The Usefulness of Quantitative EEG (QEEG) and Neurotherapy in the Assessment and Treatment of Post-Concussion Syndrome

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ABSTRACT

Mild traumatic brain injury (TBI) is associated with damage to frontal, temporal and parietal lobes. Post-concussion syndrome has been used to describe a range of residual symptoms that persist 12 months or more after the injury, often despite a lack of evidence of brain abnormalities on MRI and CT scans. The core deficits of post-concussion syndrome are similar to those of ADHD and mood disorders, and sufferers often report memory, socialization problems and frequent headaches. While cognitive rehabilitation and psychological support are widely used, neither has been shown to be effective in redressing the core deficits of post-concussion syndrome. On the other hand, quantitative EEG has been shown to be highly sensitive (96%) in identifying post-concussion syndrome, and neurotherapy has been shown in a number of studies to be effective in significantly improving or redressing the symptoms of post-concussion syndrome, as well as improving similar symptoms in non-TBI patients.

INTRODUCTION

The differential movement between the brain and the skull when the head is dealt a sharp blow produces percussive, centripetal and shearing forces resulting in traumatic brain injuries (TBI). Although maximum injury is suffered at the point of impact, the frontal and temporal regions adjacent to the sphenoidal ridges have been shown to be consistently vulnerable to contusions regardless of the direction or the point of impact.[1] A "contre coup" due to a percussion wave traveling through the brain matter and impacting the skull diagonally opposite can cause further contusion, and shear forces at the boundary between white and grey matter can result in axonal shearing.[1]

Mild TBIs, characterized by high score (>12) on the Glasgow Coma Scale (GCS), short or no period of loss of consciousness (LOC), short post-traumatic amnesia (PTA) duration, brief or no hospital stay, are generally considered benign. Yet, a significant number of patients report persistent symptomatology for weeks or months[2] and some for years after injury.[3-17] The cluster of symptoms reported by these patients is referred to as the post-concussion syndrome.[4] Amongst the reported symptoms of post-concussion syndrome are: (a) attention deficits and difficulty sustaining mental effort, (b) fatigue and tiredness, (c) impulsivity, irritability, temper outbursts and changes in affect, (d) learning and memory problems, (e) impaired planning and problem solving, (f) inflexibility, concrete thinking and lack of initiative, (g) dissociation between thought and action, (h) communication difficulties, (i) socially inappropriate behaviors, (j) self-centeredness, lack of insight and poor self-awareness, (k) impaired balance and (l) headaches[6,15,18,19] and personality changes.[20,21]

The subjective nature of these complaints are at odds with negative medical findings and have often generated controversy as to whether post-concussion syndrome has an organic or psychological etiology.[4] However, over the past 30 years evidence for an organic etiology of post-concussion syndrome has accumulated through studies of cerebral blood flow, neuropsychological deficits, evoked potential recordings, PET, SPECT, MRI and quantitative EEG (QEEG).[22-30] The nature of concussive head injury has been extensively discussed and theoretical concepts have been formulated, which are supported by electrophysiological evidence.[31,32] QEEG is particularly suitable for the evaluation of post-concussion syndrome, as it is empirical, objective, nonintrusive and has been shown to be highly accurate in identifying and discriminating various neurophysiological patterns of brain dysfunction.[33]

Neurotherapy (EEG biofeedback), is an operant conditioning paradigm whereby patients are given contingent audio/visual rewards for producing specific patterns of brainwave activity. Since the 1960s, studies have shown that, through neurotherapy, patients can be taught to promote normal functioning in the brain by normalizing dysfunctional brainwave patterns characterized by excessive

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slow wave activity.[34-37] A more recent improvement in neurotherapy is in the use of QEEG to identify the specific brainwave patterns that need to be redressed.[38-40]

This paper examines research findings that provide evidence for the effectiveness of QEEG in the assessment of the underlying neurophysiology of post-concussion syndrome, and its use in the determination of appropriate neurotherapy protocols. Furthermore, this paper reports on findings that indicate that neurotherapy is effective in the treatment of post-concussion syndrome and disorders whose symptoms overlap with post-concussion syndrome.

QEEG IN THE ASSESSMENT OF POST-CONCUSSION SYNDROME

A search of the literature from the National Library of Medicine reveals that although the key word EEG returned in excess of 34,000 citations since 1990, most of these are concerned with QEEG methodology, and few involve only visual analysis of the EEG. The use of QEEG in clinical practice for the evaluation of a wide range of mental disorders including mild TBI has been extensively researched, evaluated and reviewed.[33,41-53] The QEEG can provide clinicians with better, more targeted treatment options by providing functional information on the underlying neurophysiology associated with symptoms of post-concussion syndrome.[1,33,39,53,54] Furthermore, in medico/legal disputes, QEEG can provide important empirical evidence of functional abnormalities that correlate with symptoms of post-concussion syndrome following traumatic brain injury.

The U.S. Supreme Court's 1993 Daubert criteria of the scientific method replaced the 70-year-old Frye standards of "general acceptance" for admissibility of evidence in the U.S. Federal Court. Subsequently, Thatcher showed that QEEG met the four factors of the Daubert criteria: (a) hypothesis testing, (b) estimates of error rates, (c) peer reviewed publication and (d) general acceptance (Daubert v. Merrell Dow Pharmaceuticals, 61 U.S.L.W 4805 (U.S. June 29, 1993)).[30] Thatcher also argued that the technical aspects of QEEG in measuring the effects of neurological and psychiatric dysfunction match the Supreme Court standards of "technical" and "other specialized" knowledge (General Electric Co v. Joiner, 1997; Kumho Tire Company, Ltd. v. Carmichael, 1999). QEEG "scientific," "technical" and "other specialized" knowledge meet the standards of the Supreme Court rulings, thereby supporting QEEG as an admissible and clinically valid method in the evaluation of the nature and severity of neuropsychiatric disorders.[55]

Fenton[4] reviewed two prospective studies from the United Kingdom that looked at the evolution and course of post-concussion syndrome using psychosocial, neuropsychiatric, QEEG and brainstem auditory evoked potential measures. Findings indicated that increases in theta power occurred immediately following injury, with resolution within 10 days in most cases. However, chronic symptoms of post-concussion syndrome were noted in 13% of patients and were associated with a high prevalence of brainstem dysfunction and residual slow wave activity in the QEEG, which appeared related to the intensity of early symptoms. In an earlier study, identifiable QEEG changes were found to persist for years following moderate to severe closed head injury, with these changes related to residual cognitive deficits.[56]

Wirsen and colleagues[57] confirmed with QEEG the pathological findings detected by conventional visual EEG inspection. However, they found that QEEG topographic mapping was superior at detecting some regional abnormalities. The QEEG showed that regional excess slow wave activity corresponded well with morphological lesions in most patients. Low modal frequency of QEEG correlated both with lesion volume and injury severity and with the behavioral outcome variables.

Standard or conventional visually read EEG and conventional magnetic resonance imaging (MRI) are not considered sensitive enough or reliable enough in their detection of differences between mild and moderate TBI, nor do they predict outcome and gradations of severity of traumatic injury very well.[1] In contrast, recent studies have shown that QEEG evaluations in the post-trauma period ranging from months to several years can predict the severity of the injury and, in some cases, long-term prognosis, even if the initial GCS, LOC, and PTA information are not available.[58] Studies by Trudeau and colleagues[59] have demonstrated the high accuracy of QEEG in identifying patients with blast concussions years after the event, and studies by Hoffman and colleagues[60] have shown similar accuracy in the detection of TBI using QEEG methods in outpatients.

QEEG, conventional EEG examination and neuroimaging were performed on 100 patients by Jerrett and Corsak.[61] QEEG topographic maps were abnormal in 78% of patients with stroke, 50% with TBI and 100% of those with space occupying lesions. Of patients with abnormal EEG maps 30% had either better or sole insult localization with QEEG than routine EEG or neuroimaging procedures. EEG mapping had no false positives in localizing abnormalities. The authors concluded that QEEG mapping provided better detection of low amplitude slow wave activity than routine EEG, and faithful correspondence with neuroimaging procedures in localization of lesions. In fact, QEEG at times distinguished abnormalities not immediately definable by CT/MRI.

In a retrospective clinical study Henry and colleagues[62] investigated the neuropsychological, physiological, and behavioral functioning of 32 adult outpatients up to 65 months following non-impact (whiplash) brain injury. They found that despite significant and persistent age-adjusted cognitive deficits, mostly in executive functioning and frequent complaints of problems with attention, behavioral control, sleep and sexuality, structural neuroimaging was

not sensitive in detecting brain pathology. However, QEEG was abnormal in all the participants evaluated, with evidence of frontocentral slowing and increased spike wave activity.

Thornton[63] used QEEG variables at 19 scalp locations and five frequency bands up to 64 Hz, recorded under eyes closed condition on 91 subjects. These consisted of 32 mild TBI (MTBI) with no LOC > 20 minutes, 7 with LOC> 20 minutes and 52 normals all over the age of 14. A high frequency (64Hz) discriminant function effectively identified 87% of the minimal LOC MTBI subjects across all time periods and 100% of subjects within 1 year of accident. The combination of the multiple discriminants resulted in 100% accuracy rate for the 39 brain injured subjects.

Thornton[64] later examined the QEEG variables of 85 MTBI and 56 normal subjects during three auditory memory tasks. The MTBI subjects exhibited consistent patterns of phase and coherences anomalies and increased relative power in the beta band, which were correlated with the differences in memory recall. Memory functioning was found to be positively correlated with phase and coherence (measures of brain connectivity) and negatively correlated with increased beta activity at specific locations. It is interesting to note that Thatcher et al found similar patterns of phase, coherence and beta power anomalies when comparing TBI patients with normal database controls.[58,65]

In order to examine the accuracy of QEEG discriminant functions, Thatcher and colleagues[65] obtained measures of QEEG from 608 mild TBI patients and 108 normal subjects matched for age. A discriminant function developed from 264 mild TBI patients and 83 age-matched controls yielded a discriminant classification accuracy of 94.8%. An independent cross-validation of the discriminant function using 130 mild TBI patients and 21 controls yielded a discriminant accuracy of 96.2% for the TBI patients and 90.5% for normals. A second independent cross-validation of the discriminant function was carried out using 51 patients, and measures of test-retest reliability were carried out using 93 patients. Results yielded classification accuracies between 77.8% and 92.3%. A third independent cross-validation using 70 mild TBI patients from a different location and with different QEEG recording equipment yielded a discriminant accuracy of 92.8%.

In order to determine the prognostic strength of QEEG discriminant functions on outcome 1 year after injury, Thatcher and colleagues[58] administered a comprehensive diagnostic evaluation to 162 TBI patients within 1 to 21 days after injury. Diagnostic evaluation consisted of 19 channel QEEG referenced to age-matched norms, brainstem auditory evoked potentials, CT scan, and GCS at time of admission (GCS-A) and at time of EEG test (GCS-T). Functional outcome at 1 year following injury was assessed using the Rappaport Disability Rating Scale (DRS). The predictive power of each diagnostic measure at one year following injury was assessed using stepwise discriminant analyses and multivariate regression analyses. The best single predictors of outcome in both the discriminant analyses and the regression analyses were EEG coherence and phase. Discriminant greater than 95.8% accuracy of prediction of survival outcome 1 year after injury was obtained.[66] A prospective QEEG study, using high frequency discriminant, effectively identified 87% of TBI subjects who had not suffered significant loss of consciousness, across all time periods after TBI and 100% of subjects within 1 year of TBI.[67]

QEEG studies correlate closely with MRI findings. In a study by Thatcher, Biver and colleagues[29] brain water proton 1H T2 relaxation times and QEEG coherence were obtained from two independent groups of closed head injured patients and a group of normal control subjects. Statistically significant intercorrelations were observed between relaxation times of the cortical grey and white matter and EEG coherence. MRI findings were consistent with clinical QEEG studies in which (a) white matter lesions were related to increased delta amplitude and (b) grey matter lesions were related to decreased alpha and beta frequency amplitude. Lengthened T2 relaxation times in both the cortical grey and white matter were correlated with decreased beta and alpha amplitude and increased delta amplitude and with diminished cognitive function. The authors interpreted these findings as suggestive of a biophysical correspondence between the impaired integrity of protein-lipid structures of the brain as measured by the MRI and the scalp-recorded QEEG in TBI.[28] These studies lend more support for the routine use of QEEG in the evaluation of post-concussion syndrome, as suggested in cross validation studies reported next.

Thatcher, North and colleagues[58] evaluated the effectiveness of QEEG in the determination of severity of post-concussion syndrome. They used emergency hospital admission records of 105 patients with mild to severe TBI, 15 days to 4 years after injury, to evaluate severity of injury based on ratings of Glasgow Coma Scale (GCS), Loss of Consciousness (LOC) and Post Traumatic Amnesia (PTA). Using 16 of the highest-loading QEEG variables on each factor that differed significantly between severe and mild TBI by univariate t-test, a multivariate discriminant function, discriminated between mild and severe TBI groups with an accuracy of 96.39%, sensitivity 95.45%, and specificity 97.44%. In addition, the QEEG discriminant score also identified intermediate severity in moderate TBI patients. The discriminant function was cross-validated in 503 Veteran Affairs patients with mild to severe TBI. The validity of the discriminant function as an index of severity of injury and a classifier of degrees of severity was confirmed on the basis of significant correlations between QEEG discriminant scores, emergency admission measures, and post-trauma neuropsychological test scores.

Studies reviewed so far attest to the high reliability and discriminant validity of QEEG discriminant functions in revealing patterns consistent with MTBI or post-concussion syndrome. These studies also highlight the high correspondence of QEEG with other neuroimaging methods and in some cases the superiority of QEEG in locating the source of anomalies, be it lesions or the excessive regional patterns of excessive slow wave activity associated with TBI or post-concussion syndrome. Coup contre-coup patterns can be commonly detected through topographic QEEG maps, and changes in EEG coherences and phase delays point to regional or global lack of connectivity brought about by focal or diffuse injuries. These studies lend support to the use of QEEG as an objective assessment to evaluate whether there is an organic basis to the symptoms reported by sufferers of post-concussion syndrome who are often dismissed as malingerers or as suffering from psychological, personality or somatization disorders.

COGNITIVE REHABILITATION FOR POST-CONCUSSION SYNDROME

Cognitive rehabilitation following TBI has been widely used, consisting of attempts with computer assisted tasks to restore cognitive skills, therapist-intensive interactions with cognitive therapy and counselling to help patients adapt to their deficits. While cognitive therapy may help provide psychological support and guidance, a search of the literature, by this author, found that there appears to be no theoretical concept for cognitive rehabilitation of post-concussion syndrome, nor is there evidence in the literature of the effectiveness of cognitive therapy in improving or redressing the symptoms of post-concussion syndrome (12 months or more after injury).

Cicerone and colleagues[68] conducted an intensive survey of the literature and graded the quality of the research evidence for cognitive rehabilitation following TBI. They concluded that there was evidence for the effectiveness of several forms of cognitive rehabilitation for persons with stroke and TBI and made specific recommendations for the remediation of attention, memory, functional communication, and executive functioning after TBI. However, their metaanalysis was based on 171 studies conducted mostly within the time period during which spontaneous improvements are known to occur, and did not appear to differentiate between acute and chronic TBI states associated with post-concussion syndrome. Other studies reviewed next have found no evidence to support the use of cognitive rehabilitation when spontaneous recovery is controlled.

A study by Ponsford and Kinsella[5,69] investigated a remedial intervention program for attention deficits following TBI. The study used a computer-mediated attention enhancement program with 10 severely injured subjects and consisted of a multiple baseline across subjects design to compare the intervention with intervention plus therapist feedback and reinforcement in separate training phases lasting 3 weeks. However, when spontaneous recovery and generalization to day to day activities were controlled for, no significant gains were demonstrated.

Gansler and McCaffrey[70] evaluated an intensive attention-remediation program based on Posner's 4-component model of attention for adult male TBI patients with chronic attention deficits. The hierarchically ordered attention-remediation program using an A-B-A single case design yielded no clinically significant improvement on attentional measures, neuropsychological variables, psychological characteristics, activities of daily living, or subjective ratings of changes in attentional abilities.

Salazar, Warden and colleagues[71,72] recently reported the results of a prospective controlled randomized trial comparing an intensive inpatient cognitive rehabilitation program to a home support program for 120 active-duty military personnel. All subjects had sustained a moderate-to-severe closed head injury (GCS <=13 or PTA 24 hours or more), or evidence of focal cerebral contusion or hemorrhage on CT scan or MRI. All patients received medical treatment as needed and were randomly assigned to each group: 67 were assigned to an intensive, standardized 8-week, in-hospital cognitive rehabilitation program and 53 to a limited home program, consisting of guidance on home activities and a weekly telephone call from a psychiatric nurse. At 1-year follow-up, there was no significant difference in outcome measures between patients in the two groups on measures of fitness for return to duty, cognitive, behavioral, or quality-of-life measures. The authors concluded that these findings emphasize the importance of conducting randomized trials to evaluate TBI rehabilitation interventions.

It could be argued that cognitive therapy requires that the attentional system, executive functions and memory be unimpaired. Hence, in post-concussion syndrome and ADHD, the dysfunctional frontal lobes may interfere with the effectiveness of cognitive therapy. In contrast, the use of operant conditioning of the EEG (neurotherapy) offers hope to significantly restore normal neuronal functioning and to restore the TBI-associated deficits towards pre-morbid levels.[1,38,39,60,73-75]

NEUROTHERAPY

Brainwave activity is believed to be the manifestation of the electrical activity of columns of cortical neurons driven by subcortical generators from the thalamus, hippocampus and septum[76] There is general consensus in neuroscience that thalamo-cortical oscillations are responsible for the initiation of timing and transfer of information between various structures in the brain.[77,78] According to this view, normal human neuronal activity, manifested in the EEG, is self-regulating. "Dysrhythmia" in thalamocortical oscillations is believed to arise from dysregulation in subcortical and cortico-cortical circuits that give rise to abnormal EEG rhythms, such as excesses or deficits in delta, theta and alpha or beta activity. Such dysregulation is believed to be

responsible for a range of psychiatric disorders,[40,77-80] and theoretical mechanisms and scientific rationale for the effectiveness of neurotherapy in redressing these "dysrhythmias" have been proposed,[40,79-81] but are outside the scope of this paper.

The core symptoms of post-concussion syndrome as previously discussed include attention deficit disorder, impulsivity, mood disorders, memory difficulties and headaches. Each of these are associated with identifiable neurophysiological brain patterns, mostly associated with various degrees of excessive slow wave (theta) activity.[1,33,47,58,65,82] Each of these core symptoms has been shown to respond positively to neurotherapy. Since excess theta is causally linked to reduced cerebral metabolism and brain blood flow (ischemic hypoxia), impaired neurotransmitter synthesis/release and various forms of brain pathology, inhibition of excess theta by any means, including neurotherapy, is likely to improve brain function.[83]

During neurotherapy, real-time QEEG is displayed on a computer in the form of a game. The game software is driven by selected QEEG parameters and the patient is given contingent audio-visual rewards in the game for producing less slow wave (theta 4-7Hz) and more fast waves (beta 16-20Hz). There is now significant evidence in the literature, which suggests that around 80% of ADHD patients can learn to produce a brainwave pattern with more normal theta/beta ratios[37,40,81,84-86] by this operant conditioning process. Evidence of the effectiveness of neurotherapy in reducing excess theta and redressing TBI associated deficits are reviewed next.

Neurotherapy Treatment of TBI and Post-concussion Syndrome

Hoffman and colleagues reviewed and reported on improvements in clinical symptoms of post-concussion syndrome using neurotherapy in a clinical setting.[60,73,87] They generally started treatment at least 6 months post injury, and reported that on average 40 neurotherapy sessions were required for rehabilitation, with control measures recorded every 5 sessions. Physiological and EEG measures were quantified and tracked within each session to monitor training protocol effectiveness. Hoffman et al[60] reported that in around 80% of cases TBI patients were able to achieve a minimum 70% improvement in symptoms. Good clinical results were also obtained with chronic patients years post-injury.

Ayres reviewed her work with 250 TBI patients, summarizing more than two decades of clinical work using real-time digital EEG biofeedback for cases of head trauma, coma, and stroke.[74,75] Ayres described specific protocols used over that period, training primarily to reduce excess theta activity at locations where that activity was the highest. She reported that as the EEG operant conditioning took effect, evidenced in reduction of theta/beta ratios, patients reported improvement in energy levels, concentration, memory, reduced sensitivity to light and sound, reduced incidence and severity of headaches, and reduction in positional vertigo.[74]

Schoenberger and colleagues[88] conducted evaluation of the potential efficacy of an EEG biofeedback system in the treatment of TBI using 12 patients 12 months after injury. All patients initially reported symptoms of post-concussion syndrome with substantial cognitive difficulties, which interfered with their day-to-day functioning. Participants were randomly assigned to an immediate treatment group or a wait-list control group and received 25 sessions of EEG biofeedback. All were assessed at pre-treatment, post-treatment, and follow-up with standardized neuropsychological and mood measures. Comparison of the two groups on outcome measures indicated improvement in subjective reports of depression, fatigue, and other problematic symptoms, including improvements in occupational and social functioning following neurotherapy. There were also significant improvements in some objective measures of cognitive functioning.

Head injured patients often report tinnitus, a debilitating disorder of central auditory processing whereby the patient continuously hears varying degrees of high pitch sounds. Gosepath et al[89] treated 40 patients with tinnitus with neurotherapy, training to reward alpha-activity and inhibit the beta-activity while relaxing and orientating to sounds or music. All patients had a significant reduction on the Gobel and Hiller tinnitus questionnaire, concurrent with increased alpha and reduced beta activity. A control-group of 15 persons without tinnitus didn't register any changes of alpha or beta activity during the same period.

In a recent European study, Bounias and colleagues[90] reported in a four-part study on a random group of 27 patients, from various hospital centers with vascular, traumatic or combined head injuries, treated with neurotherapy. In part one, Bounias described the use of a meticulously designed method of typological classification of clinical syndromes. Patients were assigned scores on seven categories of post-traumatic clinical symptoms: motor functions, language, cognitive functions, psychosocial disorders, pain related disorders, neuropsychiatric impairments and metabolic disorders. Statistical methods were used to construct patients' indices of membership to each category. In part two, the study reported on individual assessment pre- and post-neurotherapy, and on treatment outcome based on the symptom indices. Results indicated class-average rehabilitation rates ranging from 59% to 87%. For the most part, patients were required to reduce slow wave (delta/theta) activity and increase fast wave (beta) activity, wherever it was excessive in the QEEG. Improvements in symptomatology were related to improvements in the QEEG parameters that the patients were trained to improve.[91] In part three, Laibow et al reported that the patients also experienced significant improvements in car-

diac parameters and peripheral temperature in addition to targeted improvements in brain functions and symptoms.[92] In part four, Bounias and Laibow described an empirically derived relationship relating length of neurotherapy treatment to the initial load of clinical symptoms and the rate of rehabilitation.[93] The researchers concluded that neurotherapy can successfully treat patients with brain injury with highly meaningful clinical results.[91]

Keller[94] investigated the usefulness of neurotherapy in TBI rehabilitation during the spontaneous recovery phase. 12 patients with moderate TBI were given contingent audio and visual feedback for increasing amplitude and duration of beta activity in their EEG at Fz. A matched control group of 9 patients were given a standardised computerized attention training program not involving EEG biofeedback. The treatment group showed statistically significant changes in their EEG beta amplitudes and in increases in the ability to sustain beta output, while no systematic increases were observed in the control group. On attentional measures, patients in both groups improved in their ability to attend for short durations on a computer task. However, only the neurotherapy group improved in tasks of sustained attention and on pencil paper tasks.

Walker and colleagues[95] investigated whether QEEG guided coherence training was effective in remediating symptoms of post-concussion syndrome. Twenty-six patients with persistent symptoms were first evaluated with QEEG 3-70 months post injury. Neurotherapy protocols were designed to remedy abnormal QEEG coherence scores. Significant improvement (>50%) in symptoms was recorded in 88% of patients (mean = 72.7%). All patients were able to return to work following treatment. On average 19 sessions were required, which is half the average number of sessions required in most studies using amplitude training.

Attention Deficits Secondary to TBI and Neurotherapy

QEEG studies reviewed by Hughes and John[33] have found similar patterns of frontal deficits in TBI as in ADHD. Both exhibit prefrontal hypercoherence and excessive slow wave (theta and alpha) activity at frontal sites, and similar dysfunctional parietal patterns.

Using QEEG-derived discriminant functions, Monastra et al and Chabot and colleagues were able to discriminate replicably ADHD versus normal children, with a sensitivity of 90% and a specificity of 94%[96,97] and ADD versus specific learning disorders with a sensitivity of 97% and a specificity of 84.2%.[43,98-100] Their most common findings were of generalized or focal theta/alpha excess mostly at frontal and central sites. Monastra and colleagues found that the power ratio of theta/beta measured at the vertex (Cz) was able to distinguish their large sample of ADHD children from normals with a sensitivity of 86% and a specificity of 98%.[96,97]

Findings of attention deficits post TBI correlate with neuroimaging studies that indicate that the same areas suffer damage in TBI as in non-TBI subjects. For example, there are strong correlations between MRI and QEEG studies suggesting that the same areas are dysfunctional in TBI-acquired attention deficits and the neurodevelopmental ADHD population.[101] In order to explore this possibility, Max et al[102] found, in a prospective study, that increases in attention deficit disorder symptoms in the first 2 years after TBI were significantly related to the severity of TBI, and consistent with brain damage. Gerring and colleagues[101] found an excess prevalence of premorbid ADHD among children who presented with moderate and severe TBI. Children with high psychosocial adversity were more likely to develop ADHD after TBI. These studies suggest that ADHD and TBI have overlapping organicity and symptoms, with cumulative effects, with attention deficits and behavioral inhibition deficit being the major overlapping features.[101]

Recent studies have highlighted the degree and nature of functional similarities between ADHD and mild TBI. Using MRI and a set of multiple logistic regression models, Gerring and colleagues[22] determined that the odds of developing ADHD were 3.64 times higher among children with thalamus injury, and 3.15 times higher among children with basal ganglia injury, supporting an association between acquired ADHD and lesion in either or both the thalamus and basal ganglia. Chabot and colleagues[43] demonstrated using VARETA on QEEG data of children with ADHD that the excess theta observed in ADHD appears to be generated within the septal-hippocampal pathways of the basal ganglia, while excess alpha is generated in the thalamus. These findings support the view that patients with TBI-acquired ADHD symptoms share common underlying mechanisms with the ADHD population that may be redressed by neurotherapy as evidenced in studies and clinical reports of neurotherapy treatment of ADHD[103] and TBI.[1]

Tinius and Tinius[104] compared treatment effects of neurotherapy in adult male patients with mild TBI and other adult male patients with attention deficits to a waiting group control over an equivalent period. Psychological and neuropsychological testing was applied before and after the treatment period in both groups. After 20 treatment sessions, results indicated significant improvements in attention and response accuracy scores, and signifiant reduction in self-report of symptoms in both treatment groups compared to control. Errors in a problem-solving task improved only in the mild TBI patient group.

Neurotherapy in the Treatment of Mood Disorders

TBI is associated with frontal and temporal lobe dysfunction and asymmetries, which in turn have been associated with headaches, seizures, anger outbursts, mood instability and psychosis.[1,30,39,65,105-112] In particular, anxiety and depression have also been linked with excessive left

greater than right (L>R) alpha asymmetry in the frontal lobes, a pattern associated with a withdrawal and avoidance style of problem solving, negativity and pessimism.[113-117] On the other hand, R>L alpha asymmetry has been associated with mania.[118] Neurotherapy has been used successfully to redress these asymmetries and reverse the symptoms of depression.[119-125] A recent report of a 5-year follow-up of 5 severe chronic mood disorder patients, who had successfully undergone neurotherapy for depression using an asymmetry correction protocol, indicated that the treatment effects had been maintained over that period. Four of the patients were still off medication, and one on reduced medication.

Neurotherapy and Memory Rehabilitation

Rozelle and Budzynski[126] presented the case of a 55-yr-old male treated with neurotherapy for 6 months beginning approximately 1 year after a left-side cerebrovascular accident. The patient complained of hesitant speech with word finding difficulty and paraphasia, difficulty focusing his right eye, lack of balance and coordination, poor short-term memory, poor concentration, anxiety, depression, and tinnitus. EEG entrainment feedback was used followed by neurofeedback to inhibit theta (4-7 Hz) and increase beta (15-21 Hz) over sensorimotor and speech areas. At the conclusion of treatment there were significant reductions in slow-wave activity and theta/beta ratio. Speech fluency, word finding, balance and coordination, attention, and concentration improved. Depression, anxiety, and tinnitus were also greatly reduced.[126]

Thornton[127,128] used neurotherapy to remediate memory deficits by applying EEG biofeedback to QEEG correlates of memory function. An activation QEEG database was obtained with 59 right-handed subjects during two auditory memory tasks consisting of prose passages and word lists. QEEG recordings of clinical cases of memory dysfunction were compared to the normative database to determine their deviations from the values that predicted success for the reference group. Neurotherapy protocols attempted to normalize the values of the specific QEEG variables that were relatively deviant in the subjects. Thornton presented 3 case examples that indicated the successful use of neurotherapy in subjects with brain injury, with improvements ranging from 68% to 181% in relation to baseline measures as a result of the intervention.

Neurotherapy in the Treatment of Headaches

Headaches following TBI can be persistent and chronic in a substantial number of sufferers. A group of 100 children, 90 after brain concussion and 10 after contusion were observed for a period of 12 months. Of these, 83% reported headaches, 56% acute, and 27% chronic, mainly tension type headaches, and in 21% the headache persisted during the whole year of observation.[129] QEEG has been used to assess the underlying neurophysiology of headache and migraine sufferers, identifying specific patterns of excessive slow wave activity. The QEEG topographic maps of 100 patients with various types of headache (classic migraine, non-classic migraine, muscle contraction, mixed and post-traumatic) were compared to the topographic maps of 38 normal controls. Patients with migraine had 11 abnormal markers. Excess theta at O1 and excess alpha at O2 and T6 identified 82% of the headache patients.[130]

In an exploratory study, Siniatchkin et al[131] showed that EEG biofeedback training was accompanied by significant reduction of slow wave cortical excitability. This was likely to have been responsible for the clinical efficacy of the training; a significant reduction of days with migraine and other headache parameters was observed. It was suggested that normalization of the threshold regulation of cortical excitability during feedback training may account for clinical improvement. The use of EEG biofeedback training to suppress excess slow wave activity has been shown to reduce headache,[132] presumably by increasing blood flow. Excess theta wave activity is causally linked to reduced brain blood flow (ischemic hypoxia) and metabolism, impaired neurotransmitter biosynthesis, and numerous types of brain pathology.[83] Consequently, reduction of excessive theta activity by EEG biofeedback is likely to be associated with the exercising and normalization of self-regulatory mechanisms that lead the brain into more normal functioning and reduction of symptomatology.[79,80]

Reviews of the usefulness of various peripheral and EEG biofeedback modalities for a variety of symptoms, including biofeedback for headaches, were carried out by a number of authors. These studies have supported the use of biofeedback for headaches, and hightlight the need to use the technique most appropriate to the etiology.[133-138] For example, Glueck and Stroebel[137] reviewed the use of various types of biofeedback: EEG, temperature, Skin Conductance Response (SCR) and EMG and general relaxation techniques. They described specific biofeedback conditioning techniques for the treatment of patients with vascular headaches, muscle contraction headaches, and Raynaud's disease and suggested that the choice of suitable treatment must be carefully tailored to the needs of the individual patient.

EEG biofeedback was used to treat 13 patients, aged 18-68 yrs, suffering chronic post-traumatic headache and cognitive dysfunction. EEG biofeedback consisted of training to inhibit 4-7Hz theta activity and increase 15-18Hz beta activity. All patients reported improvement in anxiety, depression, and irritability, and those subjects who completed 30 sessions reported significant global improvement in headache and improved cognitive dysfunction.[132]

The effectiveness of EEG biofeedback in cases of tension headache was evaluated using pre- and post-measures of EEG-alpha, electromyography, galvanic skin response, a visual analog scale, an anxiety scale, and a

behavior disorder checklist. Twenty sessions of EEG biofeedback training were given to the experimental group, followed by post-assessment. Post-assessment for the control group was carried out after an equivalent treatment-free period. Results indicated a significant reduction of headaches in the experimental group and improvements in anxiety.[139] A double blinded study of biofeedback for headaches showed significant improvement in symptoms[140]

QEEG is particularly useful in the assessment of difficult cases of headaches. For example, Soriani and colleagues[141] reported a case of an 8-year-old boy with recurrent migraine auras without headache, precipitated by minor head trauma. Aura was characterized, besides other brain-stem signs, by confusional state. Soriani found an uncommon EEG pattern, characterized by diffuse continuous beta activity, recorded during the episodes, which suggested that recognition of this finding may prevent misdiagnosis and the inappropriate prescription of medication.

QEEG studies can help to identify the underlying neurophysiology associated with headaches, as different patterns suggest different treatment approaches or biofeedback protocols. For example, a pattern suggestive of cortical irritability may be more effectively treated through suppression of fast wave activity, while a pattern of excess slow wave activity would respond better to suppression of theta. The choice of biofeedback protocols and modalities should be individually determined based on assessment using well established principles of applied psychophysiology, which help determine whether the headaches have a vascular or muscular tension etioligy, or are due to specific brainwave patterns.

Further Research

A major review of the scientific literature and clinical use of neurotherapy was published in the January 2000 edition of Clinical Electroencephalography.[1] Authors reviewed Neurotherapy in Anxiety Disorders, including PTSD,[142] Attention Deficit Disorder,[103] Affective disorders[125] Seizure disorders[143] and post-concussion syndrome.[1] The editorial opinion by Neurology Editor, Frank Duffy, M.D., stated: "The literature, which lacks any negative study of substance, suggest that Neurotherapy should play a major therapeutic role in many difficult areas. In my opinion if any medication had demonstrated such a wide spectrum of efficacy it would be universally accepted and widely used."[144]

Duffy suggested that it would be desirable to have double blind placebo controlled studies of neurotherapy. However, in practice it is impossible to give sham feedback without the clinician knowing it, and patients quickly learn that the sham EEG is not their own when eye blinks and movements are not related to artifacts in the EEG. It may also be unethical to give sham feedback over 3 to 4 months to a vulnerable population on account of the possible harm to the client due to induced learned helplessness. Lubar reviewed his early work, conducted in the 70s, when ethics approval was more relaxed. In those studies he demonstrated in several ABA design controlled studies of Neurotherapy for ADHD that the treatment effects were reversible and contingent with the training and not a placebo effect.[34]

It is imperative to note that in the reviewed studies, improvement in symptoms correlated only with changes in the EEG features being trained, for example, reduction of theta/beta ratio in attention deficits and reversal of frontal alpha asymmetry in depression. Consequently, one could argue that neurotherapy treatment protocols should target dysfunctional brain patterns identified in the QEEG topography as they relate to the patient's difficulties. Choice of protocols can be made with more confidence when there is convergence between neuropsychological findings, deficits and abnormal Z scores in QEEG patterns compared to normative databases.

Meanwhile, more studies that control for spontaneous recovery and that compare treatment effects should be carried out in varied clinical settings with larger groups of subjects. Funding for such studies is likely to be difficult to obtain unless Government instrumentalities can become involved.

CONCLUSION

A number of controlled and uncontrolled studies and anecdotal reports reviewed in this paper suggest that neurotherapy may be an effective and cost efficient method of treating patients with mild traumatic brain injury. The length of treatment or number of sessions may be related to the initial severity of injury, and further studies may elucidate the most effective protocols for specific abnormal patterns seen in the QEEG of TBI patients. The neurotherapy studies, while not substantial in number, nonetheless stand in contrast to the nearly complete absence of published studies of the effectiveness of cognitive rehabilitation or of other therapies in the remediation of post-concussion syndrome. The relative efficacy of neurotherapy in the treatment of post-concussive syndrome warrants the continued use of this treatment modality. The role of quantitative EEG or QEEG to help guide the neurotherapist in choosing which EEG variables and locations to target for biofeedback has been proven to be of value in the literature as reviewed in this paper. No comparative studies of which EEG variables may be more or less effective have been published as of this date. Currently, all of the studies cited in this review share in common a quantitative and objective approach to variable selection.

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