Traumatic Brain Injury Rehabilitation: Efficacy Review of Computers, Strategies, QEEG-Guided Biofeedback, and Medications

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Abstract

The effective remediation of the traumatic brain injured patient is problematic. Cognitive rehabilitation programs are often not offered to patients due to poor clinical results. This paper reviews the empirical reports of changes in cognitive functioning after treatment and compares the relative effectiveness of several treatments including computer interventions, cognitive strategies, EEG biofeedback, and medications. The cognitive functions that are reviewed include auditory memory, attention and problem solving. The significance of the change in cognitive function is assessed in three ways that include effect size, multiple measures of effectiveness and longevity of effect. These analyses complement the previously published meta-reviews by adding these three criteria and include reports of an intervention method called EEG biofeedback.
Key words: EEG biofeedback, traumatic brain injury, cognitive rehabilitation, neurocognitive rehabilitation, QEEG, activation QEEG, memory rehabilitation

Traumatic brain injury (TBI) is associated with impairments in cognitive functioning. Rehabilitation is designed to restore cognitive functions such as memory, attention, and problem-solving. Many research studies report statistically significant effects for treatments, with the recommendations that the treatments are effective and beneficial. However, many research findings are not more effective than placebo, and many of the improvements in test scores from pre-treatment to post-treatment are no different than the improvements in scores due to repeated administrations of the test as shown by control groups. In this paper, we review the neuropsychological evaluation of TBI including brain electrophysiology. Interventions designed to restore cognitive functions are reviewed and their effectiveness is assessed. The assessment includes an analysis of the effect size of the intervention, which is a method that quantifies the effectiveness of a particular intervention relative to some comparison and answers the question of how well does the intervention work. Clinical recommendations (3 levels) for treatment are provided based on the effect size analysis, which employs a rating scale of 0 to 4. The paper concludes with protocols for treatment of TBI using quantitative electroencephalography.

The Neuropsychological Evaluation of Traumatic Brain Injury

The NIH (1998) consensus statement indicated that: "rehabilitation of persons with traumatic brain injury (TBI) should include cognitive and behavioral assessment and intervention" (p. 23). Neuropsychological assess-

ment has long provided these cognitive diagnostic tests for the TBI patient, and the cognitive measures that are typically evaluated in the case of TBI include memory, attention, and problem-solving. The relationships between neuropsychological measures and outcome measures have attracted considerable attention over the years. Outcome measures of interventions include neuropsychological revaluations, employment status, self reports, and reports by significant others. However, several of these basic measures do not indicate if cognitive abilities are restored. For example, employment status does not directly measure ability, as the person may be employed on the basis of a variety of factors unrelated to cognitive function, such as the workplace tolerance of the employee with TBI, and working in a less skilled position. In addition, self-reports and reports of others are fraught with issues of subjectivity. The advantages of neuropsychological measures reside in the objection quantification of the changes in specific cognitive abilities.

A review of the literature on the relationship between neuropsychological measures and outcomes concludes that, "many neuropsychological tests have a moderate level of ecological validity when predicting everyday cognitive functioning" (Chaytor & Schmitter-Edgecombe, 2003, p. 181). Specifically, high scores on tests predicted full-time employment 62% of the time while low scores predicted unemployment 67% of the time (Fabiano & Crewe, 1995). While neuropsychological testing does predict return to work, the relationship is moderate and other non-cognitive factors are relevant. Although problematic in many respects, neuropsychological measures remain our best measure of rehabilitation success.

The current standard practice for the diagnosis of TBI is to conduct the clinical interview, assess the specifics of the injury, and assess standardized test performance. However, issues with respect to malingering, pre-existing status, appropriate norms, cultural background and, more recently, effort (Gavett, O'Bryant, Fisher, & McCaffrey, 2005) have rendered the diagnostic accuracy of these tests problematic in many cases. It is then important to have a measure of physiologic functioning which can be correlated with the cognitive problems.

The Quantitative EEG as a Supplemental Physical Diagnostic Tool for TBI

Modern medical diagnostic techniques such as magnetic resonance imaging (MRI), computed tomography (CT), positron emission tomography (PET), and diffusion tensor imaging (DTI) have been used to identify differences in brain states between groups of patients with TBI and normal controls (Broughton & Hasan, 1995). However, due to low sensitivity in individual and group cases these tests are generally not used to identify an individual with TBI (Thatcher, 2000). In contrast, there has been an increase in the use of quantitative electroencephalography (QEEG) in TBI evaluations to supplement neuropsychological testing. Traditional analog electroencephalography (EEG) employs an immediate paper printout of the waveforms, while the QEEG digitizes the signal and saves mathematical information regarding the waveform to a hard disk, thus enabling mathematical analysis rather than employing human judgment and classification. The QEEG analysis generates two types of variables. The first type of variable measures the strength of the brainwaves in terms of microvolt, peak amplitude, spectral power, peak frequency, and relative power at specific scalp locations in frequency ranges (delta, theta, alpha, and beta). The second type of variable addresses the relationship between pairs of locations in terms of coherence and phase, which assess the coordination of brain activity across separate brain regions within different frequencies.

Thatcher and others (Thatcher, Walker, Gerson, & Geisler, 1989) provided the initial research demonstrating the replicability of a discriminant function analysis that distinguished TBI patients and normals in three independent samples. The QEEG showed a sensitivity of 95.4% of TBI cases and a specificity of 97.4% (Thatcher, Walker, Gerson, & Geisler, 1989). While Nuwer (Nuwer, 1997), representing the American Academy of Neurology (AAN), argued that the: "QEEG remains investigational for clinical use in post-concussion syndrome, mild or moderate head injury" (p. 9), rebuttals of the AAN position paper have been published (Hoffman et al., 1999; Hughes & John, 1999; Thatcher et al., 1999). Furthermore, the QEEG has been identified as an appropriate diagnostic tool for TBI by the Electrodiagnostic and Clinical Neuroscience Society (Hughes & John, 1999) and by the Veteran's Administration (Salazar, Zitnay, Warden, & Schwab, 2000).

However, the use of QEEG data in the rehabilitation of cognitive functions is not necessarily concerned with diagnostic issues. The International Society for Neuronal Regulation has stated (Hammond et. al, 2004) that: "Unlike neurology and psychiatry, where QEEG is principally used for purposes of diagnosing medical pathology, neurotherapists who use QEEG primarily do so to guide EEG biofeedback training" (p. 6). One of the pur-

poses of this paper is to assess the efficacy of the QEEG in the rehabilitation of brain function.

Relationship between Neuropsychological Measures and Outcome

The relationships between neuropsychological measures and outcome measures have attracted considerable attention over the years. Outcome measures of interventions include neuropsychological revaluations, employment status, self reports, and reports by significant others. However, several of these basic measures do not indicate if cognitive abilities are restored. For example, employment status does not directly measure ability, as the person may be employed on the basis of a variety of factors unrelated to cognitive function, such as the workplace tolerance of the employee with TBI, and working in a less skilled position. In addition, self-reports and reports of others are fraught with issues of subjectivity. The advantages of neuropsychological measures reside in the objective quantification of the changes in specific cognitive abilities.

Treatment

The National Academy of Neuropsychology (NAN, 2002) adopted the American Congress of Rehabilitation Medicine's definition of cognitive rehabilitation as:

"… a systematic, functionally oriented service of therapeutic cognitive activities, based on an assessment and understanding of the person's brain-behavior deficits. Services are directed to achieve functional changes either by reinforcing, strengthening, or reestablishing previously learned patterns of behavior or by establishing new patterns of cognitive activity or compensatory mechanisms for impaired neurological systems." (Harley, et al., 1992, p. 63)

In 1990 there were over 700 programs for cognitive rehabilitation (Ashley, Krych, & Lehr, 1990). The majority of programs are grouped in this paper into two large classes of interventions. The first are those interventions that are introduced from "outside" the patient, which include two cognitive rehabilitation models (computers, strategies) to be discussed, while the second class are those that are based on interventions that work from "inside" the patient, which include medications and EEG biofeedback. We describe the programs, their assessment and their relative effectiveness.

The "Outside" approach - Three Cognitive Rehabilitation Models

"Outside" interventions have focused on the use of computer interventions and strategy instruction. An example is a vigilance task designed to improve attention in which the patient views a computer screen and taps the space bar on the keyboard whenever a large red circle is displayed (Gray & Robertson, 1992). Feedback to the patient is contingent upon their response speed, with increases in frequency of feedback following increases in response speed. Examples of the cognitive strategy interventions that focus on memory deficits are visualizing, creating associations, and structuring concepts.

There are three general "outside" approaches to cognitive rehabilitation. Restorative cognitive rehabilitation (RCR), which employs stimulation and practice, is based upon the concept that repetition can restore function. RCR is an attempt to reinforce, strengthen, or reestablish previously learned patterns of behavior (NAN, 2002). However, there is evidence that simple repetitive practice is of minimal or no aid in improving memory for recall (Glisky & Schacter, 1986; McKinlay, 1992). On a physiological level, reestablishing previously learned patterns of behavior should translate to reestablishing previous EEG and blood flow patterns. Thornton (2000) established that "time does not heal" (the brain does not spontaneously repair itself) but instead allocates different resources to accomplish the task with less efficient results (Thornton, 2002). This compensatory pattern of results was confirmed in a PET study showing that while both TBI patients and controls engaged frontal, temporal, and parietal regions known to be involved in memory retrieval, the TBI patients showed relative increases in frontal, anterior cingulate, and occipital activity (Levine et al., 2002). The hemispheric asymmetry that is a typically evident in controls was also attenuated in patients with TBI.

The second approach, strategy cognitive rehabilitation (SCR), focuses on developing conscious cognitive processes (strategies, mnemonic approaches) with the expectation that improvement will generalize to activities of daily living by establishing new patterns of cognitive activity (NAN, 2002). However, researchers in the field generally agree that these approaches face the problem that the subject does not continue to use the strategy after treatment terminates (Freeman, Mittenberg, Dicowden, & Bat-Ami, 1992).

The third approach, compensatory cognitive rehabilitation (CCR), provides external, prosthetic assistance

for dysfunctions (Wehman et al., 1989) and is considered to be a compensatory mechanism (NAN, 2002). This approach has received positive recommendations (Cappa et al., 2003; Cicerone et al., 2000). However, there is no evidence that indicates use of compensatory devices results in meaningful improvement in core cognitive skills (Ricker, 1998).

The "Inside" Approach - Medication and Quantitative Electroencephalography

Medication. Depression often accompanies TBI with over 50% comorbidity (Moldover, Goldberg, & Prout, 2004). Methylphenidate and other ADHD drugs (such as dexmethylphenidate HCl or Focalin®), have been recommended due to their effectiveness with attention deficit disorder (Plenger et al., 1996). Bromocriptine® (2 bromo-alpha-ergocryptin) has been recommended due to effects on working memory and executive functions, which are two of the affected cognitive abilities in TBI (McDowell, Whyte, & D'Esposito, 1998). Other medications that have been used historically include amantidine (Symmetrel®), dextroamphetamine (D-Amphetamine™), levodopa (Sinemet®, Atamet®, and Larodopa®), and modafinil (Napolitano, Elovic, & Qureshi, 2005).

EEG Biofeedback Interventions – An Alternate "Inside" Approach. EEG biofeedback interventions are the latest approaches to the rehabilitation problem. This method involves operant conditioning of brainwave patterns through the use of reinforcement. A goal of the feedback is to return the underlying electrophysiological functioning of the brain to a normative, preexisting level. The four current approaches in the implementation of EEG biofeedback in the TBI situation are a) the Flexyx Neurotherapy approach (now referred to as LENS: Low Energy Neurofeedback System), b) the standard quantitative EEG approach, c) the eye closed QEEG, and d) the activation database QEEG. The Flexyx Neurotherapy approach, which is a modified EEG biofeedback technique, combines conventional QEEG biofeedback and small radio frequency wave input that is based on the brain's dominant frequency in an effort to alter QEEG patterns associated with cognitive dysfunction (Schoenberger, Shif, Esty, Ochs, & Matheis, 2001).

Historically, the initial "standard" QEEG-guided (SQ) biofeedback focused on increasing the strength of beta activity (13-20 Hertz) and decreasing the strength of theta activity (4-8 Hertz) along the sensorimotor strip, which is located on the top central portion of head (scalp locations C3, CZ, C4) (Lubar & Lubar, 1984; Tansey, 1991; Othmer & Othmer, 1992). The next advance in the field was to compare the patient's resting, eyes closed QEEG to a reference database (EcQ) leading to more customized protocols for patients (Tinius & Tinius, 2000).

The most recent logical development of electroencephalography techniques is the use of an activation database QEEG-guided biofeedback (ActQ) approach that examines brain activity while patients engage in specific cognitive tasks (Thornton, 2001). This contrasts with the EcQ approach, which assesses brain activity while patients are resting with their eyes closed. In addition, the ActQ assesses brain activity over the frequency range of 0 to 64 Hertz, in contrast to the 0 to 32 Hz range of the EcQ. The addition of the high frequency range (32 to 64 Hz), which involves the gamma frequency (40 Hz), has been a widely studied phenomenon in cognition. The QEEG variables that are measured on patients are compared to the normative database values for attention, memory and problem-solving in order to calculate the deviations from the normal group in each cognitive task. In particular the method analyzes the variables that are related to success at the task. Treatment protocols are selected that address the deficits indicated by the comparisons. Treatment consists of the operant conditioning of the relevant QEEG variables while the subject is engaged in a relevant task, such as reading, auditory memory or problem solving.

Assessment of Cognitive Rehabilitation Programs for the TBI patient

In this section, we review and summarize the evidence for the effectiveness of the interventions in three ways. First, we summarize the conclusions from reviews of the literature completed in the last two decades (Cappa et al., 2003; Chestnut et al., 1998; Cicerone et al., 2000). Second, we use an effect size analysis, which is a statistical approach to summarize the data available in published reports (Cohen, 1969; Hedges & Olkin, 1985). We included in this review only those research articles that supplied the statistical data required to calculate the effect size. These data include pre- and post-treatment means and standard deviations of the measures of cognitive processes. A Medline search for cognitive rehabilitation and traumatic brain injury rehabilitation was conducted to include in the analyses articles published since 2000.

Third, we report the methodology of the studies in terms of use of control groups to provide the reader

with information on the quality of the research reported. The use of a control group (wait list, alternate treatment) is considered to be methodologically superior to research reports which do not employ a control group. However, many of the published studies have methodological weaknesses in terms of a lack of randomization to treatment and control groups, small sample sizes, a lack of control groups and similarity of measures. This article attempted to address the similarity of measures problem by examining studies which employed the same or similar outcome measures. Some equivalency of outcome measures was obtained with the auditory memory measures of paragraph, word list recall and problem solving. However, attention measures have a history of diverse instruments.

Due to these limitations definitive effectiveness statements are difficult to render. The reader will need to keep these qualifications in mind when reviewing the data. Relevant methodological information reported in the research articles are provided in this paper.

Position Statements and Literature Reviews on Effectiveness of Cognitive Rehabilitation

One of the initial reviews of memory rehabilitation, using strategy instruction, indicated inconsistent results, adding that the identification of specific treatment effects is hindered by methodological inadequacies (Benedict, 1989). Since that review, three additional reviews of the literature on cognitive rehabilitation have been completed in the past decade (Cappa et al., 2003; Chestnut et al., 1998; Cicerone et al., 2000).

The Agency for Healthcare Research and Quality (AHRQ) investigated whether the application of cognitive rehabilitation enhanced outcomes for people who sustain TBI (Chestnut et al., 1998, 1999). The AHRQ report is a review of 2,603 studies published from 1982 to 1997 and, via reviews of abstracts, reduced the list to 114 studies that met the eligibility requirements of Class I, II, or III studies. Well-designed randomized controlled trials (RCTs) were rated as Class I. Studies rated as Class II were RCTs with design flaws; well-done, prospective, quasi-experimental or longitudinal studies; and case-control studies. Case reports, uncontrolled case series, and expert or consensus opinion were generally rated Class III. A "gray zone" exists between Class II and definite Class III articles. Much of the research in rehabilitation uses quasi-experimental designs, which lack control over the constitution of the compared groups. Addressing cognitive rehabilitation, 16 randomized controlled trials and comparative studies that met specified inclusion criteria were placed into evidence tables. Within all these studies there was only sufficient evidence from two studies (Class I and III) that a compensatory approach reduced everyday memory failures in the TBI patient and two studies (Class I and II) that support computer assisted interventions for memory rehabilitation.

The AHRQ report concluded that there is evidence from three Class I studies using randomized controlled trials that the restorative technique of practice, both with and without the aid of a computer, operates to improve short-term recall on laboratory tests of memory for people with TBI, thus providing some evidence for the restorative cognitive rehabilitation approach. It should be noted that 70% of the research studies focused on the three specific cognitive skill areas of attention and concentration, memory, and concept formation. Table 1 presents a comparison of the effectiveness of cognitive rehabilitation programs to improve cognitive skills by reporting the number of positive and negative outcome studies for the three types of evidence (RCT, Comparative, Correlational). Comparative studies examined pre and post treatment employment outcomes or performance measures on neuropsychological instruments. Correlational outcome reports involve a significant relationship between a test and a health outcome or employment. In addition, the percentage is obtained showing positive results of studies relative to the total number of studies.

While the AHRQ report presented favorable results for cognitive rehabilitation programs, a different conclusion was reported in a review of 171 studies that addressed specific cognitive deficits in TBI (Cicerone et al., 2000). Using evidence-based clinical practice criteria, Practice Guidelines were recommended for interventions for 1) attention (during the post acute stage), with the caveat that the effects can be relatively small or task specific and there is insufficient evidence to indicate improvement over spontaneous recovery during the acute recovery stage; 2) memory, using memory notebooks as compensatory aide with mild memory deficits; and 3) problem solving. It was acknowledged that "no evidence exists to support the effectiveness of cognitive rehabilitation to restore memory functioning in subjects with severe memory impairment" (p. 1605). Practice Guideline criteria were based on well-designed class II studies (prospective cohort studies, retrospective case-control studies or clinical series with well-designed controls) with adequate samples that directly address the effectiveness of the treatment reviewed.

Table 1. A Comparison of the Effectiveness of Cognitive Rehabilitation Programs to Improve Cognitive Skills

Types of Studies		Attention and Orientation	Memory	Verbal and Language	Construction	Concept Formation	Executive Function and Motor	Global Tests (WAIS)	Total
RCT [a]	Number	12	13	1	2	1	0	1	30
	Positive Effects	0	1	1	1	0	0	0	3
	Negative Effects	12	12	0	1	1	0	1	27
	Percentage [c]	0	8	100	50	0	0	0	10%
Comparative Between Groups	Number	16	8	3	3	6	3	5	44
	Positive Effects	5	1	1	2	3	0	1	13
	Negative Effects	11	7	2	1	3	3	4	31
	Percentage	31	12	33	67	50	0	20	29%
Correlational Studies [b]	Number	16	14	5	3	9	5	10	62
	Positive Effects	9	8	0	2	4	2	6	31
	Negative Effects	7	6	5	1	5	3	4	31
	Percentage	56	57	0	67	44	40	60	50%
Total Studies		44	35	9	8	16	8	16	136

Source of studies: Chestnut et al., 1999

[a] RCT: Randomized Control Trials studies

[b] The number of correlational studies that report a significant correlation between the test and a health outcome or employment

[c] Percentage figures reflect the percentage of positive studies divided by total number of studies for each category

The report from the European Federation of Neurological Sciences (Cappa et al., 2003) concluded that "no evidence is available concerning effective restoration of memory functioning in patients with severe memory impairment" (p. 7). The authors concluded that there is enough overall evidence to recommend some forms of cognitive rehabilitation in patients with neuropsychological deficits after TBI. These include attention training after TBI in the post-acute stage and memory rehabilitation with compensatory training in patients with mild amnesia (Cappa et al., 2003).

Not included in any of the three previous reviews was a Veteran's Administration review of their cognitive rehabilitation program which failed to find any statistical significant effects (compared to a home treatment strategy training group) with cognitive rehabilitation methods in a group of moderate to severe TBI patients (Salazar et al., 2000).

In conclusion, all reviewers agreed upon the use of memory aides and two of the three reviews agreed upon attention interventions in the post-acute stage (Cappa et al., 2003; Cicerone et al., 2000). However, problematic in this memory recommendation is the long-term follow up in one study that failed to find positive long term effects of this approach at 6 months compared to supportive psychotherapy (Chaytor & Schmitter-Edgecombe, 2003; Schmitter-Edgecombe, Fahy, Whelan, & Long, 1995). These recommendations, however, must be viewed in light of the totality of research as well as the magnitude and longevity of the effects. Although no intervention is successful 100% of the time, the ratio figures presented in the AHRQ report are not encouraging (Chestnut et al., 1998).

Effect Size Analyses

We will examine this research area from a viewpoint of effect sizes and include QEEG biofeedback research which was not available at the time of the earlier reviews (Cappa et al., 2003; Chestnut et al., 1998; Cicerone et al., 2000). In order to obtain an effect size statistic, it is necessary to have the mean scores on standardized tests from both the pre-treatment and post-treatment assessments, as well as the measures of the standard deviations of the treatment group on the standardized test. The effect size (ES) for the treatment is calculated using the formula: the post-treatment mean score minus the pre-treatment mean score, divided by the standard deviation of the pre-score (Cohen, 1969). It was judged that the ES approach was the most appropriate in comparing alternate treatment interventions. This provides a change score in cognitive functioning from pre-treatment to post-treatment in standard deviation units, thus allowing a comparison of changes in functioning due to the treatment. In addition, the ES is bias-adjusted for the size of the sample (Hedges & Olkin, 1985). Appendix A presents the rationale for the effect size analysis as well as the details and examples of the effect size calculation.

The analysis of effect sizes is organized by the cognitive functions of memory, attention and problem-solving, and then a review of the effect size of followup studies. This manuscript is limited to publications with reported effect sizes or with the statistics required to obtain effect sizes, specifically the pre- and post-treatment means and standard deviations. Medline searches for medication interventions for TBI yielded articles for the effects of anti-depressants, methylphenidate and Bromocriptine but not for amantidine, Focalin, D-amphetamine, levodopa, and modafinil.

Many of the research studies employed the same standardized measures of memory, attention, and problem-solving, lending credibility to this comparison of the effectiveness of interventions. Memory ability is assessed by either paragraph recall or by list learning. Standardized tests of memory are the paragraph recall subtest of the Wechsler Memory Test – III (Wechsler, 1945), the Rey Auditory Verbal Learning Task (RAVLT; Rey, 1941), and the California Verbal Learning Test (CVLT; Delis, Kramer, Kaplan, & Ober, 1987), a well-standardized variation of the RAVLT for list learning. Several standardized tests of attention ability are the digit span of various forms of the Wechsler Adult Intelligence Scale (WAIS; Wechsler, 1981); the Paced Auditory Serial Addition Test (PASAT; (Gronwall, 1977); and variations of the continuous performance test (CPT) such as the Conner's CPT (Mental-Health Systems), the Tests of Variables of Attention (TOVA; Universal Attention Disorders), and the Integrated Visual and Auditory Continuous Performance Test (IVA; Brain Train). Attentional resources are assumed to be involved in other cognitive tasks, such as cancellation tasks, Trail Making tasks (Reitan & Wolfson, 1993), and the Stroop Test (Stroop, 1935). Standardized tests of problem solving include the Category Test (Psychological Assessment Resources, Inc., PAR), which measures concept formation, and the Wisconsin Card Sorting Task (WCST; PAR, 1993) which measures perseveration.

Effect Size Analysis of Rehabilitation of Memory

The effect sizes of studies that addressed auditory memory are presented in Tables 2 and 3. Table 2 presents a comparison of interventions to improve paragraph recall and Table 3 presents a similar analysis for word lists. Both tables present for each study the effect size (ES) and 95% confidence interval of the ES for changes in the pre- to post-intervention scores for the treatment group and the ES for the scores of the intervention group compared to scores form the control group when available. In addition, the overall clinical effectiveness rating discussed in this article is presented, which provides a rating that ranges from a low of 0 to a high of 4. Appendix B provides the sample sizes, and the number of sessions for each study.

Criteria for effectiveness

The clinical effectiveness (CE) criterion provides the following rating system.
CE 0 rating: effect sizes below .50 are considered to be not clinically significant;
CE 1 rating: effect sizes between .50 and 1.00 are considered to be mildly significant;
CE 2 rating: effect sizes between 1.00 and 2.00 are considered to be moderately significant;
CE 3 rating: effect sizes between 2.00 and 3.00 are considered to be highly significant;
CE 4 rating: effect sizes greater than 3 are considered to be extremely significant.

To obtain the clinical effectiveness of the different approaches, it was assumed that if the confidence interval of the effect size fell below 0, then there was no clinical effectiveness of the intervention. Each study was assigned a value of 0 (if the confidence interval of the ES included zero) or the effect size value. The values were then averaged across all studies for each intervention. Studies for which confidence intervals could not be calculated were reported but not included in the clinical effectiveness ratings or averaging value.

The 'outside approaches' (computers, strategies) had an average ES of .10 across both auditory memory tasks, while 'inside approaches' (QEEG, medications) averaged 2.07 ES.

Two computer intervention studies averaged 0.00 ES for paragraph recall (Table 2) and one study obtained a +.72 ES for word lists (Table 3). Strategy instruction in seven studies showed improvements averaging +.19 ES for paragraphs (Table 2) and 0.00 ES for word list recall (Table 3).

Antidepressant medications showed a +.52 ES improvement in paragraph recall (Table 2) (Fann et al., 2001) and a 0.00 ES effect on word lists. The QEEG Flexyx (LENS) approach scores on the RAVLT had a zero ES on immediate post treatment evaluation. A +.90 ES at a 2-3 month reevaluation time period was reported with the same word list, which suggests a probable practice effect (Schoenberger, et al. 2001). Due to the practice effect problem the +.90 ES was not included in the analysis. A SQ approach (Stephens, 2006) obtained a -.39 ES on word list recall

The activation QEEG treatment for the improvement of paragraph recall performance obtained gains in paragraph recall of +2.89 ES (with 95% confidence interval of 1.87 to 3.92). This represents an improvement in memory scores of 252%. A comparison of the performance on average recall (AR) between control (paid volunteer subjects recruited by advertising) and treated groups shows the effectiveness of the intervention. The pretreatment TBI group (N=15, Mean=8.75, SD=4.51) had lower scores then the control group (N=15, M = 18, SD = 2.45) for an ES of -2.48 (95% CI -3.43 to -1.53). Post-treatment scores for the QEEG group (M = 25.60, SD = 6.62) obtained an ES of 2.04 (95% CI 1.26 to 2.92).

Effect Size Analysis of Rehabilitation of Attention

Table 4 presents the comparisons of the different approaches for improvement of attention. Outside interventions averaged an ES of .23 (9 studies) while inside approaches averaged .72 ES (4 studies). The 'inside approach using medications have effect sizes that average +.00. Combined EEG biofeedback and computer training approaches (Tinius & Tinius, 2000) resulted in improvements in attention (+.94 ES) in the experimental group that were not significantly better than the control group. Keller (2001) employed the standard QEEG approach (increase beta amplitude in microvolts, decrease theta amplitude in microvolts) at the Fz location rather than commonly used Cz location. This intervention was compared to a standard computerized cognitive attention training, which focused on speed of information processing and selective attention for 10 sessions of 30-minutes (COGPACK; Marker, 1996; Siegmund, 1999). Superior results were found for the standard QEEG group (+2.09 ES) compared to the group receiving standard computerized training only for the letter cancellation.

In a second QEEG study, the Flexyx system (Schoenberger, et al., 2001) improved attention measures

Table 2. A Comparison of Interventions to Improve Memory for Paragraph Recall

Intervention and Reference	Comparison	Effect Size and 95% Confidence Interval #
Computer		
Kerner & Acker, 1985	Post-test scores Treatment Group vs. Control Group	
	Immediate testing	.26 (-.55 to 1.06)
	30-day follow-up	.26 (-.55 to 1.06)
Gray & Robertson, 1992	Post- vs. Pre-treatment scores from Treatment Group	
	Immediate memory	.12 (-.55 to .79)
	Delayed memory	-.05 (-.72 to .63)
	Post- vs. Pre-treatment scores from Control Group	
	Immediate memory	.06 (-.68 to .80)
	Delayed memory	-.16 (-.90 to .58)
	Average for Computer For Treatment Groups	**Clinical Effect = 0**
	Immediate memory	**Avg. = +0.00**
	Delayed memory	**Avg. = +0.00**
Strategies		
Ryan & Ruff, 1988	Post- vs. Pre-treatment scores from Treatment Group	
	Immediate memory	.79 (.05 to 1.53)
	Delayed memory	.74 (.00 to 1.48)
Freeman et al., 1992	Post- vs. Pre-treatment scores	
	Control Group	.45 (-.70 to 1.59)
	Treatment Group	1.15 (-.07 to 2.37)
Cicerone et al., 1996	Post- vs. Pre-treatment scores	
	Immediate Logical Memory	.35 (-.56 to 1.25)
	Delayed Logical Memory	.66 (-.26 to 1.58)

Kaschel et al., 2002	**Post- vs. Pre-treatment scores**	
	Pragmatic control group	**-.10 (-.91 to .70)**
	Imagery treatment	**.71 (-.24 to 1.67)**
Laatsch & Stress 2000	Post- vs. Pre-treatment scores	
	Immediate Verbal Memory	.46 (0.0 to .93
	Delayed Verbal Memory	.71 (.22 to 1.19)
Quemada et al, 2003	Post- vs. Pre-treatment scores RBMT	.29 (-.51 to 1.10)
Fasotti et al., 2000	Post- vs. pre-treatment group	
	RBMT	.26 (-.54 to 1.06)
	Post-treatment vs. post-control	
	RBMT	.46 (-.39 to 1.31)
	Average for Strategies	**Clinical Effect = 0**
	For Treatment Groups	**Avg. +.19**
Antidepressants		
Fann et al., 2001	Post- vs. Pre-treatment scores for Treatment Group	
	Immediate Logical memory	.70 (-.03 to 1.44)
	Delayed Logical Memory	1.05 (.29 to 1.82)
	Average Antidepressants	**Clinical Effect = 1**
		Avg. +.52
Activation QEEG		
Thornton, this article	Post- vs. Pre-treatment scores for Treatment Group	
	Paragraph recall –	2.89 (1.87 to 3.92)
	Compare treatment to control group	
	Pre-treatment group starts with lower score than control group	-2.48 (-3.43 to -1.53)
	Post-treatment group ends with higher score than control group	2.04 (1.16 to 2.92)
	Average Activation QEEG	**Clinical Effect = 4**
	Most conservative estimate of change	**Avg. +2.89**

Effect size and confidence intervals were calculated using the methods of Hedges & Olkin (1985)
See Appendix B for details on the number of subjects and length of treatment

Table 3. A Comparison of Interventions to Improve Memory for Word Lists

Intervention and Reference	Comparison	Effect Size and 95% Confidence Interval #
Computer		
Ruff et al., 1994	Pre Post-test scores Treatment Group Rey	 .72 (.02 to 1.46)
	Average for Computers	**Clinical Effect = 1 Avg. =.72**
Strategies		
Ryan & Ruff, 1988	Post- vs. Pre-treatment scores Treatment Group RAVLT	 -.02 (-.64 to .60)
Niemann et al, 1990	Post- vs. pre-treatment scores Attention training group Memory training group	 .24 (-.53 to 1.01) .41 (-.37 to 1.19)
Cicerone, 1996	Post- vs. Pre-treatment scores Rey Post – vs. Pre-treatment scores CVLT Combined 20 subjects	.33 (-.56 to 1.21) .08 (-.82 to .98)
Milders et al., 1998	Post- vs. Pre-treatment scores Rey	 .57 (-.22 to 1.35)
Fasotti et al., 2000	Post- vs. pre-treatment group Rey Post-treatment vs. post-control Rey	 .43 (-.38 to 1.24) .30 (-.55 to 1.14)
Quemada et al, 2003	Post- vs. pre-treatment scores Rey	.45 (-.36 to 1.26)
Stephens, 2006	Post- vs. Pre-treatment scores Rey total Cognitive rehab	.39 (-.49 to 1.28)
	Average for Strategies For Treatment Groups	**Clinical Effect = 0 Avg. +.00**
Antidepressants		

Fann et al., 2001	**Selective reminding LTR**	**49 (-.23 to 1.22)**
	Average	0.00
Modified QEEG		
Schoenberg et al., 2001	Post- vs. pre- treatment AVLT combined immediate and delayed	.23 (-.57 to 1.04)
	Average	**Clinical Effect = 0** **Avg. = 0.00**
Standard QEEG		
Stephens, 2006	Post- vs. Pre-treatment scores Rey total Neurotherapy group	-.34 (-1.23 to .54)
	Average	**Clinical Effect=0** **Ave. = 0.00**

Effect size and confidence intervals were calculated using the methods of Hedges & Olkin (1985)
See Appendix B for details on the number of subjects and length of treatment

Table 4. A Comparison of Interventions to Improve Attention

Intervention and Reference	Comparison	Effect Size and 95% Confidence Interval [#]
Computer		
Ruff et al., 1994	Pre Post-test scores Treatment Group	
	Digit symbol CPT	.32 (-.40 to 1.05)
Gray & Robertson, 1994	Post-treatment vs. control	
	PASAT	.46 (-.26 to 1.17)
	digit span-forward	-.02 (-.73 to .69)
	letter cancellation-errors	.33 (-.38 to 1.04)
Park et al., 1999	PASAT	
	Post- vs. pre-intervention	3.06 (2.21 to 3.92)
	Post- vs. pre-control	4.01 (3.01 to 5.01)
	Post-intervention vs. post-control	-2.64 (-3.43 to -1.85) The post-intervention group was significantly lower than post-control group
	Average	**Clinical Effect = 0 Avg. = 0.00**
Strategies		
Ryan & Ruff, 1988	Post- vs. Pre-treatment scores Treatment Group	
	RAVLT	-.02 (-.64 to .60)
Niemann et al, 1990	Post- vs. pre-treatment scores	
	PASAT-R	
	Attention training group	.58 (-.20 to 1.37)
	Memory training group	.66 (-.13 to 1.45)
	Trail-Making Test B	
	Attention training group	.73 (-.06 to 1.53)
	Memory training group	.25 (-.53 to 1.02)
	Divided attention test	
	Attention training group	.61 (-.18 to 1.40)
	Memory training group	.57 (-.22 to 1.35)
	Test d2	
	Attention training group	.61 (-.17 to 1.40)

Niemann et al, 1990	Attention training group	.39 (-.39 to 1.16)
	Memory training group	
	Average over tasks	
	Attention training group	.63
	Memory training group	.47
Cicerone, 1996	Post- vs. Pre-treatment scores	
	Combined 20 subjects	
	Digit span forward	.01 (-.87 to .88)
	Digit span backward	.25 (-.65 to 1.14)
	Trail making test B errors	-.17 (-1.07 to .74)
	PASAT	.46 (-.60 to 1.52)
	CPTA errors	-.38 (-1.28 to .53)
	Average for all tasks – reverse sign for errors	.25
Fasotti et al., 2000	Post- vs. pre-treatment group	
	PASAT	.66 (-.17 to 1.48)
	Visual simple RT	.09 (-.75 to .93)
	Post-treatment vs. post-control	
	PASAT	.17 (-.67 to 1.01)
	Visual simple RT	.32 (-.53 to 1.16)
Laatsch & Stress, 2000	Stroop speed	
	Post- vs. pre-intervention	.56 (.07 to 1.04)
Salazar et al., 2000	PASAT	
	Post- vs. pre-home	.85 (.45 to 1.25)
	Post- vs. pre-hospital	.79 (.44 to 1.14)
	Post-hospital vs. post-home	.04 (-.32 to .40)
Keller 2001	Post vs. pre-intervention (reverse sign)	
	Cog Rehab	
	Letter cancellation – post intervention	.49 (-.40 to 1.38)
	Sustained attention errors — post intervention	1.19 (.24 to 2.14)
	Choice RT – post intervention	.97 (.05 to 1.90)
Kaschel et al., 2002	Post- vs. Pre-treatment scores	
	d2 test of concentration endurance	
	Pragmatic control group	27 (-.54 to 1.07)
	Imagery treatment	.69 (-.26 to 1.64)

Stephens, 2006	Cog rehab group	
	Symbol Search	.10 (-.78 to .98)
	Trails A	.00 (-.88 to .88)
	Trails B	.07 (-.81 to .94)
	TOVA	
	omissions	.19 (-.69 to 1.07)
	commissions	.00 (-.88 to .88)
	response time	.17 (-.70 to 1.05)
		This means longer RT in post than pre
	PASAT	-.01 (-.89 to .86)
	Average	Clinical Effect = 0
		Avg. = +.21
Sq Interventions		
Tinius and Tinius, 2000	Integrative visual and auditory continuous performance test	
	Post- vs. pre-treatment scores	
	Post-treatment vs. control group	.94 (.21 to 1.67)
		-.46 (-1.13 to .20)
Keller 2001	Post vs. pre-intervention (reverse sign)	
	EEG intervention	
	Letter cancellation – post intervention	3.92 (2.56 to 5.29)
	Sustained attention errors – post intervention	1.09 (.23 to 1.94)
	Choice RT – post intervention	.97 (.05 to 1.90)
	Compare EEG intervention to Cog Rehab intervention	
	Letter cancellation – post intervention	2.09 (1.05 to 3.13)
	Sustained attention errors – post intervention	.74 (-.13 to 1.61)
	Choice RT – post intervention	.47 (-.38 to 1.32)
Stephens, 2006	Neurotherapy group	
	Symbol Search	-.12 (-1.00 to .75)
	Trails A	.23 (-.65 to 1.11)
	Trails B	-.75 (-1.65 to .16)
	TOVA	
	omissions	-.70 (-1.60 to .20)
	commissions	-.82 (-1.73 to .09)
	response time	-.30 (-1.18 to .58)
		This means shorter RT in post than pre
	PASAT	-.22 (-1.10 to .66)

	Average	**Clinical Effect=1 Avg. = .70**
MQ		
Schoenberg et al., 2001	Post- vs. pre- treatment	
	PASAT trial 4	.86 (.02 to 1.69)
	Digit span backward	.83 (0.0 to 1.67)
	Digit symbol	.67 (-.16 to 1.49)
	Average for MQ Treatment	**Clinical Effect = 1 Avg. = +.56**
Antidepressants		
Whyte et al., 1997	Vigilance task – several measures	
	behavioral inattention	
	sorting productivity	,12 (-.51 to .76)
	percentage off task behavior	-.23 (-.87 to .41)
	duration off task behavior	-.49 (-1.14 to .15)
	collage productivity	.15 (-.49 to .78)
	Phasic arousal	-.24 (-1.08 to .60)
	Baseline RT	-.39 (-1.24 to .45)
	Optimal warning time	-.12 (-.96 to .71)
	Minimum RT	
	Distraction	.81 (-.06 to 1.68)
	Performance decrement d'	-.83 (-1.70 to .04)
	Performance decrement RT	.11 (-.73 to .95)
	Performance decrement yes rate	
McDowell et al., 1998	Stroop	.7 *
	Trails	.35 *
Fann et al., 2000	Post- vs. pre-intervention	
	Digit Span	-.12 (-.84 to .60)
	Digit symbol	.42 (-.30 to 1.14)
	Trail making – composite	.58 (-.15 to 1.31
Leon-Carrion et al., 2000	Attention	
	Post- vs. pre-intervention	.66 (-.61 to 1.93)
	Post- vs. pre-placebo	1.21 (-.13 to 2.56)
	Post-intervention vs. Post- placebo	1.01 (-.31 to 2.32)
	Average	**Clinical Effect = 0 Avg. = +0.00**

Effect size and confidence intervals were calculated using the methods of Hedges & Olkin (1985)
See Appendix B for details on the number of subjects and length of treatment
* author gave effect size and means; lack of SD prevented CI calculations

Table 5. A Comparison of Interventions to Improve Problem Solving

Intervention and Reference	Comparison	Effect Size and 95% Confidence Interval #
Computer		
Gray & Robertson, 1992	Post- vs. Pre-treatment scores from Treatment Group	
	WCST errors	.56 (-.13 to 1.24)
	WCST perserverative	.42 (-.30 to 1.13)
	Post- vs. Pre-treatment scores from Control Group	
	WCST errors	.63 (-.10 to 1.35)
	WCST perserverative	.61 (-.08 to 1.30)
	Average	**Clinical Effect = 0 Avg. = 0**
Strategies		
Cicerone et al., 1996	Post- vs. Pre-treatment scores	
	WCST	.20 (-.60 to 1.00)
	Category Test	.11 (-.91 to .69)
Laatsch & Stress, 2000	WCST perseverative errors on category test	
	Post- vs. pre-treatment	.67 (.17 to 1.16)
	Average for Strategies For Treatment Groups	**Clinical Effect = 0 Avg.= +.34**
EcQ & Strategies		
Tinius & Tinius, 2000	Post- vs. Pre-treatment scores from Treatment Group	
	WCST trials	91 (.18 to 1.64)
	WCST perserverative	.77 (.05 to 1.49)
	Post- vs. Pre-treatment scores from Control Group	
	WCST trials	.16 (-.46 to .78)
	WCST perseverative	.12 (-.50 to -.74)
	Average intervention group Eyes Closed	**Clinical Effect = 1 Avg. = +.84**
Medication		
McDowell et al., 1994	Bromocriptine vs. placebo	
	WISC perseveration	.55 *
	Average medication	**Clinical Effect = 1 Avg.= +.55**

Effect size and confidence intervals were calculated using the methods of Hedges & Olkin (1985)
See Appendix B for details on the number of subjects and length of treatment

Table 6. A Comparison of Long Term Effects of Interventions

Intervention and Reference	Comparison	Effect Size and 95% Confidence Interval #
Gray & Robertson, 1992	6 month followup vs. pre-intervention	
Computer intervention	for intervention group	
	LM immediate memory	.43 (-.25 to 1.11)
	LM delayed memory	.51 (-.17 to 1.19)
	6 month followup vs. pre-intervention	
	for control group	
	LM immediate memory	.30 (-.45 to 1.04)
	LM delayed memory	.26 (-.48 to 1.01)
	6 month followup vs. pre-intervention	
	for intervention group	
	WCST errors	-.57 (-1.25 to .12)
	WCST perserverative	-.51 (-1.19 to .18)
	6 month followup vs. pre-intervention	
	for control group	
	WCST errors	-.70 (-1.47 to .06)
	WCST perserverative	-.62 (-1.38 to .14)
Kerner & Acker, 1985	Memory index	
Computer intervention	30-day follow-up vs. pre-intervention	.35 (-.46 to 1.16)
	45-day follow-up vs. pre-intervention	.19 (-.61 to .99)
	Average for computer interventions	**Clinical Effect =0**
	For Treatment Groups	**Avg. = 0.00**
	For Control Groups	**Avg. = 0.00**
Strategies		
Kaschel et al., 2002	Follow-up vs. Pre-treatment scores	
	Pragmatic group	
	RBMT immediate	.40 (-.41 to 1.20)
	RBMT delayed	.49 (-.33 to 1.30)
	Imagery group	
	RBMT immediate	1.89 (.78 to 3.00)
	RBMT delayed	2.10 (.87 to 3.14)
	Follow-up vs. 2nd-baseline scores	

Kaschel et al., 2002	Pragmatic group	
	RBMT immediate	12 (-.68 to .92)
	RBMT delayed	.06 (-.74 to .86)
	Imagery group	
	RBMT immediate	1.24 (.23 to 2.25)
	RBMT delayed	1.16 (.16 to 2.16)

	Average Strategies- Imagery	**Clinical Effect = 2 Avg. = +1.20**
Schoenberg et al., 2001 Modified QEEG	3 month followup vs. post-intervention score Attention	
	PASAT	.36 (-.44 to 1.17)
	Digit span	.18 (-.62 to .98)
	Digit symbol	.18 (-.62 to .98)
	3 month followup vs. pre-intervention score Attention	
	PASAT	1.22 (.35 to 2.09)
	Digit span	1.01 (.16 to 1.86)
	Digit symbol	.85 (.01 to 1.68)
	Average intervention group	**Clinical Effect=2 Avg. = +1.02**

Effect size and confidence intervals were calculated using the methods of Hedges & Olkin (1985)
See Appendix B for details on the number of subjects and length of treatment
* Author gave effect size and means; lack of SD prevented CI calculations

Table 7 – Recommendation Criteria

Levels of Recommendation	Memory for Paragraph Recall	Memory for Word Lists	Attention	Problem Solving
1 – Not Recommended	CE=0 -Computer, Strategies	CE=0 - Computers, Strategies Flexyx/MQ	CE=0 - Computer, Strategies Medications	CE=0 Computer Strategies Medications EcQ/strategies
2 – Mild Recommendation	CE=1 - Medications **CE=2 - Imagery	CE=1 - Computers	CE=1 -Mq, Sq **CE=2 -Mq	CE=1 - ECq
3 - Moderate Recommendation	CE=4 -Activation QEEG			

CE – clinical effectiveness

**long term effectiveness rating

mmediately following treatment (+.56 ES) and at a 3 month follow (+1.02 ES). In summary, the four EEG biofeedback interventions averaged improvements of +.63 ES on attention measures. The two medication studies on attention showed an ES of 0.00.

In conclusion, only the qEEG approaches consistently showed improvements above +.50 ES while the computer interventions and strategy training averaged below +.50 ES.

Effect Size Analysis of Rehabilitation of Problem Solving

Table 5 presents the treatment effect comparison across the different approaches to problem solving abilities.

Outside interventions averaged .34 ES while a combined inside and outside approach obtained a .84 ES. Computerized interventions that were designed to improve attention obtained no significant generalization to problem-solving measures in terms of decreased errors on the WCST, no difference from the control group (Gray & Robertson, 1992). A more general neuropsychological rehabilitation program (that did not specify the interventions) did not show significant reduction in the perseveration score on the WCST or reduced error scores on the Category Test (Cicerone et al., 1996). Another strategy intervention approach to improving problem solving (Laatsch & Stress, 2000) did show a significant ES effect (.67), resulting in an average improvement of .33 ES for the strategy approach. The medication approach demonstrated a +.55 ES improvement with Bromocriptine on the WCST perseveration measure (McDowell et al., 1994).

Employing an EcQ intervention model, improvements (decreased error scores) were obtained on the WCST number of trials to complete category #1 (-.91 ES) and perseveration errors (-.77 ES) compared to an effect of +.00 (increased number of trials, .17 ES) and non significant perseverative score change (0.00 ES) for the control group that received no intervention (Tinius & Tinius, 2000).

In conclusion, only the combined strategy training and QEEG approach was respectable in ES improvements.

Effect Size of Follow-up Studies

As shown in Table 6, there are five studies that included data on the follow-up effectiveness of interventions. For computer interventions, there is an average improvement in memory and problem-solving of 0.00 for both the treatment group and the control group. All effect sizes include zero in the confidence intervals, suggesting two conclusions. First, the interventions do not have an effect that is statistically reliable. Second, the ES that is found is shown by the control group and may be attributed to the repeated administrations of the measures of memory and problem-solving. The strategies intervention used by Kaschel et al. (2002) shows a follow-up ES for memory improvement of 2.00 compared to a control group ES of .45, which includes a zero in the confidence interval. The strategies intervention is clearly effective and differs from the improvements expected on repeated test administrations. In contrast, the strategies program by Milders et al. (1998) showed an ES of .15 at 6 month follow-up, which was markedly less than the .78 ES for repeated test administrations for the control group. The QEEG Flexyx approach (Schoenberger, et al., 2001) showed an ES of .56 for 3 measures of attention. The lack of a control group for follow-up precludes a comparison to ensure that the improvements are not attributed to repeated testing.

Summary of Effect Size Analyses & Recommendations

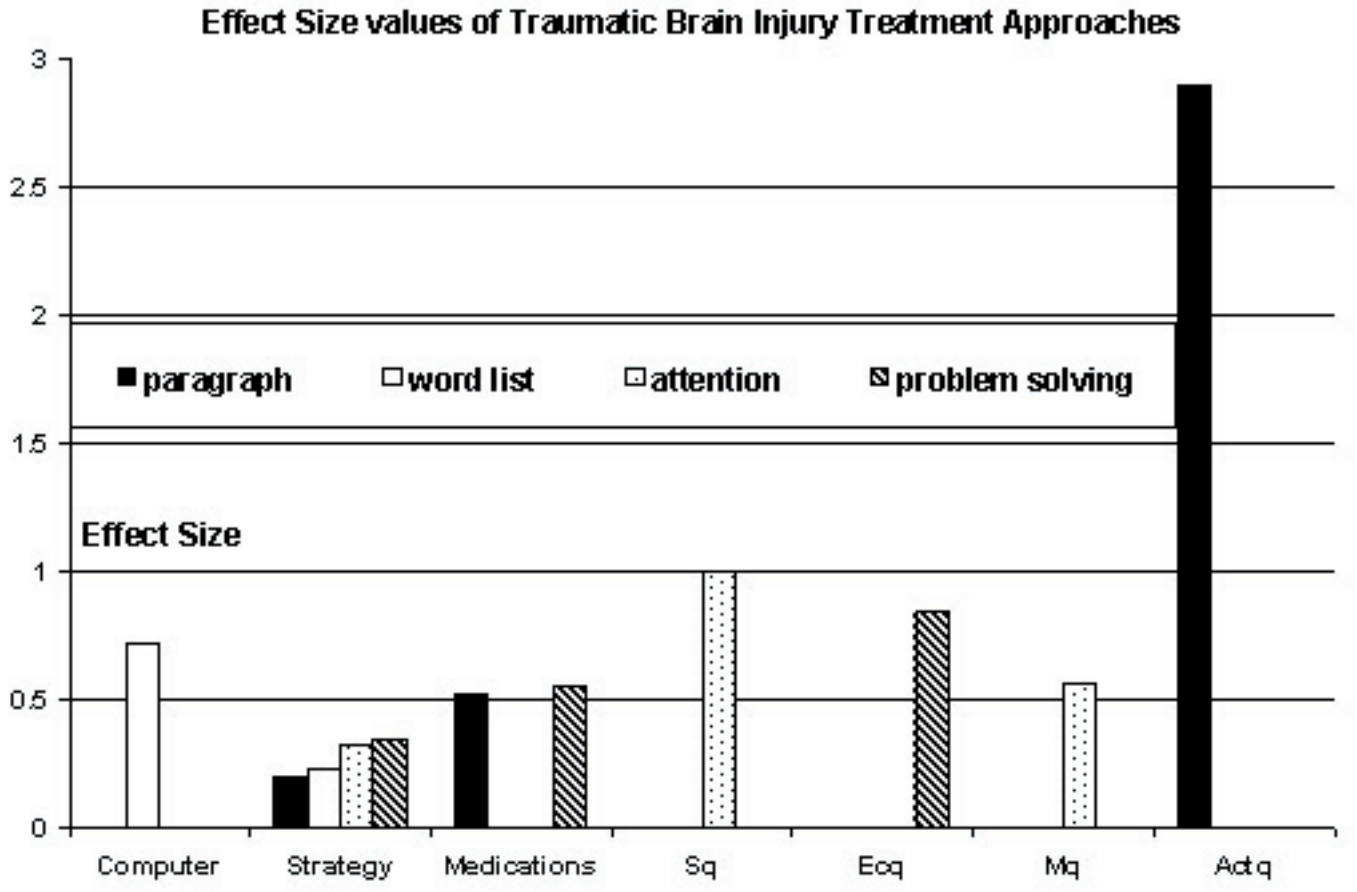
Figure 1 presents the results of the comparisons. Overall there were five qEEG biofeedback studies, five computer intervention studies, fourteen studies which involved strategy interventions and four studies involving the effect of medication. For paragraph recall mild recommendations could be made for medications and imagery (based on long term effects) and moderate recommendations for the ActQ intervention model. For word list recall the only a mild recommendation for computer intervention could be rendered. For attentional abilities, the modified QEEG and standard QEEG approaches both received mild recommendations. For problem solving only the eyes closed QEEG approach obtained a mild recommendation.

Additional effectiveness issues involve generalization to other cognitive abilities, long term effects and time or cost. Since most interventions did not obtain clinically significant results, generalization becomes impossible to meaningfully measure. Only two approaches demonstrated any long term effects; imagery for paragraph recall and the modified QEEG approach for attention. The auditory memory improvements were maintained from 1 month to 11 months on repeat testing for four subjects in an ActQ treated TBI sample (Thornton & Carmody, 2005). The QEEG biofeedback literature indicates that the effects of QEEG biofeedback can last up to ten years (Lubar, Swartwood, Swartwood, & O'Donnell, 1995; Tansey, 1993). Intervention times ranged from 10 to 132

sessions. The correlation between the ES effect (ignoring issues of confidence intervals) and number of sessions was +.09 (9 studies) for paragraph recall and -.17 for attentional abilities (12 studies).

It should be kept in mind, however, that to expect any significant change in 10 sessions is overly optimistic for a brain injured subject. While cost is always a factor, the long term costs of failure to rehabilitate far outweigh the type of cost structures evident in this analysis. The four QEEG approaches dominate the recommendation results in Table 7 and appear to be the most promising to obtain meaningful results.

Figure 1
Comparison of Effect Sizes of different interventions across different cognitive abilities



QEEG Biofeedback Treatment Schedules

There are many choices available to the practitioner using QEEG biofeedback. Among the choices to be made are the scalp location(s) for measuring brain activity during baseline and feedback, the frequency band (delta, theta, alpha, or beta and their respective definitions in Hertz), the parameter of the frequency band (magnitude, relative power, peak frequency, etc.), amplitude relationships between locations (symmetry) and/or the connection variables between locations (coherence, phase). In addition, protocols can be set either to inhibit or reward a single variable or simultaneously reward and inhibit a set of variables. A treatment protocol can address any of these variables. The number of possible single variable protocols (reward only) available when addressing data up to the 64-Hertz range is 2,945, assuming the entire frequency range can be divided into 5 frequencies (delta, 0-4 Hertz; theta, 4-8 Hertz; alpha, 8-13 Hertz; beta1, 13-32 Hertz; beta2, 32-64 Hertz). If two variables are to be simultaneously addressed, such as simultaneously rewarding one frequency band and inhibiting a second band, the number of possible protocols increases to almost 5,890. Any further divisions of the frequency ranges would

only increase these numbers dramatically. We review several of the more popular protocols used for improving the cognitive abilities of attention, memory and problem-solving. However, only a few of these have reported the means and standard deviations necessary for this analysis, which we have reported.

Protocol 1: Location Interventions: Beta enhancement and theta inhibition

In a single case study, the subject was treated for 31 sessions with two intervention protocols. The first was designed to suppress 4-7 Hz while enhancing 12-15 Hz and 15-18 Hz beta amplitude activity at Cz (top central location on head) and the second was designed to increase 15-18 Hz at T3 & C3 while inhibiting theta amplitude (4-7 Hz). Improvements were found when comparing pre- and post-administered cognitive problem solving measures (Category test, WCST) and other cognitive measures including verbal fluency and IQ scores (Byers, 1995). Theta amplitudes decreased an average of approximately 37% across the 3 locations, T3, C3, and Cz. on post testing. However, beta amplitudes also decreased an average of about 41%. There have been other research reports focusing on the theta and beta variables with the TBI patient (Ayers, 1993; Bounias, Laibow, Bonaly, & Stubbelbine, 2001; Bounias, Laibow, Stubbelbine, Sandground, & Bonaly, 2002; Laibow, Stubbelbine, Sandground, & Bounias, 2001; Norris & Hoffman, 1996; Stephens, 2006)

In a group study, 16 mild TBI patients were treated on the basis of QEEG normative reference group data (Thatcher database, 1987) by reducing theta activity (20 sessions) at location Cz (then C3 and C4, if necessary) if theta was too high (Tinius & Tinius, 2000). For the patients with theta values that were low in comparison to the database, the treatment was to increase SMR activity (12-15 Hz). Additional interventions addressed coherence training. The locations and frequencies were defined by comparing each patient to the database. The coherence values were increased when database comparison indicated low values and decreased when database comparison indicated higher than normative values. A comparison to the control group showed that TBI patients improved their attention and problem-solving abilities.

One study increased beta amplitudes (13-20 Hz) at location Fz for 10 sessions in the moderate TBI experimental group and compared the effectiveness to a matched control group who received standard cognitive rehabilitation attention training (Keller, 2001). Eight of the 12 TBI patients increased their beta levels and sustained the level for longer periods of time, while the remaining four (who started with high beta levels) showed a decrease in beat 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 amplitude levels nor their performance on the post treatment attentional measures of letter cancellation, simple choice reaction task and a sustained attention task. The author concluded that amplitudes "may not be the most important factor in cognitive change" (Keller, 2001, p. 26).

Stephens (2006) employed the Fz, Cz, P4 & C4 locations and inhibited theta (4-7 Hz) in some patients and alpha (7-13 Hz) in others while rewarding SMR (12-14 Hz) at those locations. As the intervention protocols were not consistently tied to the QEEG database analysis, this research was treated as an SQ method due to its consistency in inhibiting theta and rewarding low beta (12-14 Hz) microvolts. Across the 6 subjects on whom a post QEEG was conducted the consistent finding was an increase in beta (12.5-25 Hz) absolute microvolts at the F7 location.

Schoenberger et al. (2001) employed the Flexyx system (now called LENS), an QEEG biofeedback program that provides extremely low energy electromagnetic stimulation based on the dominant EEG amplitude, and which is designed to reduce EEG amplitudes. Wait list controls were employed. The subjects underwent 25 sessions of treatment. The results are presented in the tables. Subsequent research (Ochs, 2006) found that the operative mechanism was that the subject's dominant amplitude was being measured and then very low energy electromagnetic pulses were being delivered down the electrode wires. These electromagnetic pulses were offset at a higher frequency than the frequency of the dominant amplitude.

Protocol 2: Relationship Interventions: Coherence and Phase

Research conducted by Thornton (1999a, 1999b, 2002a) has documented the TBI deficit pattern which was most markedly demonstrated in the high frequency range (32-64 Hz) for coherence and phase relationships from frontal lobe locations. Thatcher (Thatcher, 2000) had previously concluded, "EEG coherence has been shown to be the most sensitive QEEG measure of TBI" (p. 42).

Self-report measures supportive of the treatment value of coherence interventions has been documented.

Walker addressed the cognitive problems in 26 mild TBI patients with QEEG biofeedback and employed the NX Link database (John, Prichep, Fridman, & Easton, 1988) to determine deficits (Walker, Norman, & Weber, 2002). Interventions focused on the coherence abnormalities for an average of 19 sessions to a maximum of 40. 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, as discussed earlier, these outcome measures are problematic and do not necessarily reflect restoration of cognitive abilities.

The activation database was employed in 9 cases of mild to moderate TBI to identify specific deficits in functioning for treatment intervention (Thornton & Carmody, 2005). The main variable that was identified was the deviation in the phase or coherence measures from the normative database. The deviations were addressed, one by one, with appropriate treatment protocols and employed a neuroanatomical/neuropsychological understanding of cortical location and function until the subject's values were at the normative value or above.

Protocol Recommendations

An assessment of the physical nature of the injury, time since injury and other relevant neurodiagnostic evaluations (SPECT, fMRI, CT, etc.) are useful sources of information in any TBI situation. Previous diagnostic studies may provide some relevant group patterns for the TBI patient. The patterns indicated in the literature concern all four frequency bands and coherence and phase values. The research reports do not always distinguish between or report microvolts and relative power values. However, as these measures are highly correlated the distinction is not critical at this point. The findings indicate higher magnitudes of the delta and theta frequencies and decreased magnitudes of the alpha band with conflicting evidence with respect to the beta frequency, possibly due to the time since injury variable. Decreases in the coherence and phase values in the high frequency band of 32-64 Hz appear to be the most salient finding for the TBI group (Thornton, 1999a, 1999b, 2002b).

Frequency Level

Thatcher (Thatcher, Walker, Gerson, & Geisler, 1989) reported decreased alpha in posterior locations in his discriminant function under eyes closed condition. Thornton (1999a – unpublished data) found increased theta relative power at O1-O2 under eyes closed condition. The Hughes and John (Hughes & John, 1999) review supported both results by indicating "increased focal or diffuse theta, decreased alpha" in TBI patients. Other research has also indicated potential problems in the theta frequency (Hooshmand, Beckner, & Radfar, 1989) for subjects 1 to 22 years post injury, while (Tebano et al., 1988) had confirmed the decreased alpha pattern (10.5-13.5 Hz) in cases of mild TBI at 3 to 10 years post injury.

Beta relative power and amplitude variables have proven to be more inconsistent in the research results. For example, one study reported a decrease in beta in the 20.5 to 36 Hz range (Tebano et al., 1988), while another report indicated increased amplitudes and amplitude variances in the beta frequency under task conditions for subjects 2-4 years post injury (Guskiewicz et al., 2003). An examination of the relationship between cognitive function, EEG amplitudes and MRI findings showed that the increased white matter T2 relaxation times in the TBI patient were related to increased amplitudes of the delta frequency, which was associated with cognitive dysfunction (Thatcher, Biver, McAlaster, & Salazar, 1998).

The T1 and T2 relaxation measures involve the relaxation (return to base functioning) time periods after the radio frequency pulse of the MRI has ceased. T1 relaxation (spin-lattice) corresponds to restoration of equilibrium between the numbers of nuclei in the high and low energy spin states. T2 relaxation, or spin-spin relaxation, occurs when the spins in the high and low energy state exchange energy but do not loose energy to the surrounding lattice.

Increases in gray matter T2 relaxation times were positively correlated with decreased amplitudes in the 7-22 hertz range. Thus one would expect the TBI patient's cognitive problems to be related to increased delta amplitudes, reflecting white matter effects, and to decreased alpha to beta amplitudes, reflecting gray matter effects. From this information, it would be appropriate to increase beta amplitude (in microvolts) and decrease delta amplitude in the TBI patient. However, this pattern was not replicated by Thornton (1999a, 1999b) who reported significant differences in the TBI group (under 1 to 43 years post injury), compared to normal controls in terms of diffuse increases (Thornton, 2003) in the relative power of beta1 (13-32 Hz) activity, under 3 cognitive condi-

tions which was consistently a negative predictor of cognitive performance. The control group consisted of paid volunteers who were recruited to establish a normative reference group. The pattern of increased relative power of beta activity (13-32 Hz) in response to a TBI, would strongly argue against beta interventions (either microvolts or relative power due to high inter-correlation) as appropriate for the TBI patient, especially in the one-year post injury time frame (this conclusion is also supported by Ayers, 1999).

Thornton (1999a) examined the effect of time on QEEG variables in TBI subjects with a discriminant function (eyes closed condition), which returned a .90 hit rate in distinguishing the groups (under one year post accident and greater than one year post accident). The comparison (previously unreported) indicated greater post-accident time was associated with decreases in beta1 at frontal and left posterior locations (relative power, peak amplitude, magnitude) and in beta2 at left posterior locations (magnitude, peak amplitude). Time since accident also was related to increased coherence alpha relationships (posterior, T5 in particular) and increased posterior phase beta1 (T5 in particular) and frontal phase theta (F7).

It is somewhat problematic in this beta conflict that the higher frequency ranges are intimately tied to metabolism rates. Oakes et. al. (2004) studied the relationship between the EEG frequency ranges and metabolic activity. The results indicated negative relationships between metabolic activity and lower EEG frequencies. The following correlations were obtained between Positron Emission Tomography (PET) measures of metabolism and a frequency: -.25 (6.5 – 8 Hz); -.38 (8.5 - 10 Hz); +.08 (10.5 - 12 Hz); -.06 (12.5-18 Hz); +.19 (18.5 – 21 Hz); +.34 (21.5-30 Hz); +.48 (36.5 – 44 Hz). Thus, the increased beta activity in the TBI subject could reflect the brain's compensatory attempts.

Coherence and Phase Relationships

The most powerful effects for coherence and phase relationships (in TBI cases) were reported in the Thornton (2003) study. Decreased phase and coherence values for the beta2 frequency were found at all cortical connection patterns, and were particularly dominant in the frontal regions. Lower frequency coherence patterns were also affected by the TBI, but to a lesser degree. Both the Thornton (1999b) study and the Thatcher (1989) study reported increased theta coherence values in the left frontal region, while Hughes and John (1999) reported decreased coherences (frequency not specified) in their review. Thatcher's discriminant function (1989) results 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). Particularly noteworthy (Thornton, 1999b) was the lack of spontaneous change in the coherence and phase beta2 patterns in the TBI subject, supportive of the conclusion that "time does not heal."

Neurofeedback training to decrease delta and theta values, increase alpha, and to increase coherence beta (32-64 Hz) would appear to be appropriate on the basis of group data research. Beta interventions are problematic given the conflicting research results. Given the complexity of the human brain and need to determine if and how a particular individual fits the typical TBI pattern it is advisable to obtain as much relevant information as possible (via use of a database) in order to individualize the treatment and maximize the potential success of the interventions.

Many of the early intervention studies (Ayers, 1993; Byers, 1995; Keller, 2001; Norris & Hoffman, 1996; Tinius & Tinius, 2000) provided positive feedback for increases in the amplitude (in microvolts) of the EEG in the beta band and/or for decreases in the amplitude of the EEG in the theta band (generally in the 4-8 Hz range). Ayers primarily inhibited theta, and only very minimally reinforced 15-18 Hz beta. Two studies that reported standard deviation effects used a database as a reference to guide the interventions for individual subjects (Thornton & Carmody, 2005; Tinius & Tinius, 2000).

The protocol discussion has not discussed the issue or value of addressing specific locations or relevance to specific cognitive function. The value of such specificity can be demonstrated in one specific clinical example. The subject was a middle age stunt actress who had experienced multiple mild-moderate TBI injuries during her career. The activation evaluation revealed deficits in coherence beta2 projection activity from the right frontal (F4) to left posterior locations (T5-P3-O1) during reading recall conditions. The interventions were directed towards this specific problem. In five sessions she improved the coherence values 6.32 SD and her reading memory improved some 91%. This case example, and others not presented, documents one of the problems with generic interventions. It is important scientifically to prove that by improving a known relevant variable to a specific

cognitive task, the performance on that specific cognitive task improves. Due to the complexity of traumatic brain injuries and positive preliminary results obtained from research guided by QEEG database comparisons, it appears that QEEG-guided neurofeedback with TBI may result in higher improvement rates.

Pre vs. Post QEEG results

Of particular importance is the examination of the effects of the different forms of rehabilitation on the QEEG. Only Stephens (2006) 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" (p. 182).

One post EEG biofeedback treatment QEEG case study has been reported (Thornton & Carmody, 2005). An additional subject is added in this report. The two subjects received only activation database guided EEG biofeedback and both improved on subsequent cognitive retesting (first case included in data reported). For the first case study subject, the main protocol intervention involved Fz coherence beta2 to all other locations. The post QEEG eyes closed documented a global (across all connections) average gain (compared to pre QEEG eyes closed) of +3.66 SD in beta2 coherence values, +1.84 SD in coherence beta1 values and +1.29 SD in coherence alpha values (eyes closed values). Similar improvements were seen in phase beta2 (+2.46 SD), phase beta1 (+.62) and phase alpha (+2.30 SD) (eyes closed). Under the listening to paragraphs condition the increased coherence beta2 values held (+1.66 SD increase from previous listening condition response pattern) while coherence beta1 increased +2.39 SD and coherence alpha +.92 SD. Global relative power changes showed an increase in delta (+1.33 SD), decrease in theta (-1.81 SD), no change in alpha (-.02 SD), decrease in beta1 (-1.04 SD), and small increase in beta2 (+.34 SD). The findings reflect the interventions' capacity to generalize to other frequencies not specifically addressed.

In the second case study subject, multiple protocols were employed, guided by the original evaluation, to reduce delta at T5 and P3 and increase coherence activity (alpha, beta1 (13-32 Hz), beta2 (32-64 Hz). A re-evaluation revealed global (across all locations and nine cognitive tasks) standard deviation reductions in delta microvolt measures (-.67 SD) and increases in global coherence values (alpha - +.47; beta1 - +1.29 SD; beta2 - +1.19 SD). The comparison involved the pre-treatment QEEG eyes closed with post treatment QEEG eyes closed.

Cost Issues

A cost-benefit analysis reported that for the 9,744 long-term disability claims over a 6-year period at Northwestern National Life, there was an average savings of $35 in disability reserves for every dollar spent on rehabilitation services (Cherek, L., & Taylor, M. (1995) It was also estimated that medical case management savings for NWNL increased from about $500,000 in 1987 to 8.1 million in 1993. The financial value, as well as the humanitarian value, of continuing to search for improvements in rehabilitation services is self-evident.

Conclusions

The AHRQ report (Chestnut et al., 1999) on the status of TBI rehabilitation treatment concluded that "The proper interpretation would be that, in the presence of a need for treatment and the absence of clearly superior alternatives, choices must be made between therapies without proven superiority over others based on clinical pragmatism" (p. 147). Given the results of the preceding analysis, there is tentative evidence that an alternative may presently exist – QEEG biofeedback. Further independent research initiatives are required with larger sample sizes, control groups and sham treatment groups to further explore the encouraging effectiveness of the QEEG biofeedback approach.

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Appendix A. Calculation of Effect Size

Effect size is a way of quantifying the size of the difference between two groups (Coe, 2002). It quantifies the effectiveness of a particular intervention relative to some comparison and answers the question of how well does the intervention work. An effect size of zero means that the mean scores of two groups are identical, while an effect size of 1 indicates that the mean scores of one group are superior to a second group by a value of one standard deviation. Some examples of other effect sizes show the overlap in the distributions of scores. An effect size of .20 indicates that the treatment moved a subject from the 50th percentile to the 58th percentile, while an effect size of .50 means that the subject is now performing at the 69th percentile, and an effect size of .80 means that the subject is now performing at the 79th percentile.

Olejnik and Algina (2000) describe the history of methods for calculating effects size. Cohen's effect size (1969), d, was the first commonly recognized effect size. It represented mean differences in units of common population standard deviation. Glass et al. (1981) proposed a modification of the Cohen d where the common standard deviation was replaced with the standard deviation of the control group. Hedges (1981) suggested that a better estimate of effect size would use the pooled variance and standard deviation rather than the standard deviation of one of the groups. There are also differences in the literature on which estimate of variance to use – typically it is the control group, which is the one you would expect to represent the population. Others argue for a pooled estimate when there is no control group but rather two treatment groups and the population variance is unknown. As indicated by Coe (2002), when using the pooled standard deviation to calculate the effect size, which generally gives a better estimate than the control group SD, it is slightly biased and gives a value slightly larger than the true population value. This bias is corrected using a formula (Hedges & Olkin, 1985, p. 80).

While Cohen (1988, p. 25) warned that he arbitrarily chose values to classify the interpretation of size of the effect, many studies continue to interpret an effect size of .2 as a small effect, a .5 as a medium effect, and a .8 is a large effect (Coe, 2002). The interpretation is improved by using confidence intervals that provide a range of values around the effect size to determine the likelihood of the effect size occurring due to chance. Greater accuracy of the effect size is more likely when based on a large sample rather than a small sample. If the confidence interval includes the value of zero, then the effect size is statistically equivalent to no effect. If the confidence interval does not include the value of zero, then the effect size is statistically significant.

In the effect size analysis of the interventions for TBI, we included research reports that provided the statistics necessary to obtain an effect size. These statistics included the means and standard deviations of the treatment and control groups. In the studies where there was no control group, then we used the means and standard deviations of the pre-treatment and post-treatment scores of the treatment group.

We provide an example of how we obtained the effect size and confidence intervals for three interventions that addressed memory. Kerner et al. (1985) treated 12 subjects with TBI using a memory retraining software and showed improved memory scores for the treatment group (M = 34.75, SD = 12.53) compared to 12 subjects in a control group (M = 30.42, SD = 11.41). The pooled standard deviation is 11.98. The effect size, using Hedge's bias correction for sample size, is .35 with a 95% confidence interval of -.46 to 1.16. Using Cohen's terms, the effect size of .35 is small to moderate. However, the confidence interval includes the value of zero, making the effect size not statistically different from zero. The conclusion, using the effect size and 95% confidence interval, is that the memory retraining software intervention is no different than the control group treatment.

In a second example, Schoenberger et al. (2001) treated 12 TBI subjects with 25 sessions of Flexyx Neurotherapy System. Immediate and delayed memory scores were obtained using the Rey's Auditory Verbal Learning Test (AVLT). Six subjects were treated first for five to six weeks while six were in a wait-list control group. Then the six subjects in the wait-list group received treatment. We can assess the effect size for the treatment by using pre- and post- treatment scores for the entire group of 12 subjects. There was no significant effect size for immediate memory score. The pre-treatment scores (M = 10.50, SD = 2.11) were no different than the post-treatment scores (M = 10.17, SD = 1.90), ES = -.16 with a 95% confidence interval of -.96 to .64. The authors reported a significant effect ($p < .10$) for treatment with a significant improvement in the delayed memory scores between pre-treatment (M = 9.67, SD = 2.39) and post-treatment scores (M = 11.08, SD =2.54); however the ES was .55 with a 95% confidence interval ranging from -.26 to 1.37.

In the third example, on data reported in this paper, 15 subjects with TBI were given QEEG treatment.

Their pre- and post-treatment scores were compared to a control group of 15 subjects. The TBI subjects improved their scores on paragraph recall from pre-treatment (M = 8.75, SD = 4.51) to post-treatment (M = 25.6, SD = 6.62), in addition the ES was 2.89 with a 95% confidence interval ranging from 1.87 to 3.92. The confidence interval does not include the value of zero. Clearly the treatment was effective.

Appendix B. Sample sizes and durations of interventions

Intervention	Reference	Number subjects	Number sessions
Computer	Kerner & Acker, 1985	12	12
	Gray & Robertson, 1992	31	17.5
	Ruff et al, 1994	15	20
	Park et al., 1999	23	20
	Niemann et al, 1990	29	36
Strategies	Ryan & Ruff, 1988	20	132
	Freeman et al., 1992	6	15
	Cicerone et al., 1996	20	6 months
	Novak et al., 1996	22	20
	Milders et al., 1998	13	12
	Fasotti et al., 2000	12	7.4
	Laatsch & Stress, 2000	16	Mean of 32
	Quemada et al., 2003	12	120
	Kaschel et al., 2002	12	30
	Stephens, 2006	10	20
	Salazar et al., 2000	120 67 in hospital treatment 53 home treatment	32
Medications	McDowell et al., 1994	24	Subjects tested twice - with placebo and with Bromocriptine
	Whyte et al., 1997	19 Ss completed some tasks 9 Ss completed all tasks	Subjects tested twice - with placebo and with Methylphenidate
	Leon-Carrion et al., 2000	10	Cytidinediphosphocholine for 3 months
	Fann et al., 2001	15	sertraline for 8 weeks
Eyes Closed QEEG			
	Tinius & Tinius, 2000	16	20
Standard QEEG			
	Stephens, 2006	6	20
Modified QEEG			
	Keller, 2001	12	10
	Schoenberg et al., 2001	12	25
Activation QEEG			
	Thornton & Carmody, 2005	7	80
	Thornton & Carmody, this article, paragraph recall	15	54