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REVIEW ARTICLE

Neurofeedback and traumatic brain injury: A literature review

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BACKGROUND: Neurofeedback is a form of biofeedback whereby a patient can learn to control measurements of brain activity such as those recorded by an electroencephalogram. It has been explored as a treatment for sequelae of traumatic brain injury, although the use of neurofeedback remains outside the realm of routine clinical practice.

METHODS: Google Scholar™ was used to find 22 examples of primary research. Measures of symptom improvement, neuropsychological testing, and changes in subjects' quantitative electroencephalogram were included in the analysis. A single reviewer classified each study according to a rubric devised by 2 societies dedicated to neurofeedback research.

RESULTS: All studies demonstrated positive findings, in that neurofeedback led to improvement in measures of impairment, whether subjective, objective, or both. However, placebo-controlled studies were lacking, some reports omitted important details, and study designs differed to the point where effect size could not be calculated quantitatively.

CONCLUSIONS: Neurofeedback is a promising treatment that warrants double-blind, placebo-controlled studies to determine its potential role in the treatment of traumatic brain injury. Clinicians can advise that some patients report improvement in a wide range of neuropsychiatric symptoms after undergoing neurofeedback, although the treatment remains experimental, with no standard methodology.

KEYWORDS: neurofeedback, traumatic brain injury, quantitative electroencephalogram

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INTRODUCTION

Traumatic brain injury (TBI) is a prevalent and serious problem. In the United States alone, 1.7 million TBIs result in 235,000 hospitalizations each year, and 3.2 million individuals live with a resulting disability.[1] Patients who have experienced a TBI are overrepresented as 5.5% of completed suicides.[2] In 2000, the estimated cost of TBI occurrence in the United States was $60.4 billion, even when excluding the costs of long-term care and reduced quality of life.[1] In the military, mild TBI (mTBI) is termed the "signature wound" of the conflicts in Iraq and Afghanistan, with an estimated 17% to 22% of returning soldiers having sustained a TBI while deployed.[3] Cognitive and functional rehabilitation, in addition to pharmacologic treatment of pain and neuropsychiatric symptoms, are the mainstays of treatment for patients with TBI.[4]

There are a number of reasons TBI is difficult to treat. It is a heterogeneous disorder, with different presentations depending on the nature of the injury.[5] Diffuse axonal injury has a probabilistic distribution that is difficult to detect with conventional methods.[5] Some patients make an apparent spontaneous complete recovery, while others have lingering nonspecific postconcussive symptoms.[6] Patients who do recover are thought to do so through the process of vicariation, whereby neurons lost via injury are effectively replaced by "redundant" neurons, but this process itself is difficult to measure.[7]

Neurofeedback is biofeedback, or operant conditioning, of any measure of brain functioning. We use the term *neurofeedback* to refer to the use of the electroencephalogram (EEG) to produce biofeedback, although the use of other measurements, such as cerebral blood oxygenation, are also possible.[8,9] Neurofeedback has led to symptom improvement for patients with a history of mTBI, but previous reviews have cited study limitations that necessitate further research.[10] This literature review was conducted to expand on those recommendations.

In the practice of neurofeedback, an auditory or visual cue is used to guide the patient toward a "healthy" EEG signal as defined by a sample of healthy subjects.[11] This behavior has not been found to correlate with any type of subjective thought process on the part of the patient,[12] although understanding of the paradigm and attention to the task are typically presumed prerequisites. Treatment usually is broken into 5 to 60 sessions, each lasting 30 to 60 minutes, depending on the patient's condition and response to treatment.[11] Treatment can be administered by a technician, and there are anecdotal reports that periodic encouragement during training can aid the patient's motivation, although a protocol for interaction with the patient has not been formalized.[11]

Double-blind, placebo-controlled studies have shown that neurofeedback can be effective for the treatment of refractory epilepsy,[13] attention-deficit/hyperactivity disorder,[14] and obsessive-compulsive disorder.[15] Side effects typically last a few hours after treatment and include headache, nausea, fatigue, dizziness, agitation, cognitive interference, and destabilization.[11]

Neurofeedback often is guided by the patient's quantitative electroencephalogram (QEEG), typically a Fourier transform of EEG data. This provides power spectral density measurements at each EEG channel, and measures of "coherence," or power density that correlates between 2 channels. Power and coherence measurements at each of 64 frequencies for 19 channels provide thousands of measurements that can be the target of biofeedback.[16] Targets often are chosen with the help of a normative database, built from the QEEGs of healthy subjects.[11]

Neurofeedback has not gained popularity in clinical practice. Lack of empirical evidence[10] and QEEG's lack of diagnostic specificity[16] are cited as factors contributing to its experimental status. It is a highly technical treatment, and making necessary adjustments during therapy remains an operator-dependent process.[11] As of 2013, neurofeedback devices are FDA approved as relaxation devices only, and treatment of any specific disorder is relegated to off-label use.[17]

While the keyword *neurofeedback* is standard in databases for medical publications,[18] therapies outside the definition of operant conditioning have adopted the term neurofeedback, reducing the precision of academic inquiry.[10] In keeping with biofeedback nomenclature, neurofeedback is operant conditioning of quantitative measurements of brain activity.[19] Neurofeedback utilizes neuronal circuits of reward-based learning, as has been demonstrated by functional magnetic resonance imaging.[20] This is in contrast to a number of entrainment programs, whereby an audiovisual[21,22] or electromagnetic[23,24] stimulus oscillates in a waveform that is similar to the patient's EEG. In these modalities, plastic changes are hypothesized to take place in response to altered physiological activity.[25]

One problem with neurofeedback is that its mechanism of action remains a topic of investigation. In the context of brain injury, a full understanding would require measurement of network dynamics before injury, after injury, and after treatment. The hypothesis of the first author (G.M.) is that neurofeedback uses reward-based learning to induce vicariation in patients for whom it does not occur spontaneously.

Vicariation often is discussed in the context of loss of motor or sensory function due to cerebrovascular accidents, and subsequent recovery of function through training,[26] because these functions are directly linked to observable behavior.[10] Use of an EEG makes some aspects of cognition directly observable, opening psychological skill sets to the process of vicariation. Although the training time is relatively short compared with motor vicariation tasks, lasting effects of EEG neurofeedback on neural circuitry have been demonstrated by QEEG,[13] transcranial magnetic stimulation,[27] and functional magnetic resonance imaging.[25] In contrast to some sensorimotor vicariation paradigms, where spatially distant cortical regions can resume the task of lost neurons,[26] EEG operant conditioning would necessarily rely on vicarious neural networks to be spatially similar to the pretrauma configuration.

Ayers was the first to report a positive effect of EEG neurofeedback for TBI-related symptoms.[28] He reported that 250 patients "were relieved of their post-concussive symptoms," including decreased energy, depression, irritability, photophobia, phonophobia, attention deficit, dizziness, headache, and short-term memory loss. QEEG reportedly normalized as well; however, no quantitative results were reported despite a large cohort.

The current review was conducted to assess the strength of the available published literature on the therapeutic efficacy of neurofeedback for TBI and provide recommendations for future research in this area. We used guidelines[29] issued jointly by the Association for Applied Psychophysiology and Biofeedback (AAPB)[30] and the International Society for Neurofeeback and Research (ISNR)[31] to classify neurofeedback studies (**TABLE 1**).

Due to the paucity of published literature, subjects with all levels of injury severity are included in this review. Any intervention that involves operant conditioning of the EEG is included. All comparisons, outcome reports, and study designs were considered.

TABLE 1
Classification rubric for levels of evidence

1	Anecdotal evidence
2	Uncontrolled case study
3	Historical control
4	Observational studies without randomization
5	Randomized wait-list or "intention to treat" controls
6	Within-subject and intrasubject replication designs
7	Single-blind, random assignment control design, either sham or active (behavioral, psychological, or pharmacologic) treatment controls
8	Double-blind control studies, sham or active controls, random assignment
9	Treatment equivalence or treatment superiority designs with placebo control
10	Other designs, eg, double dummy, Solomon four-group

Source: Reference 29.

METHODS

Google Scholar™ was chosen as a search engine, as it returned a higher number of relevant results than traditional clinical databases such as PubMed. The terms *neurofeedback* and *TBI* were used in the search. Google Scholar™ automatically included the terms *brain* and *injury* in the search process. Search results were restricted to work with human subjects. No outcome measures were excluded. In November 2012, this method returned 999 search results.

Of the 999 search results, 6 could not be found, 26 were not published in English, 3 were animal studies, and 647 were not primary sources of information. Of the remaining 317 articles, 202 studied a sample of subjects who had never had a TBI. Of the remaining 115 studies, 82 did not use neurofeedback.

Of the remaining 33 articles, 8 were duplicate reports, and 4 articles detailed a series of measurements on the same cohort of patients and were therefore treated as a single article for this review.[32-35] Of the remaining 22 articles, 8 were cohort studies, and 14 were either case studies or case series. The articles were then categorized according to strength of evidence.

RESULTS

No double-blind, randomized, placebo-controlled studies (level 8 evidence) were found in our search. The studies that did include control subjects used healthy volunteers or patients with TBI who received alternative therapy or were wait-listed. None of the studies were blinded. Two

TABLE 2
A summary of level 3 and 5 evidence

Level of evidence	Citation	Number of subjects	Description	Results
5	Tinius and Tinius, 2000[36]	16 NF patients and 15 healthy controls	Psychological and neuropsychological testing was performed before and after NF treatment; controls did not receive treatment	Broad improvement in NF group, significant after Bonferroni correction
5	Keller, 2001[37]	21 patients with a history of TBI	12 patients received 10 sessions of NF, 9 patients received computer attention training	Patients improved significantly relative to controls in measures of attention; patients showed increased time spent in beta rhythm during NF
3	Bounias et al, 2001[32-35]	27, grouped into 5 clusters	Patients were clustered based on symptoms, and response to NF was correlated with cluster type	More symptoms require more sessions; more sessions lead to greater improvement
3	Hoffman et al, 1996[39]	14 patients status post-mTBI from MVA	Unspecified	General improvement in symptoms, quality of life, and MicroCog™ assessment
3	Walker et al, 2002[40]	26 patients with a history of TBI	Coherence abnormalities on QEEG were corrected 1 by 1 until patients reported improvement	50% improvement or more by self-report in 88% of patients
3	Zelek, 2002[38]	10 patients with loss of consciousness of >30 minutes	QEEG and RBANS were given before and after 30 sessions of NF	RBANS improvement, with coherence abnormalities as opposed to power abnormalities predicting successful treatment
3	Rostami et al, 2011[41]	12 patients with a history of TBI	6 patients received NF and 6 were wait-listed controls	Statistically significant improvement in QEEG findings in the treatment group
3	Zorcec et al, 2011[42]	6 patients with a history of TBI	All 6 patients received NF training	Fewer perseverative errors in WCST; no reported change in the Stroop test

mTBI: mild traumatic brain injury; MVA: motor vehicle accident; NF: neurofeedback; RBANS: Repeatable Battery for the Assessment of Neuropsychological Status; QEEG: quantitative electroencephalogram; TBI: traumatic brain injury; WCST: Wisconsin Card Sorting Task.

articles met level 5 criteria.[36,37] Both studies with a control group also used randomization, so no studies were categorized as level 4. Six studies met criteria for level 3.[32-35,38-42] Ten were level 2, as case studies or case series.[43-52] Five were self-published, so they were treated as level 1.[28,53-56] Four publications were associated with Thomson Reuters Impact Factors of 3.333[38,50,57] and 3.455.[43]

TABLE 2 is a summary of the studies meeting criteria for levels 3 and 5 evidence. **TABLE 3** is a summary of levels 1 and 2. Below is a summary of findings from studies of levels 3 and 5.

Neuropsychological measures showed broad improvement as a result of treatment. Attention, impulse control, and processing speed, as measured by a continuous performance task, each demonstrated statistically significant improvement after Bonferroni correction, to a level of insignificant difference from healthy controls.[36] When compared with patient controls who received computer-based attention training, patients treated with neurofeedback improved on a combined measure of omission and commission errors in 1 of 3 tasks as well as processing speed in 1 of 3 tasks; both findings were statistically significant.[37] Measures of short-term memory improved, with inadequate study size for statistical comparison.[50] Set shifting, as demonstrated by the Wisconsin Card Sorting Task (WCST), showed clinically and statistically significant improvement.[36] The Repeatable Battery for the Assessment of Neuropsychological Status (RBANS) showed statistically significant cognitive improvement after treatment.[38] Results of MicroCog™ assessments were not reported.[39]

Patients reported improvement in a wide range of symptoms. Global measurements of impairment symptom scales showed significant improvement in multiple

TABLE 3
A summary of level 1 and 2 evidence

Level of evidence	Citation	Number of subjects	Description	Results
2	Bratic et al, 2006[43]	2 patients with mTBI	1 patient received slow cortical potential training, the other received alpha training	Both patients demonstrated normalized EEG and improvement in symptoms
2	Byers, 1995[44]	1 mild head injury	31 sessions of increasing 12 to 18 Hz, decreasing 4 to 7 Hz	Improvement in psychological tests and self-report of symptoms
2	Greuling et al, 1998[45]	1 severe head injury, loss of consciousness over 1 month	14 sessions of NF 2.5 years after injury	Improved QEEG, FIM, and symptom report by patient's family
2	Hammond, 2005[46]	2 with several mild injuries and 2 status post stroke	9 to 50 sessions	Improvement in a wide range of self-reported symptoms
2	Malkowicz and Martinez, 2009[47]	1 severe head injury, LOC 18 months with secondary seizure disorder	42 sessions	Dramatic improvement in seizures, sleep, and motor control
2	Nash, 2005[48]	1 patient status post MVA	24 sessions, decreasing abnormal alpha power and increasing phase synchrony	Normalization of QEEG, improved symptoms and improved IVA
2	Reddy et al, 2009[49]	1 mild head injury	20 sessions of 45 minutes each	Improvement in measures of learning and memory
2	Thornton, 2000[50]	2 head injuries and 2 healthy controls	NF based on QEEG collected during written task	Improved recall of spoken paragraph in all patients
2	Thornton, 2002[51]	4 head injuries and 1 healthy control	NF based on QEEG collected during written task	Improved recall of spoken paragraph in all patients
2	Wing, 2001[52]	1 open TBI as pedestrian in MVA with incidental cerebellar astrocytoma removed	20 sessions 7 years status post injury	Improvement in coordination
1	Ayers, 1987[28]	250 patients	24 sessions aimed to decrease 4 to 7 Hz, then reward 15 to 18 Hz	Qualitative symptomatic improvements reported
1	Ayers, 1991[53]	12 patients with TBI: 6 receiving NF and psychotherapy, and 6 receiving psychotherapy	24 sessions of 30 minutes each, decreasing 4 to 7 Hz and increasing 15 to 18 Hz	NF patients report symptomatic improvement vs psychotherapy patients
1	Castillo-Ruben et al, 2006[56]	20 patients an average of 5 years status post injury	Average of 43 sessions of 20 minutes each, goal to reduce theta and increase beta	Improvement in QEEG variables, no report of symptoms or psychological testing
1	Poettker and Wilson, 2005[55]	1 patient with open TBI	90 hours of neurofeedback treatment	Improvement in psychological tests
1	Surmeli 2007[56]	24 patients with mild TBI, median duration of 5 years	40 half-hour sessions attempting to normalize power spectrum and coherence measures as measured by QEEG	Significant improvements in MMPI, TOVA, Beck Depression Inventory, and symptom report

FIM: Functional Independence Measure; LOC: loss of consciousness; MMPI: Minnesota Multiphasic Personality Inventory; mTBI: mild traumatic brain injury; MVA: motor vehicle accident; NF: neurofeedback; RBANS: Repeatable Battery for the Assessment of Neuropsychological Status; QEEG: quantitative electroencephalogram; TBI: traumatic brain injury; TOVA: Test of Variables of Attention.

studies.[39,40] Remission of 59% to 37% of reported symptoms was achieved in another study, with success in a diverse set of symptom clusters, including motor, language, cognitive, conduct, substance abuse, and pain. Less success was found among patients who demonstrated a lack of insight and reported depressive, anxious, or posttraumatic symptoms.[32] As shown in **TABLE 3**, symptom remission was achieved for an array of symptoms in case reports.

Assessments of functioning are sparse. All patients in 1 study who were employed prior to injury returned to work following treatment, although premorbid level of functioning was not reported.[40]

QEEG changed after treatment to resemble healthy subjects more closely. One study found that all subjects normalized their measure of beta power, whether it was initially high or low; post hoc analysis showed that all subjects had learned to raise their beta power above resting mean.[37] Another study showed a decrease in the power of high and low (24 to 32 Hz and 3 to 7 Hz) ranges, with an increase in the middle (8 to 18) Hz range.[33] Another reported normalization of both power and coherence measurements, with coherence improvement being a better predictor of symptom remission.[38]

Study designs, inclusion criteria, treatment paradigms, and the type of outcome reported varied significantly. The details available for each study in levels 3 and 5 appear below.

The first reported use of normative QEEG to guide neurofeedback therapy was accomplished by Hoffman and colleagues.[39] They treated 14 patients with motor vehicle–related TBI. QEEG *z* scores were measured along with symptom reports and MicroCog™ computerized assessments,[58] before and after treatment. Patients were reported to have improved symptoms and quality of life. Some findings were omitted, as only the abstract was published.

Tinius and Tinius[36] treated patients with mTBI, defined by a loss of consciousness of <30 minutes and a stunned or dazed feeling at the time of injury, or posttraumatic amnesia lasting less than 24 hours. Treatment parameters were determined by both the QEEG and clinical symptoms. In subjects with increased theta (4 to 8 Hz), inhibition of this band was the goal. In subjects with decreased theta, parameters were set to increase the sensorimotor rhythm (11 to 14 Hz). Fifteen patients with mTBI received neurofeedback combined with computerized cognitive training.[59] Sixteen healthy controls also underwent repeated neuropsychological testing 8 weeks apart with no treatment. Ten of 12 neuropsychological measures showed significant gains for the treatment group, from a pretreatment level significantly worse than the control group to a posttreatment level indistinguishable from the control group.

In a series of 4 publications, Bounias et al[32-35] provided a systematic description of the evaluation and treatment of 27 patients, of whom 21 were traumatically injured. Their goals were to identify clinical predictors of improvement and to determine the number of neurofeedback sessions needed to reach symptom resolution. In the first article,[32] 27 patients were classified into 5 different symptom clusters based on 48 signs and symptoms. In the second article,[33] clinical, physiological, and QEEG data were correlated before and after extensive neurofeedback therapy. Neurofeedback treatment parameters were determined based on QEEG in the central region at Cz. In the third article,[34] blood pressure and fingertip temperature trended toward normal for both hypertensive and hypotensive patients. In the final article,[35] regression was calculated based on the initial symptom loading, the percentage improvement, and the number of treatment sessions needed to achieve maximal improvement. Across the 5 syndrome classes and symptom loadings, the study authors found an average of 83% improvement in symptoms and an average session number of 82. The improvement rates as a function of duration of treatment fit both a linear and hyperbolic model.

Attention deficits were targeted specifically in patients with TBI by Keller.[37] The treatment group consisted of 12 patients with TBI with a mean initial score on the Glasgow Coma Scale of 11.8 (range 7 to 12), who received positive feedback when power in the beta range (13 to 20 Hz) at Fz (frontal region) exceeded the baseline QEEG beta power. The control group consisted of 9 patients with TBI who received computerized cognitive training using COGPACK®[60] and Neurosoft[61] suites.

Coherence training to treat 26 patients with a history of TBI and posttraumatic symptoms lasting more than 3 months was first published by Walker et al.[40] The NeuroGuide database[62] in the baseline QEEG was used to choose coherence abnormalities to train. After every 5 sessions, symptoms were reassessed and a new electrode placement was determined. Therapy was terminated after patients reported a symptomatic improvement of >50% or until 40 sessions had been completed. A mean of 19 sessions were required to see improvement in self-report. Mean improvement was 72.7%, with >50% improvement in 88% of patients.

Zelek[38] treated 10 patients with TBI with loss of consciousness for >30 minutes. QEEG and RBANS[63] were measured before and after 30 sessions of neurofeedback therapy, the parameters of which were omitted in the published abstract. RBANS improved from 1.70 (SD, 1.34) to 12.30 (SD, 10.84) ($P < .01$). Zelek suggested that QEEG coherence abnormalities are a better predictor of improvement than are power abnormalities.

Preliminary data are being gathered by Zelek and colleagues (V. Zelek, PhD, written communication, January 2013) on another set of patients with moderate to severe TBI. Their team is finding that neurofeedback has a significant effect size as measured by RBANS. The effect is matched against a group of wait-listed control subjects, although the control group has a higher degree of impairment at baseline.

An abstract from Rostami and colleagues[41] described neurofeedback administered to 6 patients and 6 wait-listed control subjects. Wilcoxon analysis of QEEG showed significant change toward healthy controls in the treatment group. No neuropsychiatric symptoms were reported.

"Sensory motor rhythm," defined as high alpha (around 14 Hz) over the sensorimotor cortex, has been a focus of treatment in other disorders, including epilepsy.[13] Zorcec and colleagues[42] trained 6 patients with a history of TBI by encouraging the production of sensorimotor rhythm. A number of measures from the Stroop test and the WCST were assessed before and after treatment, with statistical significance emerging from the number of perseverative errors in the WCST.

CONCLUSIONS

All published data reported positive effects of neurofeedback in the improvement of both subjective reports and objective measures of neuropsychiatric symptoms of mild to moderate TBI. Although these findings are promising, there are shortcomings in the published literature. No standard protocol for treatment exists, and none of the published protocols have been compared with a sham-control group.

The patient population was heterogeneous, which affects any given study's generalizability. One solution is to define narrow inclusion criteria based on the mechanism and severity of injury, along with neuropsychiatric symptoms and initial QEEG or imaging findings. The other is to record these data within a much larger sample size to analyze the contribution of each factor.

A lack of standardized outcome measurements makes study replication and comparison among protocols difficult. The TBI Clinical Trials Network suggested a battery of measures consisting of: the Extended Glasgow Outcome Scale; the Controlled Oral Word Association Test; the Trail Making Test, Parts A and B; the California Verbal Learning Test–II; the Wechsler Adult Intelligence Scale–III Digit Span subtest; the Wechsler Adult Intelligence Scale–III Processing Speed Index; and the Stroop Color-Word Matching Test, Parts 1 and 2.[57]

These neuropsychological measurements are of special importance due to their resistance to the placebo effect; subjective reports of symptom improvement have been demonstrated in both real and sham neurofeedback.[13] Despite this, the studies that used objective neuropsychological means of measurement[36,37] still showed improvement at a level of both clinical and statistical significance, especially in the arenas of attention, impulse control, executive functioning, processing speed, and overall measures of cognition.

An additional question the literature addresses is how much improvement can be attributed to spontaneous recovery.[6] This issue was best addressed by the Keller study, which used a control group of subjects with a history of TBI, where there were statistically significant differences between groups.[37]

Neurofeedback remains a promising yet unproven treatment for traumatic brain injury, and it has been both promising and unproven for many years. All experiments in this review reported positive findings, and study designs ranged from case reports to well-designed prospective cohorts. Randomized, double-blind, placebo-control studies are feasible and needed before this therapy can be unequivocally recommended. If the existing published studies are generalized to even a segment of the TBI population, neurofeedback is a therapy that will be of great benefit to those patients. Though the field is not yet mature, the literature strongly suggests that this therapy warrants further investigation. ■

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