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Diagnosis and Treatment of Head Injury

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Brain injury appears to affect from 132 to 367 people per 100,000. Traditional methods for diagnosing mild head injury, such as medical history, CAT scan and MRI, often show normal test results even though patients complain of significant neurocognitive dysfunctions. Robert Thatcher compiled a normative reference EEG database in 1979. The norms were replicated in several studies. These EEG databases allow a patient's EEG to be compared with a reference population. An emerging and promising treatment approach to mild head injury is the use of Quantitative EEG technology and EEG neurofeedback training.

Definitions

There are a number of definitions for mild traumatic brain injury most of which include some interval of consciousness and/or some period of posttraumatic amnesia. A commonly used definition is unconsciousness for less than twenty minutes, or no loss of consciousness with a Glasgow Coma Scale of 13 or above without deterioration and post traumatic amnesia less than 48 hours. The criteria of loss of consciousness and amnesia is controversial and several clinicians believe them to be of little value for many populations suffering neurocognitive symptoms (Hoffman, 1994).

Although traditionally mild traumatic brain injury has been associated with motor vehicle accidents and work related injuries like slip and falls, similar neurocognitive symptoms occur with subtle brain dysfunction resulting from a number of other sources, including silicone toxicity, environmental toxins, autoimmune disorders, multiple sclerosis, and even chemotherapy (Hoffman, et al., 1995; Ritchlin, et al., 1992).

Symptoms of mild traumatic brain injury are typically classified into three categories, which include physical/somatic problems, cognitive dysfunctions and emotional/personality difficulties. Specific physical/somatic problems may include headaches, dizziness, fatigue, sleep disturbance, sensitivity to light or noise, loss of sexual drive, nausea, blurred vision, vomiting, tinnitus and seizures. Cognitive problems feature impaired attention and concentration, slow speed of processing information, impaired complex thinking, problems organizing, distractibility, short term memory problems, forgetfulness, trouble finding the right word, word substitutions and word reversal. Emotional/personality symptoms may consist of low frustration tolerance, irritability, mood lability, anxiety, depression, impatience, anger, explosive temper and feeling overloaded by too much stimulation.

Epidemiology

Brain injury appears to occur at a frequency ranging from 132 to 367 per 100,000 population. The variability of these figures lies in the different criteria for diagnosis used in studies (Silver, et al., 1994). It is estimated that almost 2 million head injuries of non-fatal brain injuries occur per year. People 15 to 24 years of age are at highest risk, with a decline in risk occurring during the middle age years and an increase after age 60. Men have a 2-2.8 times higher frequency than women. Mild brain injuries, those with a Glasgow Coma Index scale of 13-15 are by far the greatest proportion. Eighty percent appear to be mild, 10% moderate (Glasgow Coma Score 9-12), and 10% severe (Glasgow Coma Score 8 or less). Slightly less than half of brain injuries are concussions without fracture of the skull. Other causes are hemorrhage, contusion and laceration. In mild brain injury 35% of reported cases demonstrate no loss of consciousness, although this percent may be much higher since often patients are not diagnosed with mild head injury unless they have loss of consciousness or post-traumatic amnesia.

Current Diagnosis of Mild Traumatic Brain Injury

Mild traumatic brain injury causes a great deal of angst for the diagnostic clinician as well as the legal system. Traditional methods of evaluation consist of history, neurologic exam, CAT Scan, MRI, standard EEG, and neuropsychological testing. Although patients complain of significant neurocognitive dysfunctions, not uncommonly most of these standard tests are normal.

Neuropsychological testing appears to be the current mainstay in recognizing cognitive functioning, however, there are several problems with relying heavily on this as a diagnostic tool for mild traumatic brain injury. Interpretation of neuropsychological tests has considerable variability and subjectivity, which currently causes the legal system concern, since two opposing expert neuropsychologists often differ in their interpretations. As many as 50% of subtle injuries are not picked up through neuropsychological testing (Posthuma, 1988). Many neuropsychologic tests have a low ceiling in which pre-morbidly high-functioning individuals can still score in the normal range, thus not being sensitive enough to measure a decrease in functioning. The low ceiling also may account for inaccuracies or inability to pick up more mild injuries.

SPECT scan, which records cerebral blood profusion that may be decreased in damaged areas, is demonstrating usefulness in the head injured population and, in the future, may prove a valuable diagnostic tool (Gray, et al., 1992; Ichise, et al., 1994).

Mechanism of Injury of Mild Traumatic Brain Injury

Most mild traumatic brain injuries are caused by some form of mechanical forces such as slip and fall, or motor vehicle accidents. Typically there is a rapid acceleration/deceleration effect on the brain as well as rotational and twisting forces around the brain stem. A classical injury would demonstrate contusions of the brain by the bony structures of the skull which occur in the frontal and frontal-temporal lobes. These include the orbital plate of the frontal bone, the sphenoidal ridge, the petrous portion of the temporal bone, and the sharp edges of the falces. The forces also

account for a shearing effect, known as diffuse axonal injury, in which the myelin, or insulation of the nerves in the white matter, begins to unravel, making the fidelity of the electrical signal less than optimal. Secondary complications also develop as a result of metabolic changes. In the absence of diffuse axonal injury one might also expect to see focal abnormalities or a coup-contra-coup effect in which both the injured site and 180 degrees opposite the injured site are both affected when the forces cause the brain to bounce against the skull along the trajectory of the point of impact.

Advances in Diagnosis of Mild Traumatic Brain Injury

In March 1979, Robert Thatcher, Ph.D. began work on a normative or reference EEG database. Seven hundred and twenty three subjects were tested from 1979 to 1985. Of these, 511 met the criteria for ' normalcy and were included in the database. The criteria included an uneventful prenatal, perinatal, or postnatal period, no disorders of consciousness, no head injury with cerebral symptoms, no history of central nervous disease, no seizures, and no abnormal deviations with regard to mental and physical development. Additional criteria for entry were a full scale I.Q. greater than 70, a WRAT-R school achievement score greater than 89 on at least two subjects, and a grade point average of C or better in major academic classes. Both off-line and on-line artifact rejection was utilized. The age distribution consisted of a developmental period from two months through adulthood. More subjects were skewed toward the younger ages since development is most rapid in children. Demographically there was a mixture of males and females, different ethnic backgrounds and socioeconomic status representative of the North American population. The normative database provides a statistical analysis in which a patient's EEG can be expressed as a deviation from the normative group in Z score units, i.e., standard deviations from the mean. The EEG norms were replicated in several places (Matousek and Petersen, 1973; Thatcher, 1980; Epstein, 1980; Hudspeth, 1985; and Hudspeth and Pribram, 1990 & 1991). The advent of such databases allows a patient's EEG, when digitalized (quantitative EEG, QEEG or computer enhanced EEG), to be compared with a reference population.

If a statistically significant number of Z scores greater than 2 appears when comparing the patient's EEG with the reference database, and the pattern forms around an anatomical location, pathology can be assumed when correlated with clinical history and presentation. This technique, for the first time, aids in the examination of subtle and even microscopic changes which previously went undetected by standard methods of evaluation. In 1989, Thatcher and his colleagues developed a discriminant analysis for mechanical head injury and diffuse axonal injury for the mild head trauma population which identifies a particular pattern of pathology EEG power spectral analysis was obtained on 608 mild head trauma patients and 108 age matched normal subjects. Three classes of neurophysiologic variables were attributed to mechanical head injury. These consisted of increased coherence and decreased phase in the frontal and frontal-temporal regions, decreased power differences between anterior and posterior cortical re-ions, and-reduced alpha power in posterior cortical regions (Thatcher, et al., 1989).

Coherence measures commonality between two different anatomical locations, that is, how they share activity or energy in the brain. It demonstrates how synchronously the brain is interacting

with itself. Since the human brain is multifaceted and able to multitask, coherence has a normative value. Mild head injury frequently causes increased coherence, resulting in the brain digressing to a more primitive level by acting more uniformly. This makes multi-tasking difficult and slows mental processing. Much like the fuzziness on a television screen when the cable insulation is damaged, the fidelity of the brain's electrical circuitry presents the patient with a foggy, unclear pattern. Some lesions actually cause neuronal uncoupling, in which case coherence would decrease.

Phase is the time delay between two points of the brain. With loss of myelin integrity there is more "noise" to the signal. According to Thatcher, clinically this may be seen in such symptoms as difficulty focusing or holding a thought. This finding can be responsible for some patients' complaints of forgetfulness and short term memory problems since the amount of time one can track a thought helps determine if it gets imprinted and, thus, is retrievable later as a memory.

In summary, QEEG, through the use of a digitized signal, allows the opportunity to compare the patient's brain with a reference database, and then further submit it to the multi-variant regression analysis which discriminates for mechanical head injury and, in part, diffuse axonal injury. Utilizing QEEG in the assessment of mild traumatic brain injury is beginning to appear in professional literature as an additional testing standard (Taylor and Price, 1994; Rumpl, 1993; McAllister, 1994; Packard and Ham, 1994; Duffy, 1994).

Treatment of Mild Traumatic Brain Injury

Traditionally, treatment for mild closed head injuries has included early intervention, education, family and professional support, group therapy, individual psychotherapy, marital and family therapy, stress reduction, coping skills training, multidisciplinary pain management, neuropsychological rehabilitation, vocational counseling, work skills training and multidisciplinary psychological rehabilitation (Howard, 1993).

Treatment has focused on medical management, individual counseling and psychotherapy, coping skills, pain management, vocational rehabilitation and cognitive rehabilitation. These treatments have been provided by a collection of professionals including physicians, psychologists, social workers, speech therapists, occupational therapists, physical therapists, chiropractors, massage therapists and biofeedback therapists, to mention a few.

Approximately two-thirds of mildly injured patients will regain 80% of their functioning within the first six months of recovery and continue to improve over the next one and one half years. Treatment therefore, should be relegated to those patients who are not improving, whose improvement has reached a plateau, or who after six months still have significant dysfunction. The literature is controversial in terms of the role cognitive rehabilitation plays in mild traumatic brain injury (Rattok and Ross, 1994). Clearly, many patients develop compensatory strategies for their neuro-functioning.

Cognitive rehabilitation is often divided into two general levels: functional and generic. The functional level trains abilities necessary for practical functioning, such as activities of daily

living. The second level trains generic cognitive skills such as attention, memory, and problem solving. While it is clear that moderate or severe injuries respond to this type of intervention, the literature is mixed on its effect in the mildly injured patient. In research performed by Niemann, et al., (1990) results of cognitive rehabilitation were equivocal. One reason may be that the neuropsychological batteries used were not sensitive enough to detect small changes in specific cognitive functions. Other treatments were also ongoing at the time, as it is almost impossible to provide cognitive rehabilitation in isolation of other forms of therapy. In general, traumatic brain injured patients can receive up to two to three hundred hours of treatment, which varies with the severity of the injury.

An emerging and promising treatment approach is the use of quantitative EEG technology and EEG neurofeedback training (neurotraining) for the treatment of mild traumatic brain injury. Since the end of the 1960's, Barry Sterman demonstrated the ability to control brain wave rhythms through operant conditioning and biofeedback techniques in the epileptic population through suppression of SMR (12-14 Hz) (Sterman and Wyrwicka,1967; Sterman, Wyrwicka and Roth, 1969; Wyrwicka and Sterman, 1968; Sterman and Friar, 1971; Sterman, Macdonald, and Stone, 1974; Sterman and Macdonald, 1978; Sterman and Shouse, 1980). Joel Lubar and Sterman then continued this work, discovering that patients demonstrated increased attentiveness, focus and concentration during feedback. Lubar went on to find that improvements in distractibility and attentional gains in academic settings resulted in increased school performance and grades (Lubar and Shouse, 1976a, b; J. O. Lubar and J. F. Lubar, 1984).

Since the early 1980's there have been reports of clinicians treating the symptoms of mild traumatic brain injury with EEG neurofeedback. Many approaches have been utilized. Bruner (1989) described using alpha training in three cases, demonstrating a return to pre-morbid functioning. It should be noted that his definition of alpha was 10-14 hertz which spread between what normally would be considered high alpha and SMR. Tansey (1983, 1991) and others have used SMR, or more precisely 14 hertz reinforcement in their treatments. Both referential and bi-polar placements have been reported.

Ayers has probably done the most work with the head injured population by inhibiting theta (4-7 hertz) and enhancing 15-18 hertz beta. By looking at a digitally filtered raw wave of theta, she may have identified high peaks of voltage that occur in some forms of brain damage. While these findings are often seen in the sensory motor strip, she trains wherever the damage is most prominent and where the neuroanatomy fits the deficits. This work centers around the concept of normalizing the EEG.

It should be noted that most of these treatment approaches have one thing in common; the reduction or inhibition of 4-7 hertz theta.

The most common question asked about using EEG neurofeedback for the treatment of mild traumatic brain injury centers around the training protocol. The authors' view of the use of this technology for the treatment of mild traumatic brain injury is that there is no one protocol. We advocate designing the treatment protocol based on the full diagnostic workup described above. The purpose of the training is to normalize the EEG. We find that this training tends to correlate

with a resolution of symptoms. As a result, a number of different training protocols have been used, as mentioned above, including beta enhancement with theta suppression, SMR enhancement with theta suppression, alpha enhancement with beta suppression, alpha enhancement with theta suppression, and ipsilateral as well as contralateral bandwidth or coherence training.

To know where to begin with the EEG neurofeedback training, a diagnostic evaluation is needed. The evaluation includes a comprehensive history, mental status examination, review of past medical records, a medical evaluation, neuropsychological screening, open and direct communication with all professionals dealing with the patient, and a computer enhanced EEG assessment including discriminate analysis, reference database comparison and spectral analysis. The purpose of this evaluation is twofold. First, it establishes the statistical likelihood of either diffuse axonal injury and focal injuries from contusion or coup-contra-coup effects. Additionally, this evaluation process establishes an appropriate baseline and designs a treatment protocol for the EEG neurofeedback training.

Our approach to the treatment of mild traumatic injury is multi-modal and has a strong educational orientation. The average number of training sessions for the head injury rehabilitation work using EEG neurofeedback is 40, significantly less than other traditional types of cognitive rehabilitation. As part of the training, a patient's physical/somatic, cognitive, and emotional/personality symptoms are reevaluated every five training sessions. The presence of physiologic trending of the data in the desired directions is also quantified and tracked each session. Patients come in for a one hour neurofeedback session at least twice weekly. It is not uncommon to see an improvement in some symptoms in as few as five sessions. Our clinical experience in using this treatment is that 80% of patients learn neuronal control and attain a minimum of 70% improvement in self reported symptoms. Generally we do not begin treatment until at least six months post injury. We have also attained good clinical results with patients several years after injury.

In the field of brain injury diagnostic and rehabilitation work, the use of EEG based technology for evaluation and treatment demands further attention and research.

Moderate to Severe Head Injury

As mentioned earlier, it was Sterman's work that demonstrated that patients with seizures unresponsive to medication have done well with EEG neurofeedback and which began the field of self-regulation and control of EEG. By extrapolating this and Lubar's discoveries with attentional- problems, as well as the work done with mild traumatic head injury through Ayers' contributions, it appears that with the use of the right equipment and technology, treatment of moderate to severe head injuries is also possible. According to Ayers, when combining EEG neurofeedback with traditional rehabilitation such as physical and occupational therapy, patients suffering from stroke, cerebral palsy, major trauma, coma, and even persistent vegetative states improve dramatically. Thatcher (1991) has written about the ability to predict outcome of head injured patients, demonstrating a 85% degree of accuracy in predicting the level of a patient's

functioning one year after trauma.

Conclusions

The diagnostic use of QEEG in mild traumatic brain injury is growing in popularity and usefulness, the advancements of which are acknowledged in recent professional literature. Treatment for this population, as well as the more severely injured patients, requires more traditional scientific studies to continue demonstrating its effectiveness. It may ultimately promise the shortest, most cost-effective treatment method, offering the greatest improvement of any cognitive rehabilitation to date.

References

Ayers, M. (1981). A report on the study of the utilization of electroencephalography (neuroanalyzer) for the treatment of cerebral vascular lesion syndromes. In C. Taylor, M. Ayers & Tom (Eds.) *Electromyometric Biofeedback Therapy,* Chapter VII. Biofeedback and Advanced Therapy Institute.

Ayers, M. (1983). Electroencephalographic feedback and head trauma. *Head and Neck Trauma: The Latest Information and Perspectives on Patients with a Less Than Optimal Recovery.* U.C.L.A. Neuropsychiatric Institute.

Ayers, M. (1993). A controlled study of EEG neurofeedback training and clinical psychotherapy for right hemispheric closed head injury. *Biofeedback and Self-Regulation,* 18(3), Sept.

Bruner, R. (1989). *Treatment of post concussion syndrome with alpha EEG biofeedback training, three case studies.* Presented at The Pennsylvania Society of Behavioral Medicine and Biofeedback.

Duffy, F., Hughes, J., Miranda, F., Bernad, P., Cook, P. (1994). Status of quantitative EEG (QEEG) in clinical practice. *Clinical Electroencephalography,* 25(4), VI-XXII.

Epstein, H. (1980). EEG developmental stages. *Developmental Psychobiologv,* 13, 629631.

Gray, B., Ichise, M., Chung, D., Kirsh, J., Franks, W. (1992). Technetium-99HMPAO SPECT in the evaluation of patients with a remote history of traumatic brain injury: a comparison with X-ray computed tomography. *Journal of Nuclear Medicine,* 33(l), 52-58.

Hoffman, D. (1994). Subtypes of post concussional disorder. *Journal of Neurapsychiatry and Clinical Neurosciences, 6* (3), 332-333.

Hoffman, D., Stockdale, S., Hicks, L., Schwaninger, J. (In press). Neurocognitive symptoms and quantitative EEG results in women presenting with silicone induced autoimmune disorder. *International Journal of Occupational Medicine and Toxicology.*

Howard, M. E. (submitted 1993). Mild brain injury: causes, damages, diagnosis, and treatment. In *Damages in Tort Actions.* Matthew Bender and Sons.

Hudspeth, W. (1985). Developmental neuropsychology: functional implications of quantitative EEG maturation. *Journal of Clinical and Experimental Neuropsychology, 7,* 606.

Hudspeth, W., Pribram, K-, (1990). Stages of brain and cognitive maturation. *Journal of Educational Psychology,* 82, 881-884.

Hudspeth, W., Pribram, K. (1991). Physiological indices of cerebral maturation. *International Journal of Psychophysiology,* 12, 19-29.

Ichise, M., Chung, D., Wang, P., Wortzman, G., Gray, B., Franks, W. (1994). Technetium99-HMPAO SPECT, CT and MRI in the evaluation of patients with chronic traumatic brain injury: a correlation with neuropsychological performance. *Journal of Nuclear Medicine,* 35(2), 217-225.

Johnstone, J., Thatcher, R. (1991). Quantitative EEG analysis and rehabilitation issues in mild traumatic brain injury. *Journal of Insurance Medicine,* 23(4), 228232.

Lubar, J. F., Shouse, M. N. (1976a). EEG and behavioral changes in a hyperkinetic child concurrent with training of the sensorimotor rhythm (SMR): a preliminary report. *Biofeedback and Self-Regulation,* 3, 295-306.

Lubar, J. F., Shouse, M. N. (1976b). Use of biofeedback in the treatment of seizure disorders and hyperactivity. *Advances in Clinical Child Psychology, 1,* 203-265.

Lubar, J. 0., Lubar, J. F. (1984). Electroencephalographic biofeedback of SMR and beta for treatment of attention deficit disorder. *Biofeedback and Self-Regulation, 9(l),* 1-23.

Matousek, M., Petersen, L. (1973). Frequency analysis of the EEG background activity by means of age dependent EEG quotients. In Kellaway and Petersen (Eds.), *Automation of Clinical Electroencephalography,* New York: Raven Press, New York.

McAllister, T (1994). Mild traumatic brain injury and the post concussive syndrome." Inj. Silver, S. Yudofsky, R. Hales (Eds.), *The Neuropsychiatry of Traumatic Brain Injury (pp* 357-392), Washington D.C.: American Psychiatric Press.

Niemann, H., Ruff, R., Baser, C. (1990).Computer-assisted attention retraining in head-injured individuals: a control efficacy study of an outpatient program. *Journal of Consulting Clinical Psychology,* 58, 811-817.

Packard, R., Ham, L. (1994). Promising techniques in the assessment of mild head injury. *Seminars in Neurology,* 14(l), 74-83.

Posthuma, A., Wild, U. (1988). Use of neuropsychological testing in mild traumatic head injuries. *Cognitive Rehabilitation,* March/April, 22-24.

Rattok, J., Ross, B. (1994). Cognitive Rehabilitation. Inj. Silver, S. Yudofsky, R * Hales (Eds.), *The Neuropsychiatry Of Traumatic Brain Injury* (pp 703-729), Washington D.C.: American Psychiatric Press.

Ritchlin, C., Charabot, R., Alper K, Buyon, M., Belmont, H.M., Roubey, R., Abramson, S. (1992). Quantitative encephalography: a new approach to diagnosis of cerebral dysfunction and systemic lupus erythemato*sus. Arthritis and Rheumatism,* 35(11), 13301342.

Rumpl, E. (1993). Craniocerebral trauma. In F. Niedermeyer, L. Da Silva (Eds.), *Electroencephalog7-aphy,* (pp 383-403) Baltimore: Williams & Wilkins.

Sterman, M.B., Wyrwicka, W. (1967). EEG correlates of sleep: evidence for separate forebrain substrates. *Brain Research, 6,* 143-163.

Sterman, M. B., Macdonald, L. R., Stone, R. K. (1974). Biofeedback training of the sensorimotor electroencephalogram rhythm in man: effects on epilepsy. *Epilepsia,* 15, 395416.

Sterman, M. B., Macdonald, L. R. (1978). Effects of central EEG feedback training on incidence of poorly controlled seizures." *Epilepsia,* 19, 207-222.

Sterman, M. B., Wyrwicka, W., Roth, S.R. (1969). Electrophysiological correlates and neural substrates of alimentary behavior in the cat. *Annals of New York Academy of Science,* 157, 723-739.

Sterman, M. B., Friar, L. (1972). Suppression of seizures in an epileptic following sensorimotor training. *Electroencephalography and Clinical Neurophysiology,* 33, 89-95.

Sterman, M. B., Shouse, M. N.(1980).Quantitative analysis of training, sleep EEG and clinical response to EEG operant conditioning in epileptics. *Electroencephalography and Clinical Neurophysiology,* 49, 558-576.

Tansey, M., Bruner, R. (1983). EMG and EEG biofeedback in the treatment of a 10-year old hyperactive boy with a developmental reading disorder. *Biofeedback and Self Regulation,* 8, 25-37.

Tansey, M. (1991). Wechsler (WISC-R) changes following treatment of learning disabilities via EEG biofeedback training in a private practice setting. *Australian Journal of Psychology,* 43 (3), 147-153.

Taylor, C., Price, T. (1994). Neuropsychiatric Assessment. In J. Silver, S. Yudofsky, R. Hales (Eds.), *The Neuropsychiatry of Traumatic Brain Injury* (pp 81-160), Washington D.C.: American Psychiatric Press.

Thatcher, R. (1980). Neurolinguistics: theoretical and evolutionary perspectives. *Brain and Language, 11,* 235-2 60.

Thatcher, R. (1987). Normative EEG database. Personal Communication Copyright.

Thatcher, R., Walker, R., Gerson, I., Geisler, F.,(1989). EEG: discriminant analysis of mild head trauma. *Electroencephalography and Clinical Neurophysiology*, 73, 94-106.

Thatcher, R., Cantor, D., McCalaster, R., Geisler, F., Krause, P. (1991).Comprehensive predictions of outcome in closed head injured patients: the development of prognostic equations. *Annals of New York Academy of Sciences, 620, 82-101*.

Wyrwicka, W., Sterman, M. B. *(1968).* Instrumental conditioning of sensorimotor cortex EEG spindles in the waking cat." *Physiology and Behavior, 3, 903-907.*

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