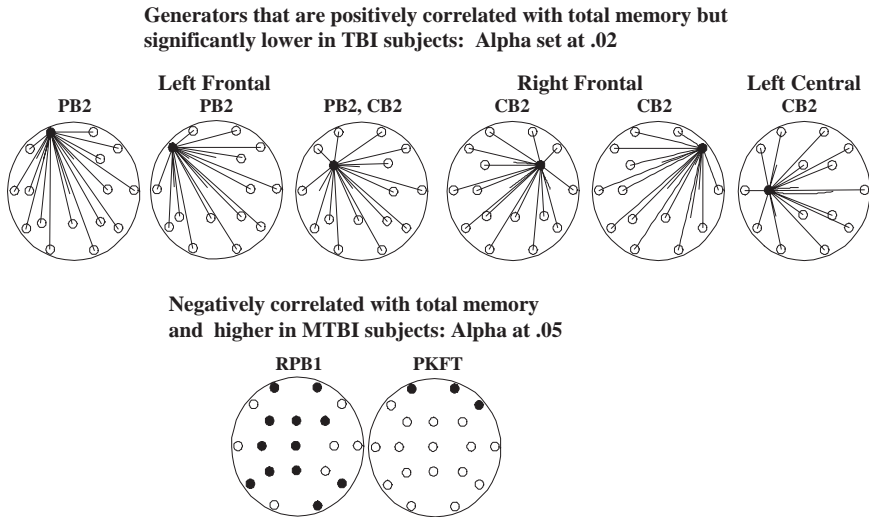


Fig. 3. Normal Vs traumatic brain injury: listening-to-paragraphs TBI ($n = 80$; normal = 49). PB2, phase beta 2; CB2, coherence beta 2; RPB1, relative power beta 1; PKFT, peak frequency theta; beta 1, 13–32 Hz; beta 2, 32–64 Hz.

locations. These values were significantly negatively related to the total memory score (Fig. 3). This pattern was not observed when analyzing the reading task and reading memory scores (K.E. Thornton, PhD, unpublished data).

Different QEEG variables are associated with success on the memory task in the two groups. In a normal adult group, auditory memory performance correlates positively with coherence alpha “flashlight” projections from predominantly left hemisphere locations (eg, T3, F7) (K.E. Thornton, PhD, unpublished data) [48]. As the value of coherence alpha increases, there are increases in the memory score. Within the TBI group, the positive correlates of successful recall include “flashlight” effects involving higher phase values from the right temporal location (T4 in the beta 1 frequency range) and left frontal location (F7 in the beta 2 frequency range) [46]. It seems that patients with TBI compensate by shifting the response pattern from the left temporal to the right temporal location and engaging the higher frequencies to complete the task successfully.

Critical review of quantitative electroencephalographic studies of traumatic brain injury

Several difficulties limit the degree to which firm conclusions can be drawn from the literature in this area. (1) Variations exist among studies in the use of specific frequency ranges and locations. (2) The eyes-closed condition does not directly investigate brain function during specific tasks. (3) Most studies do not

include the frequency range above 32 Hz. (4) The implicit concept behind many of these studies is that a particular set of locations is sufficient to understand how the brain functions. This view is akin to a previous popular concept of a modular functional model of brain activity. Lloyd's [49] review of 36 functional neuroimaging studies suggests that functions are distributed over multiple regions and most brain regions are multifunctional. (5) The age groups under consideration also differ across studies.

To address these limitations, future research should (1) use standard band definitions across different studies and tasks, (2) study the relationship between task performance and the qEEG variable during the task, (3) use higher frequencies (above 32 Hz), (4) study all locations, all available variables, and under different tasks, and (5) use separate databases for adult and children for the activation approach.

What is electroencephalographic biofeedback?

Neurotherapy (or EEG biofeedback) is the operant conditioning of the EEG. Electrodes are placed on the scalp of a subject, and the electrical information is sent to a recording unit. The unit uses a software interface to present the status of selected EEG variables to the subject in visual or auditory modality. When the subject's EEG signal meets the desired goal, the subject is presented with a reward in the form of selected sounds and displays. When the subject's EEG signal produces a value that is not desired, a different sound or visual image is presented to the subject to inhibit that particular signal. Because the brain is an adaptive organ, it attempts to satisfy the demands made on it by the software and changes its activity to meet these requests. The exact mechanism is unknown.

Treatment effects of neurotherapy with reading disability

No outcome research published to date has addressed the efficacy of neurofeedback specifically for reading disability. Several studies of the effect of neurofeedback on attention deficit hyperactivity disorder (ADHD), however, have provided suggestive preliminary evidence that this intervention modality can result in improved cognitive function in general.

A case study of a 13-year-old child with ADHD demonstrates the effectiveness of 45 EEG biofeedback sessions [50]. The cortical sites that were monitored were C3 (designed to increase 15–18 Hz and decrease 2–10 Hz) and C4 (designed to increase 12–15 Hz and decrease 2–7 Hz). There was marked improvement (tested at preintervention and at the twentieth and fortieth sessions) in processing speed and processing speed variability, a 19-point IQ increase (Kaufman Brief Intelligence Test), a 7.5 grade level increase in reading

scores (Kaufman test of Educational Achievement–Brief Form), and significant behavioral improvements, as indicated by report of parents and patient. Follow-up at 17 months demonstrated that the behavioral and QEEG changes were maintained.

With samples of learning disabled subjects and subjects with and ADD and ADHD (total sample size $n = 155$), four independent researchers have demonstrated significant increases in IQ averaging 15 points (one SD) as a result of EEG biofeedback [51–54]. Only one study [51] used a control group, which did not demonstrate improvements on the IQ measures. A deficit in 40-Hz activity has been reported in children with learning difficulties [55,56] and can be enhanced through EEG biofeedback [57,58].

Efficacy of electroencephalographic biofeedback with traumatic brain injury

Frequency interventions

In a single case study, Byers [59] found that 31 sessions of EEG biofeedback increased the magnitude of EEG in the 12- to 18-Hz range and suppressed EEG magnitude in the 4- to 7-Hz range. The patient who had mild TBI improved cognitive flexibility and executive function. Hoffman et al [60] used EEG biofeedback techniques on 14 patients with TBI and reported that approximately 60% of the patients with mild (M)TBI showed improvement in self-reported symptoms or cognitive performance as measured by the MicroCog assessment battery after 40 sessions. The degree of improvement noted ranged from 23% to 62%. The authors also noted significant normalization of the EEG in subjects who showed clinical improvement. There were no controls in this study. A subsequent open trial case series ($n = 14$) showed significant improvement after five to ten sessions in self-report symptom checklists [61,62].

Keller [63] demonstrated with a group of patients with TBI ($n = 12$) that ten sessions of EEG biofeedback (13–20 Hz, increase mean amplitudes) improved attentional abilities (in 8 patients) and was superior to ten 30-minute sessions using two standard software computerized attention training programs [64,65]. The EEG biofeedback subjects showed significant improvement on the cancellation task (improvement more than 3 SD) and nonsignificant improvements in other error measures (eg, choice reaction, sustained attention), whereas subjects in the computer-based training improvements showed no improvements on any measure. Significant improvements (more than 2 SD) for the EEG biofeedback group also were noted on number of errors and crossed out stimuli on the cancellation task, choice reaction time speed (milliseconds), and reaction time (milliseconds) on the sustained attention task. The computerized intervention program also showed significant improvements (more than 2 SD) on the number of crossed out stimuli in the cancellation tasks (≥ 2 SD) and choice reaction time (milliseconds) (≤ 2 SD), however.

Coherence interventions

Walker et al [66] studied 26 patients with MTBI within 3 to 70 days of injury with an eyes-closed QEEG. EEG biofeedback treatment protocols (average of 19 sessions) that addressed the deviations from the normative database for the abnormal coherence values were then implemented. Five sessions were directed toward each coherence problem until the patient reported significant improvement or until 40 sessions were completed. No controls were used. Significant and substantial improvements (>50%) on a global improvement self-rating scale were reported by 88% of the patients. All patients were able to return to work.

Coherence and magnitude interventions

Tinius and Tinius [67] performed 20 EEG biofeedback sessions and cognitive retraining with a group of patients with TBI ($n = 16$) and ADHD ($n = 13$). Progress was assessed with neuropsychological measures of attention and problem solving and compared with a control group (negative history of neurologic or neuropsychological problems, not matched for age or education) that received only the cognitive retraining intervention. The QEEG studies were conducted for all subjects. Intervention parameters were determined by reference to the qEEG database comparison [26]; EEG biofeedback training targets included coherence and magnitude abnormalities. Both groups were treated with visual and auditory cognitive training exercises [68,69]. The subjects with MTBI and ADHD in the EEG biofeedback treatment groups improved significantly (+.5 to +1 SD) in comparison to the control group on the attention tasks (intermediate visual and auditory attention) [70]. The MTBI group showed significant improvement compared with controls on the Wisconsin card-sorting problem-solving task in terms of a decrease in the number of trials and perseverative errors.

Schoenberger et al [71] developed an alternative EEG biofeedback approach with patients with TBI that involved conventional EEG biofeedback and subthreshold photic stimulation. The clients wore glasses that had light-emitting diodes embedded in the lenses. The EEG sensors were moved to different locations on the head during the treatment. The client's momentary dominant or peak EEG frequency was measured and used to reset the frequency at which the light-emitting diodes pulse, which in turn affected the EEG. The goal of the intervention was to reduce slow-wave activity (4–8 Hz) and increase activity in the 12- to 18-Hz range. Ochs [72] previously reported positive effects in clinical cases (with a wait-list control group) using this approach with patients with TBI. The Schoenberger study examined 12 subjects who had experienced mild to moderately severe TBI and were 36 months to 21 years post trauma. Neuropsychological measures of memory, attention, information processing, verbal fluency, and integrated functions were administered, as was the Beck Depression Inventory and the Multidimensional Fatigue Inventory. The

researchers used a wait-list control group (who subsequently received the treatment) and random assignment to the treatment and control groups. The subjects received 25 sessions, with session length varying between 5 seconds and 15 minutes, over a 5- to 8-week period. The dominant frequency that was stimulated varied between 5 and 20 Hz. Significant improvements were reported on the emotional (Beck Depression Inventory, Multidimensional Fatigue Inventory) and the neuropsychological measures, and 7 subjects reported returning to a productive work life. Additional benefits included a reduction of medication usage for 2 of the 8 subjects taking medications, with cessation in 3 subjects. Potential problems of practice effects were addressed with alternate measures when available. Three subjects did not respond positively to the treatment.

In summary, qEEG biofeedback interventions have proved to be a useful approach to remediation of cognitive difficulties in patients with TBI, whether the approach was directed toward coherence or magnitude measures. Limitations of these studies include a lack of specificity between the cognitive task and its relationship to the qEEG variables, failure to obtain or indicate that the cognitive improvements were concomitant with changes in the qEEG measures, lack of high frequency analysis, and long-term follow-up.

An alternate electroencephalographic biofeedback approach: development and clinical application of an activation database

Whereas the eyes-closed condition provides clinically relevant information regarding the nature of state of the brain, it does not provide information on the brain's active functioning. A logical next step in the development of this field is the use of a qEEG activation database in the rehabilitation process. Thornton developed such a database with normal child and adult subjects (K.E. Thornton, PhD, US patent #6309361 B1) [45,46,73,74]. The criteria for inclusion in the database were no self-report of neurologic or psychiatric problems or history of learning disabilities/ADD or seizure activity. The database includes 30 child subjects between the ages of 10 and 14 and 60 adult subjects over the age of 14. The age cut-off for the adult group was derived from Piaget's concept of formal operations beginning at approximately age 13.

The EEG was recorded in a resting or baseline condition with eyes closed and eyes open and during 24 different cognitive activation tasks. These tasks focused on auditory and visual attention, auditory memory (eg, paragraphs, word lists), visual-verbal memory (eg, names of faces, reading) and visual information. All memory tasks involved collection of data during the input stage and during immediate and delayed recall periods. Additional cognitive tasks included problem solving (Raven's Matrices), pronunciation of nonsense words, spelling, mathematics (internal spatial addition and multiplication tables), autobiographical memory, and visualization (K.E. Thornton, PhD, US patent #6309361 B1).

Treatment protocols and intervention methods using the activation database

The treatment consists of subjects either listening to audiotapes or reading while the appropriate protocols are being used. The purpose of this approach is to train the brain under the appropriate and relevant task conditions. The initial evaluation provides four baseline measures of auditory memory. During treatment, the subject's progress is tested with novel stories that contain approximately 20 to 25 pieces of information. The subject listens to the story at the beginning of the session and recalls the story immediately to the clinician to obtain an immediate memory score. At the end of the session, the subject is asked to recall the story to obtain a delayed memory score. The scores are compared with the baseline to assess improvement in functioning. Treatment for children with learning disability or ADHD typically involves 40 sessions, although the program can last longer for patients with TBI.

Clinical case examples

Learning disabled case reports

Case examples previously have been reported in peer-reviewed journals [73,74]. This report provides additional information and includes additional subjects. A control group used in the previously reported research did not demonstrate any significant gains as a result of practice effects or the passage of time between first and second testing. Outcomes are reported for all variables that were available for analysis.

Case 1 involves an 8-year-old boy who was diagnosed with ADHD (no official diagnosis of reading disability) and underwent 25 hours (50 sessions) of qEEG biofeedback. The focus of the treatments, based on findings from his qEEG activation study, was on decreasing relative power of delta and theta and increasing relative power of beta1 (13–32 Hz) under auditory memory conditions in central and posterior locations. The data (Table 1) reflect the changes in the qEEG variables during a reading task in the left posterior region (T5-P3-O1) after the treatment and the resultant improvements (gain of percentile rank of 50%) on the reading subtests of the Terra Nova test (Table 2).

Table 1
Child with attention deficit disorder with excessive theta: case 1

Relative power	Initial evaluation	Session #50 value	Change in standard deviation units
%Beta1	23.1	26.8	1.23
%Theta	15.5	13	-0.86
%Delta	26.2	17.2	-1.91

Change in standard deviation units uses standard deviation of normative database under reading conditions.

Table 2
Changes in % rank on Terra Nova: case 1

Scale	Terra	Nova	% change
	March 2002	March 2003	
Reading	27	75	48
Vocabulary	21	72	51
Reading composite	25	75	50
Language	56	58	2
Math	77	65	-12
Math computation	48	72	24
Math composite	67	69	2
Total score	55	67	12
Science	39	54	15
Social studies	35	77	42
Word analysis	45	75	30

Values represent subject's percentile rank.

The improvement in the values during the auditory input condition generalized to the reading condition. His auditory memory improved 205%. The SD value represents the standard deviation in the normative reference group of the relative power figures during a reading task.

Case 2 involves a 9-year-old girl whose parents reported a history of learning problems. Neither academic records nor formal educational or neuropsychological testing completed were examined to verify the presence of learning disability, however. Her absolute levels for theta were more than -0.50 SD below the norm, and her values for relative power of beta 2 (frequency range 32–64 Hz) were 2 to 3 SD above the norm, which indicated that she did not fit into the high theta/low beta pattern seen in many children with learning disability. The coherence alpha projections during the input stage and the coherence and phase alpha during the immediate recall period were significantly below the norm, however, and became the main focus of the treatment. She improved approximately 1069% in auditory memory (total memory score from 1.8–19.25) and 400% in reading memory (total score from 2.5–14) during the 40 sessions. Table 3 presents her improvements toward the end of the treatment.

Two subjects have been treated at different clinics using the activation approach, which reflects generalizability of the approach.

Case 3 represents another example of a child with significant history of reading problems who did not demonstrate any problems in theta, delta, or beta

Table 3
Improvement in auditory and reading memory scores after the treatment: case 2

	Immediate pre/post	Delayed pre/post
Auditory memory score	1.8 10	0 9.25
Reading score	3.5 7	0 7

Scores represent pieces of information recalled.

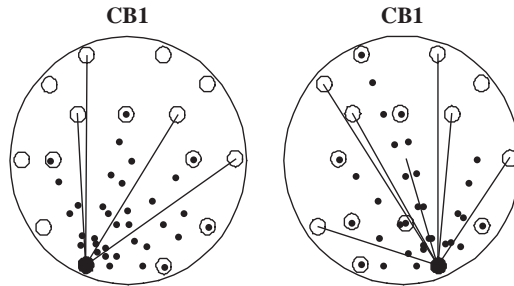


Fig. 4. Case 3: reading disability. CB1, coherence beta 1 (13–32 Hertz). Lines represent values that are between -1 SD and -2 SD below norm. Dotted lines represent values that are between -2 SD and -5 SD below norm.

values but showed significant problems in connectivity issues. The parents had spent approximately US\$25,000 in alternate standard treatment programs to improve his reading ability, which resulted in no significant gains. Fig. 4 presents some of the coherence abnormalities found in the beta1 frequency.

Many additional deviations from database averages also were observed with this child. The treatment was directed toward the posterior connection problem from the occipital positions under reading and auditory memory conditions. His auditory memory functioning increased 589% from baseline by the end of the twenty-fifth session. He also improved on the standardized reading inventory (SRI) from the previous year’s testing (a standardized reading inventory measure administered by the school system) from a Lexile score of 360 before treatment to 753 after approximately 40 sessions, which is a much larger change than the typical improvement of 75 to 100 Lexiles per year. Table 4 shows his pre- to posttraining changes, expressed as Z scores, on the QEEG variables addressed during the interventions.

Table 4
Z score changes of case 3 as a result of treatments

Measure	Preintervention	Current activity	Z score change
O1-O2 Coherence:beta 1	-3.3	-1.6	+1.7
O1-O2 Phase:beta 1	-1.3	-0.6	+0.7
O1-O2 Coherence:beta 2	-4.3	-2.0	+2.3
O1-O2 Phase:beta 2	-4.1	-2.3	+1.8
O2-T5 Coherence:beta 1	-2.0	+0.4	+2.4
O2-T5 Phase:beta 1	-3.0	-0.1	+2.9
O2-T5 Coherence:beta 2	-1.6	+0.5	+2.1
O2-T5 Phase:beta 2	-3.0	+0.2	+3.2
O2-Pz Coherence:beta 1	-3.0	+0.3	3.3
O2-Pz Phase:beta 1	-1.6	-1.0	+0.6
O2-Pz Coherence:beta 2	-2.5	+1.0	+3.5
O2-Pz Phase:beta 2	-2.0	-2.0	0.0

Beta 1: 12–32 Hz; Beta 2: 32–64 Hz.

His mother reported her impression of increased self-confidence, greater reading fluency, and ability to present information orally in school. There were no grades available for comparison from the resource room to which he was assigned.

Case 4 involved a 17-year-old subject with reading disability. After 20 sessions he increased his comprehension score (on the Burns Roe Reading Passages) from 45% to 90% (on alternate versions –eighth grade level) and from 20% to 70% (on tenth grade level). His performance on alternate versions of the Cognisys Story Recall Test had increased by 3 SD. On the Wechsler Individual Achievement Test reading comprehension subtest he attained a standard score of 99 for age and grade level.

Neurotherapy for the patient with traumatic brain injury

Case 5 involves that of a 37-year-old woman (with a PhD) who experienced a mild TBI during an auto accident. She was particularly concerned that she recover her auditory memory ability to return to work as a psychotherapist. Results are presented in Fig. 5. The predominant problems were with coherence beta 2 (32–64 Hertz) that emanated from the Fz location. The figure presents the deficit connection pattern as if there was a flashlight emanating from that location

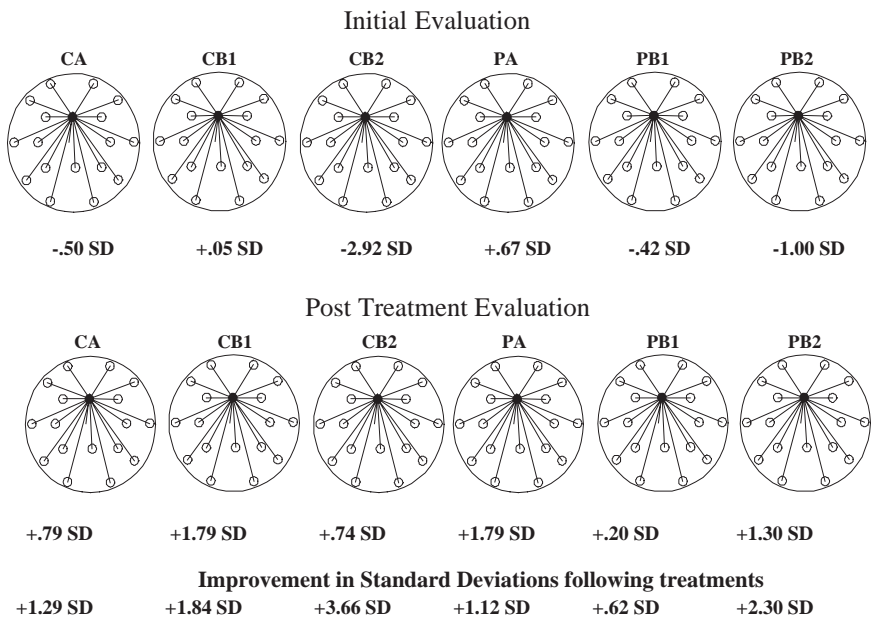


Fig. 5. Example of patient with TBI (Case 5). The qEEG coherence and phase deficits. CA, coherence alpha; CB1, coherence beta 1; CB2, coherence beta 2; PA, phase alpha; PB1, phase beta 1; PB2, phase beta 2; treatment interventions directed only toward the Fz CB2 deficit. SD, standard deviation.

Table 5
Case 5

	Pre	Post (13 mo>pre)
Continuous performance		
Errors	10	2
Logical memory:		
immediate	26%	82%
delay	44%	86%
Shipley IQ	101	123
California verbal learning test		
Total raw (all five trials)	47	61
Long delayed free recall	10 (–2 SD)	15 (+1 SD)

and projecting its beam to the rest of the head. Interventions were directed toward increasing coherence beta 2. The subject was involved in neurofeedback on a weekly basis for more than a year. The training was targeted at normalizing these beta 2 coherence abnormalities. Significant improvements (3.7 SD increase) in the EEG were seen in the areas targeted and in other connectivity variables.

Improvements in cognitive functioning at 13 months after initial testing were as follows (Table 5): Shipley verbal IQ score improved from 101 to 123 (approximately 7 SD improvements in continuous performance test errors); Wechsler Logical Memory improvements (35% to 84% overall ranking) for immediate and delayed recall; raw score increase from 40.5 to 62.5, or a 54% improvement. On the California Verbal Learning Test the patient improved 3 SD on long delay free recall and recognition hits and 2 SD on several measures (short delay, free and cued; long delay cued recall). She also increased total memory score from 47 to 61, while the rest of the measures changed in a positive direction (except for perseverations, which increased significantly—5 SD). The Wisconsin Card sorting performance showed an increased correct score (from 61–71), whereas the other measures remained approximately the same and within the average range. Changes in a negative direction included an increase in errors on the category test (69–81) and an increase in the number of trials to complete category 1 (11–18) on the Wisconsin Card sorting test.

Case 6 (Fig. 6) involved a female patient with mild TBI with deficits in the high frequency range that emanated from the F4 location. She entered neurofeedback 3 years after the accident. Interventions were directed toward the F4-Fz, F4-C3, and F4-Fp2 relationships. The subject's neurofeedback treatment notes showed 2 SD in improvements in the beta 2 coherence at F4-Fz. Her auditory memory score improved 110%. Although the interventions were not directed at the normal positive correlates of memory (eg, T3 coherence alpha values) there were substantial improvements in this skill.

Case 7 involved a 69-year-old woman who was hit by a car at a shopping mall and remained unconscious for 3 months. An MRI evaluation revealed a left frontal hematoma. Her QEEG study revealed abnormalities in left frontal connectivity (particularly FP1-F3 PB2, F3-T3 PA). Neurofeedback began 24 months after the accident. The treatment protocols were directed toward

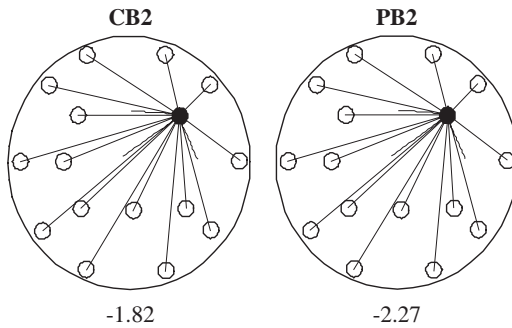


Fig. 6. TBI (Case 6). Values on bottom represent standard deviation difference from norm.

these connection problems. After 54 sessions, the values of Fp1-F3 PB2 (phase beta 2) increased from 36 to 70 (4.9 SD) and for F3-T3 PA (phase alpha) from 62 to 68 (1.25 SD). Her auditory memory improved from 10 to 34 (340%) pieces of information. The case is of particular importance because it involved structural damage to the brain in an elderly patient, factors that would intuitively be thought to be negative treatment indicators.

Comparisons of effectiveness of interventions

Comparisons of the outcomes of neurotherapy with traditional interventions demonstrate their relative effectiveness in treating reading disability and TBI. Fig. 7 shows the outcomes of treatments in standard deviation units for several programs in current use and for the different forms of EEG biofeedback: standard EEG biofeedback (increased beta/decrease theta at central locations) and activation qEEG-guided biofeedback. For reading disability, the current programs show improvements that range from 0 to +0.40 SD on verbal skill measures, +0.60 to +1 SD for “standard” EEG biofeedback on attention and IQ measures, compared with the +3 to +3.3 SD changes for the hi-frequency activation database-guided qEEG biofeedback (reading and auditory memory). For the TBI cases, attention is improved +0.45 SD by cognitive exercises and +1.63 SD by “standard” EEG biofeedback. Problem solving is not improved by cognitive exercises but is improved by +0.60 to +0.71 SD with “standard” and hi-frequency activation database-guided QEEG biofeedback. Finally, memory for paragraphs is improved by cognitive exercises +0.57 SD (short-term assessment), whereas hi-frequency activation database-guided QEEG biofeedback showed average increases of +3 SD (up to 1 year follow-up assessment in one case). These results offer encouragement to the continued application of QEEG-guided interventions for cognitive improvement in these groups.

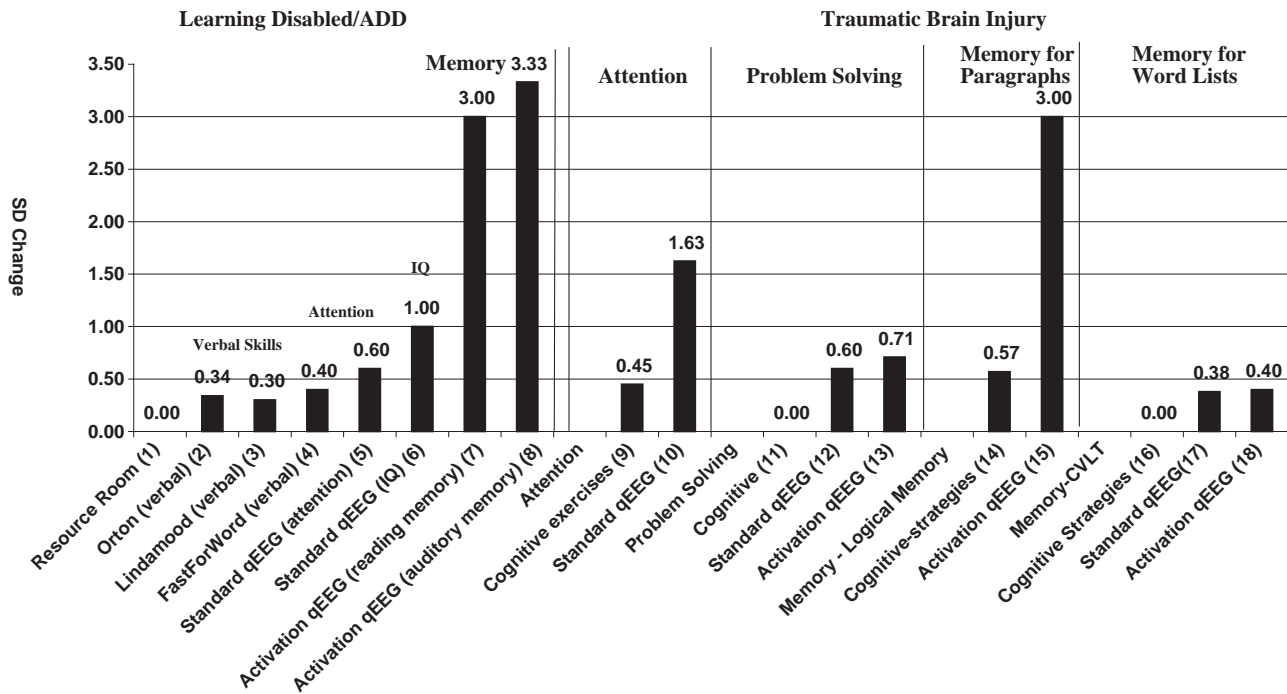


Fig. 7. SD changes in learning disability and subjects with TBI across different intervention modes and different cognitive abilities. The following numbers, which follow the treatment type listed in the figure, draw their values from the reference number in brackets. 1 [75], 2 [21], 3 [22], 4 [23], 5 [50,67,76], 6 [51-54], 8 [73,74], 9 [77], 10 [63,67], 11 [67,77], 12 [67], 14 [77], 15 [73,74], 16 [77], 17 [71].

Summary

Our society has spent billions of dollars on efforts to remediate the cognitive and behavioral dysfunction in individuals with learning disabilities and TBI through various cognitive-based strategies. The evidence accumulated to date indicates that few of these intervention efforts demonstrate efficacy. When change is measured for the more traditional approaches, the change scores typically result in improvements in the +0.00 SD to +0.50 SD range, often after lengthy intervention periods. Research completed to date and clinical reports show greater improvements with EEG biofeedback with these two groups.

The application of neurofeedback with reading disability and TBI is relatively recent. Although no published studies have assessed the efficacy of neurofeedback for subjects specifically diagnosed with reading disability, many studies have assessed the effectiveness of qEEG with the ADHD population, which is known to have a high rate of comorbidity for learning disabilities. These findings suggest the possibility that neurofeedback specifically aimed at remediating reading disability would be effective. Clinical experience, as evidenced by the case examples, provides strong initial support for this suggestion. In particular, there is reason to believe that assessment and training under task conditions are likely to be fruitful. Further research is required to confirm these initial findings. Given the significance of the problems and the absence of proven alternatives for remediating reading disability, efforts to complete the needed research seem warranted. Given the absence of proven alternatives, clinical use of this intervention also seems to be warranted with informed consent acknowledging the absence of empirical efficacy data.

More work has been reported on the assessment of the efficacy of neurofeedback for TBI. The results of these studies indicate that neurofeedback shows promise in this area. There is reason to believe that assessment and training under task conditions are likely to be fruitful. Further research replicating these findings with larger numbers of subjects and better controls is needed before strong claims can be made. Clinical work using neurofeedback with patients with TBI has been consistent with the indications of efficacy found in the research. Given the significance of the problems and the absence of proven alternatives for remediating the cognitive and behavioral effects of TBI, efforts to complete the needed research for clinical use seem warranted.

Acknowledgments

The authors would like to express their appreciation to Alena Appelbaum, Dale Paterson, and Roger Riss, PhD, for their continued support in the development and clinical application of the QEEG activation approach and their editorial and subject contributions for this article.

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