

CES For Mild Traumatic Brain Injury

By Daniel L. Kirsch, PhD, FAIS



Based on the consistently robust results of cranial electrotherapy stimulation (CES) seen throughout this preliminary data, the use of this safe and effective electromedical modality as an adjunct to medical and psychological interventions for mild traumatic brain injury patients should be considered.

Cranial electrotherapy stimulation (CES) is the generic term for a medical treatment that entered the USA from the USSR and Europe in the 1960s as “electrosleep.” It involves the passage of small levels of microcurrent stimulation in a specific waveform through the brain to normalize and bring back into homeostatic balance the brain’s electrochemistry which can be thrown into disarray by physical or psychological trauma. Once back into a prestress homeostasis, the brain can function normally again, carrying out its myriad systemic regulation duties.

CES treatment is generally administered for 20 minutes to one hour per day, at least once a week, but as often as daily in severe cases. Most symptoms improve significantly during a customary initial three week evaluation period, although patients are often provided a CES unit to have at home to use from time to time in order to prevent symptoms from returning.

This article reviews the promising available information on cranial electrotherapy stimulation (CES) for mild traumatic brain injury (mTBI) in the civilian sector but it is equally applicable to the military rehabilitation setting since mild traumatic brain injury (mTBI) is one of the signature injuries of the current wars in Iraq and Afghanistan.

Electroconvulsive Therapy, Electroanesthesia, and CES

CES was advanced in Europe under the assumption that if the strong current used in electroconvulsive therapy (ECT) were turned down sufficiently, electroanesthesia could be produced so that surgery could be performed on an unconscious patient without the danger of concomitant seizure activity. Once electroanesthesia devices were available, physicians who had an interest in sleep therapy or who merely wanted to provide a non-drug way of assisting insomniacs, had the electroanesthesia current turned down another substantial notch to microcurrent levels and obtained a treatment originally called electrosleep, the forerunner of CES. The idea was to induce sleep, then turn off the CES device to allow the patient to complete a good nights rest.

Much research was done in American, Russian, and European medical schools and other research centers in the 1960s and early 1970s to learn what parameters—waveform, frequencies, current levels, etc.—were necessary to reliably induce sleep in their patients. These researchers were never able to find a set of electrical stimulation parameters that reliably induced sleep.¹⁻⁵

A serendipitous discovery, however, was that the patients who were given even one

treatment with CES reported general feelings of relaxation and a substantial reduction of anxiety.⁶⁻¹⁰ When treated daily over a few weeks to a month, even severe cases of anxiety and depression resolved.¹¹⁻¹³

CES Research In The US: Stress, Rehabilitation, Cognitive Improvements

A major reorientation of CES studies and clinical use followed. Controlled scientific studies began on the substance abstinence syndrome—with its major symptoms of anxiety, insomnia and depression—of patients withdrawing from illicit drugs and/or pharmaceuticals, alcohol or nicotine.¹⁴⁻¹⁹ Other studies looked at the stress of graduate students in a business management training program,²⁰ incarcerated prisoners on a prison psychiatric ward,²¹ and of psychiatric patients in general.²²⁻²⁴

Because of its ability to treat stress under such a wide assortment of patient populations, CES soon became a major component of rehabilitation medicine. Patients in rehabilitation programs are well known to suffer from extreme stress, including anxiety and depression as they and their therapists work to get their lives back to a semblance of normality.^{25,26} It found use in treating the stress-related symptoms in para- and quadriplegic patients and, in doing so, it was found to sig-

nificantly reduce their muscle spasticity.^{2,27} CES also proved beneficial for cerebral palsy patients in gaining control of primitive reflexes and brought many of their other neuromuscular symptoms under control.²⁸⁻³⁰

Another serendipitous finding was that in every case where patients experienced an improvement in their stress level, they also experienced a dramatic improvement in cognitive function, with an average gain of 12 to 18 points on standardized IQ tests administered just previous to and following three weeks of daily CES treatment. It was in this manner that researchers found that so-called permanent brain damage in drug and alcoholic addicts was not permanent. While the cognitive abilities of most such patients would approach normal following two years of total abstinence, it could return to normal with just three weeks of CES treatment.³¹⁻³³

Research attention was then turned to patients with mild traumatic brain injuries (mTBI) incurred in various ways such as

remove restraints or tubes.”

A 33-year-old male Rancho IV patient was given p.r.n. droperidol along with CES. The patient developed meningitis at age 14 which left him with a generalized tonic-clonic seizure disorder. Seizures were controlled until two years later when he suffered a significant concussion playing high school football. At that time he experienced up to seven generalized tonic-clonic seizures a day, uncontrollable by medication. In 1989, he underwent a right temporal lobectomy which resulted in a disappearance of the seizures for two years. In 1991, he experienced the acute onset of status epilepticus followed by a prolonged coma. He was diagnosed with viral encephalitis. On awakening, his behavior deteriorated into confusion, sexual inappropriateness, and dangerous aggression. MRI showed left temporal ischemia and atrophy with enlargement of the left temporal horn and atrium of the left temporal ventricle. Topographical EEG showed increased right temporal

fusals was down 29%, and elopement attempts were down 33%. Because of these dramatic changes in behavior, the patient no longer required continuous 1:1 supervision, and p.r.n. injections for explosive behavior became infrequent, then completely unnecessary. Although the patient was still confused and required a closed treatment setting, he was no longer aggressive, which clearly had been his most problematic and dangerous behavior.³⁴

Case 2: Aggressive Behavior

A 57-year-old unmarried Caucasian woman was admitted to the maximum-security unit of North Texas State Hospital at Vernon, after having been found to be Manifestly Dangerous at another State hospital where she had carried out 17 assaults on peers and staff over a two-month period. In spite of numerous medication changes, 1:1—and even 2:1—staff coverage and other specialized interventions, she continued to attack, throw furniture, kick walls and doors, and required frequent restraints. She would fall down up to eight times a day, accuse staff of shoving her, and make false reports to the Department of Regulatory Services. She was floridly paranoid, developed grudge lists, and would follow peers and staff around yelling at them to get away from her. At other times, she would target peers for assault when they were taking staff's time and attention, which she was demanding. She sometimes expressed remorse over her actions, but did not change her behavior.

The patient's history of psychiatric hospitalizations began nearly 40 years prior, having first been hospitalized at age 15. Since 1991, she was in Texas State hospitals 11 times and was in prison for two years for stealing a car with a baby in the back seat. For the past 13 years, she was homeless when not incarcerated. Throughout the years, antipsychotic medication, including clozapine, would produce a certain level of improvement in the schizoaffective disorder, in that her hallucinations would become quiescent, but she was never able to be maintained in halfway houses or nursing homes because of her violent behavior. Her last such placement ended when she broke an attendant's arm. She was thought to be of borderline intelligence, but had obtained a General Educational Development (or GED) test while in prison. The patient had grown up in a sexually and physical-

“...in every case where patients experienced an improvement in their stress level, they also experienced a dramatic improvement in cognitive function, with an average gain of 12 to 18 points on standardized IQ tests administered just previous to and following three weeks of daily CES treatment.”

motor vehicle accidents or falls from high elevations on construction projects. That group drew special attention because the majority of them were known seizure patients and little was known of the effects of CES on seizure patients.

Case Summaries

Case 1: ‘Rancho Level IV’ Patients

Confused and agitated TBI (or post-anoxic or post-stroke) patients may be classified as ‘Rancho Level IV’ from the Rancho Los Amigo I-VIII Scale and would be described as follows:

“The patient is in a heightened state of activity with severely decreased ability to process information. He is detached from the present and responds primarily to his own internal confusion. Behavior is frequently bizarre and non-purposeful relative to his immediate environment. He may cry out or scream out of proportion to stimulus even after removal. He may show aggressive behavior and attempt to

and frontal slowing. Brain stem auditory, middle latency, and 40 Hz evoked potentials were all abnormal. Haldol, Norpramin, Verapamil, lithium, sodium amytal, and lorazepam were all ineffective.

In the first seven months of his inpatient stay, he was physically aggressive 247 times, made 58 verbal threats, engaged in door rattling 1,983 times, refused tasks 453 times, and attempted to elope 200 times. On introduction of droperidol p.r.n., the assaultive episodes decreased from an average of 8 to 4 per week, with substantially diminished duration.

Five weeks after beginning droperidol, 30 minutes twice daily, CES was added. There was a lessening of tension in the first three weeks of treatment, and the violent assaults ceased. In addition to extinguishing the physical aggression, the p.r.n. droperidol/CES treatment was successful in decreasing the other four behaviors: door rattling was down 76%, verbal threats were down 100% (to zero), task re-

ly abusive home, had started using alcohol and street drugs at age 12, and by 21 she described herself as an alcoholic like her father.

In the first three months at NTSH-V, she was treated with maximum doses of quetiapine and ziprazidone along with a large dose of oxcarbazepine and escitalopram. She had 12 episodes of physical assault in this pre-CES period, requiring 12 restraints and 66 PRN medication administrations. In spite of the large doses of medicine, she was sleepless many nights, ate irregularly, and was deeply paranoid and withdrawn between aggressive outbursts.

CES was started at 0.5 Hz, one hour twice daily and 15 to 45 minutes, up to three times daily, for her frequent agitated episodes. Compliance with CES improved after two weeks and she began sleeping and eating better. Oxcarbazepine and ziprazidone were discontinued and a small dose of clozapine (200mg/day) was added. Two weeks later, the quetiapine dose was cut in half and she continued the escitalopram. In the first month of CES, she had only five aggressive episodes, requiring four restraints and PRN's dropped to 19.

After six weeks of CES, her personality changed dramatically. She became outgoing, was no longer accusatory, and her grooming and hygiene became exceptional. Her assaultive behavior stopped altogether, as did the necessity for PRN's and other interventions. At the end of three months of CES, she passed the Dangerousness Review Board and was returned to the referring hospital. There was no recurrence of her illness on discontinuation of the CES treatments. Observers familiar with the patient from her years at both hospitals commented on what a different person she had become.³⁵

Double Blind Pilot Study of CES for TBI

A double blind pilot study was conducted on 21 closed TBI patients who were living in a supervised care home. Their time since injury ranged from six months to 32 years and their ages ranged from early teenagers to those in their 40s and 50s (average age of 30 years).

The subjects were randomly assigned to CES treatment (N=10), sham CES treatment (N=5), or "wait in line" controls (N=6). The therapists, patients, and the statistician all remained blind to treatment conditions.

Profile of Mood States Subscale	CES treatment pre to post N = 10	Sham treatment pre to post N = 5	Control pre to post N = 6
Tension Anxiety	12.33 ± 7.36 to 8.78 ± 5.09	13.00 ± 6.21 to 14.36 ± 8.25	12.33 ± 8.07 to 12.50 ± 5.87
Depression Dejection	17.11 ± 12.35 to 12.06 ± 8.71	20.91 ± 17.79 to 18.18 ± 12.47	20.00 ± 14.45 to 16.17 ± 9.48
Anger Hostility	13.67 ± 11.20 to 10.39 ± 7.49	16.73 ± 8.27 to 17.55 ± 12.22	14.83 ± 11.50 to 14.83 ± 6.18
Fatigue Inertia	7.44 ± 6.75 to 5.33 ± 3.96	9.46 ± 7.83 to 8.09 ± 6.63	8.17 ± 7.41 to 6.50 ± 5.82
Confusion Bewilderment	8.50 ± 6.75 to 6.22 ± 3.96	10.55 ± 5.87 to 10.27 ± 5.10	9.67 ± 6.15 to 10.50 ± 5.01
Total Mood Disturbance	45.11 ± 41.95 to 31.89 ± 23.84	52.73 ± 41.95 to 52.33 ± 36.64	47.83 ± 43.25 to 45.67 ± 24.16

TABLE 1. Profile of Mood States pre- and post-scores from CES, sham CES, and wait in line controls in a double blind pilot study on traumatic brain injuries.

CES or sham CES was administered below sensation threshold Monday through Thursday for three weeks for a total of 12 one hour sessions. It was found that anxiety and depression scores improved significantly in the treatment group, but not in the placebo (sham treated) group, or the wait in line control group. Their fatigue scores also improved significantly, as did their cognitive function scores and their Total Mood Disturbance score on the Profile of Mood States psychometric test as shown in Table 1.

During the study, one of the subjects who had brain cancer had a seizure and was immediately removed from the study by the principal investigator. Following the study, the 11 patients in the two control groups were given CES for three weeks. It had been learned that the patient who had the seizure during the double blind phase of the study was receiving sham CES treatment. Upon the insistence of his parents, he also received actual CES treatment for three weeks following the study. Neither he nor any of the other subjects in the study experienced a seizure while receiving actual CES treatment and, according to house attendants, their seizure experience in the weeks following the study was unremarkable.³⁶

CES Mechanisms and EEG Studies

Possible mechanisms of CES have been studied. Researchers at the University of Wisconsin found that even though the current applied was small (in the mi-

croamperage range), 42% to 46% of the current applied externally actually went through the entire brain, but canalized especially along the limbic system and its centers of emotional experience and expression.³⁷

Scientists at the University of Tennessee Medical Center completed a series of five studies that used various drugs to deliberately cause Parkinson-like symptoms in canine subjects. They found that once brain homeostasis was thrown into dramatic disarray, the application of CES could bring it back into apparent neurochemical homeostasis within 3 to 7 hours. Left to their normal care—but without CES—the dogs required 4 to 7 days to return to normal behavior once the drugs had been removed.³⁸

Over the years, a number of EEG studies have been done pre- and post-CES treatments. Kennerly did the best EEG study to date revealing significant increases in alpha activity denoting more relaxation and significant decreases in delta activity that accounts for the increased alertness typically seen following CES.³⁹ Cox did a crossover study on a female depressed patient and found that following CES, but not sham CES, she became sleepy and drowsy for the first time in months, exhibiting a well-developed alpha rhythm in the occipital cortex.⁴⁰ Empson studied student volunteers in a sleep lab and found that CES treatment was accompanied by an EEG state suggesting an alteration in the mood of tense,

anxious students to one of relaxation.¹

Heffernan completed two studies of the EEG spectrum in chronic pain patients in which he completed fast fourier transformation and chaos correlation dimension analyses and found CES to bring the EEG back into a coherent, pre-stress pattern.^{41,42} Itil used a computerized frequency analyzer to study the EEG's of ten male volunteers in a crossover design. He found that the effect of CES on the EEG depended on whether the subjects were resting or involved in reaction measurements. Those who began with an EEG suggesting a relaxed state became even more relaxed, while those who began in an alert state remained in the alert state. During reaction time measurements, there was an increase in 5-10 Hz activity and a decrease in fast alpha and beta activity following CES.⁴

McKenzie completed an EEG study with eight psychiatric patients suffering chronic anxiety with depression and in-

any of these patients.⁴⁴

There were other EEG studies, but as can be seen, the findings in the above studies varied depending on the subject population, EEG testing parameters and so on, yet in every case there was a robust normalizing trend found in the EEG following CES treatment, whether the subject population were addicts, patients undergoing treatment in a psychiatric hospital, patients in a sleep laboratory, or simply students in a graduate school experimental EEG laboratory. There was no instance in which an EEG indicated adverse effects from CES treatment.

After several years of using CES alone, it was discovered that it potentiated biofeedback, including the speed of learning, length of retention, and ongoing patient improvement if given just before or along with biofeedback of various kinds.⁴⁵⁻⁴⁷ It potentiated the hypnotherapy process, increasing the speed and depth of induction, and often permitted

days, semi-comatose for ten days, and in a state of coma vigil for 20 days. Three months after the accident he showed a dense left hemiparesis, and a Wechsler Memory Scale (WMS) score of 61, Full Scale IQ of 71. Six months after the accident his IQ improved to 80, but the WMS score was essentially unchanged at 63. He showed marked deficits in short-term retention of visual and verbal information. By eight months after the accident his WMS was 83, but acquisition of new learning was significantly impaired. This patient was treated with CES 40 minutes daily for three weeks. He averaged 29 correct responses immediately after CES, and 30 correct responses (delayed recall) 30 minutes later. At the end of three weeks of CES, immediate recall was in the 36-37 point range, while delayed recall increased to 40 (33% improvement). Ten days following discontinuation of treatment, he averaged 45 in immediate recall (55% improvement), and delayed recall improved to an average of 47 (56% increase over baseline).

The other report was of a 58-year-old orthopedic surgeon who sustained a closed head injury in a motor vehicle accident in 1984, with extensive lacerations and a broken leg. Initial CT scan revealed intraventricular hemorrhages within the occipital horns of both ventricles. An area that appeared consistent with an infarction of the left anterior thalamus was also noted. An EEG three weeks later showed slowing consistent with diffuse encephalopathic process. CT scans one month later showed clearing of the hemorrhages and progressive dilation of the ventricles. From the day of injury, the patient was extremely confused, disoriented, and demonstrated severe memory deficits. Twelve weeks after the injury he was transferred to a rehabilitation hospital where he exhibited disorientation, memory disturbance, and delusions of being dead. He had difficulty distinguishing between fantasy and reality, and experienced overwhelming anxiety during periods of disorientation. His problems were with new memory, exemplified by his successful completion of a state medical board exam after which he was unable to find his way out of the building. He could not drive because he could not remember where he was going. He was diagnosed with diencephalic amnesia secondary to trauma. Baseline scores averaged 29 for immediate recall, and 23 for delayed recall. After

“Childs reported on the effectiveness of CES in two cases of post-traumatic amnesia. The first was a 21-year-old male who sustained a TBI following a motorcycle accident but recovered much of his tested memory recall functions following a series of CES treatments administered three and one-half years after the accident.”

somnia and compared them with four normal staff controls. Following one CES treatment a day for five days, the patients showed increased quality and quantity of alpha with increased amplitude in the occipital-parietal leads.⁴³ Magora studied 20 hospitalized patients suffering from long-lasting insomnia with anxiety, obsessive and compulsive reactions, morphine and barbiturate addiction, and involuntal depression. They were give two to four CES treatments weekly for two to three hours a day for a total ranging from 10 to 20 treatments. A majority (75%) of the patients were labeled as responders to the treatment with a return to a normal sleep pattern as measured by their EEG. Parallel with the return to a normal sleep pattern, all the other psychiatric signs (e.g., anxiety, depression, agitation, delusions, abstinence syndrome) improved significantly so that all were able to be discharged from the hospital. There was no relapse on an 8 to 12 month follow up in

hypnosis resistant patients to be hypnotized.⁴⁸ Similarly, it was found that it potentiated the effects of psychoactive medications,³⁴ and also general anesthetics in surgery patients by approximately 37%, allowing the patient to remain anesthetized with less anesthesia as the surgery progressed, waking sooner following surgery, and experiencing less pain during recovery.^{26,49,50}

Post-traumatic Amnesia

Childs reported on the effectiveness of CES in two cases of post-traumatic amnesia. The first was a 21-year-old male who sustained a TBI following a motorcycle accident but recovered much of his tested memory recall functions following a series of CES treatments administered three and one-half years after the accident. Post-accident, computerized tomography (CT) revealed a right lateral basal ganglia hemorrhage and hemorrhages into both ventricles. He was totally unresponsive for ten

one week of CES, immediate recall averaged 35 and delayed recall averaged 25. After three weeks of CES, immediate recall averaged 35 and delayed recall averaged 31. During three weeks following discontinuation of treatment, he was tested three times and averaged 37 on immediate recall (28% improvement), and 32 on delayed recall (39% improvement).

Subjective observations by staff indicated visible improvements in mood, spontaneity, and initiative in both patients, but deteriorated rapidly after the treatment was stopped. Nevertheless, the authors stated that the clinical improvement in these two patients cannot be ignored.⁵¹

Combined TBI and Global RSD

Another case was reported of a 60-year-old male treated with CES for an intracranial TBI coupled with full body reflex sympathetic dystrophy (global RSD). In spite of severe disabilities of his brain and body, WHH continued to serve his country in his position on the Executive Staff of the President's Committee on Employment of People with Disabilities. Daily 20 minute treatments of CES provided satisfactory pain relief for WHH to complete his tasks and enjoy a relatively higher quality of life than he was able to have with drugs alone.

Prior to CES, WHH has been prescribed numerous medications including Prozac 20mg q.i.d., Catapres Tab 20mg q.d., Effexor 100mg in AM and 50mg at bedtime, Levo-Dromoran 1mg b.i.d., Balofen 10mg split AM and PM, Risperdal 7.5mg at bedtime, Kolopin 0.5mg 1 tab t.i.d. to q.i.d. per day as needed, C-Dextromthroph 60mg t.i.d. and Fentanyl patches for four years. This regime did little to reduce his whole body chronic intense critical pain and burning. Nor did it relieve his difficulty sleeping. Transcutaneous electrical nerve stimulation did not help. WHH claims these treatments made him worse and expressed concerns about the short and long term side effects the drugs had on his ability to function.

WHH was provided CES at George Washington University Medical Center prior to oral surgery. He exhibited marked relaxation from CES, with a reduced anxiety level and a significantly enhanced pain threshold. Based on these positive results he was prescribed 20 minute CES treatments daily via ear clip electrodes. WHH credits the CES treatment for allowing him to return to work,

and for improving his family and social life. Prior to CES he claimed that "life was not worth living to the degree that suicide was an attractive option." He found this treatment provided him a moderate improvement of 50-74% relief from his pain, anxiety, depression, headaches, and muscle tension, and a marked improvement of 75-99% in his insomnia.

A single CES treatment lasted 6 to 8 hours, allowing him to get through the day, then the pain gradually returned. In his own words, "The Alpha-Stim 100 [the CES unit used] has given me short term relief from my pain levels that medications have not been able to accomplish. While the relief periods may only be for 8 hours or so, these near pain-free hours allow my body to recycle itself, granting me an improved quality of life. Without this therapy, the constant 'level 10' debilitating pain levels leave me with no physical or emotional reserves to carry on daily life. The CES therapy has no side effects, whereas my medicines have profound, crippling and lasting side effects that have impaired my bowel and colon. These impairments can not be reversed." On a zero (no pain) to 10 (maximum pain) scale, He says CES reduces his pain level from a 10 to a 3 which he describes as "the difference between standing on a busy street in New York at 5 PM and fly fishing on a tranquil creek." He added "CES provides me with a measure of pain relief that brings me back from the depth of despair and gives me a wedge of hope."

CES reduced his pain level to a point where he was able to perform his daily exercise routine. He was also able to rest better at night, which he credited as creating a "positive emotional and physical self-environment." He felt more rested in the morning. He was able to work 30 to 40 hours per week, up from a maximum of 15 hours prior to CES.

Following CES, his medication had been reduced to Prozac 10mg q.d., Catapres Tab 0.1mg b.i.d., Effexor 50mg AM and 25mg PM, Levo-Dromoran 1mg b.i.d., Restoril 7.5mg at bedtime, Kolopin p.r.n., and Neurontin 400mg p.r.n. 52

Conclusion

Some researchers have said that the current mass pandemic of fibromyalgia patients may be due to brain dysfunction following whiplash injury or similar traumas to the brain.⁵³ That concept is still under discussion, but meanwhile two independ-

ent double blind studies have shown CES to be a very effective treatment for pain and mood disorders in fibromyalgia patients.^{54,55} CES has also shown to be effective in two double blind studies of spinal cord injuries, an anatomically-related pathology.^{56,57}

This preliminary evidence supports the hypothesized ability of CES to functionally stabilize the traumatized brain and return it toward a condition of pre-injury homeostatic functioning. Additional research will likely confirm these findings and definitively prove CES to be an effective treatment for patients with traumatic brain injury or, at the very least, a significantly beneficial adjunct to other forms of physical, and psychological therapies for this heavily-medicated population. ■

Daniel L. Kirsch, PhD, FAIS is an internationally renowned authority on electromedicine with over three decades experience in the field who currently serves as the Electromedical Dept. Editor of Practical Pain Management and a Contributing Editor of the Journal of Neurotherapy. He was board-certified as a Diplomate of the American Academy of Pain Management in 1990 and was named a Fellow of the American Institute of Stress in 1997. He is a Member of the International Society of Neurofeedback and Research and a Member of Inter-Pain (an association of pain management specialists in Germany and Switzerland). He served as Clinical Director of The Center for Pain and Stress-Related Disorders at Columbia-Presbyterian Medical Center, New York City, and of The Sports Medicine Group, Santa Monica, California. Dr. Kirsch is the author of two books on CES titled, The Science Behind Cranial Electrotherapy Stimulation, 2nd Ed. published by Medical Scope Publishing Corporation, Edmonton, Alberta, Canada in 2002; and Schmerzen lindern ohne Chemie CES, die Revolution in der Schmerztherapie, Internationale Ärztgesellschaft für Energiemedizin, Austria 2000 (in German). Dr. Kirsch is a research consultant to the US Army and VA Medical Centers and currently spends much of his time giving lectures at national military conferences and grand rounds at Veterans Affairs Medical Centers and U.S. Army hospitals such as Brooke Army Medical Center, Walter Reed Army Medical Center, and William Beaumont Army Medical Center. Best known for designing the Alpha-Stim line of medical devices, Dr. Kirsch is Chairman of Electromedical Products International, Inc. of Mineral Wells, Texas, USA with additional offices in Europe and Asia. Dr. Kirsch can be reached at dan@epii.com.

References

1. Empson JAC. Does electrosleep induce natural sleep? *Electroencephalography and Clinical Neurophysiology*. 1973. 35(6):663-664.
2. Forster S, Post BS, and Benton JG. Preliminary observations on electrosleep. *Archives of Physical Medicine and Rehabilitation*. 1963. 44:481-489.
3. Frankel BL, Buchbinder R, and Snyder F. Ineffectiveness of electrosleep in chronic primary insomnia. *Archives of General Psychiatry*. 1973. 29:563-568.
4. Itil T, Gannon P, Akpınar S, and Hsy W. Quantitative EEG analysis of electrosleep using frequency analyzer and digital computer methods. *Electroencephalography and Clinical Neurophysiology*. 1971. 31:294.
5. Maagora F, Beller A, Aladjemoff L, Magora A, and Tannenbaum J. Observations on electrically induced sleep in man. *British Journal of Anaesthesiology*. 1965. 37:480-491.
6. Long RC. Electro-sleep therapy. Some results with the use of electrically induced sleep in the treatment of psychiatric patients. *Journal of the Kansas Medical Society*. 1966. 67(2):81-85.
7. Rosenthal SH and Calverly LG. electro-sleep: Personal subjective experiences. *Biological Psychiatry*. 1972. 4(2):187-190.
8. Rosenthal SH and Wulfsohn NL. Electro-sleep: A clinical trial. *American Journal of Psychiatry*. 1970. 127(4):175-176.
9. Singh JM, King HA, and Super WC. Effects of transcranial electrotherapy (TCT) in stress related illness. *Pharmacologist*. 1974. 16(2):264.
10. Rosenthal SH and Wulfsohn NL. Electro-sleep: A preliminary communication. *Journal of Nervous and Mental Disease*. 1970. 151(2):146-151.
11. Rosenthal SH and Wulfsohn NL. Studies of electro-sleep with active and simulated treatment. *Current Therapeutic Research*. 1970. 12(3):126-130.
12. Rosenthal SH. Electro-sleep: A double-blind clinical study. *Biological Psychiatry*. 1972. 4(2):179-185.
13. Moore JA, Mellor CS, Standage KF, and Strong H. A double-blind study of electro-sleep for anxiety and insomnia. *Biological Psychiatry*. 1975. 10(1):59-63.
14. Bianco Jr F. *The efficacy of cranial electrotherapy stimulation (CES) for the relief of anxiety and depression among polysubstance abusers in chemical dependency treatment*. Ph.D. Dissertation. The University of Tulsa. 1994.
15. Gold MS, Pottash ALC, Sternbach H, Barbaban J, and Annitto W. *Anti-withdrawal effects of alpha methyl dopa and cranial electrotherapy*. Society for Neuroscience, 12th Annual Meeting Abstracts. October, 1982.
16. Gomez E and Mikhail AR. Treatment of methadone withdrawal with cerebral electrotherapy (electrosleep). *British Journal of Psychiatry*. 1978. 134:111-113.
17. Schmitt R, Capo T, and Boyd E. Cranial electrotherapy stimulation as a treatment for anxiety in chemically dependent persons. *Alcoholism: Clinical and Experimental Research*. 1986. 10(2):158-160.
18. Smith RB and O'Neill L. Electro-sleep in the management of alcoholism. *Biological Psychiatry*. 1975. 10(6):675-680.
19. Patterson MA, Firth J, and Gardiner R. Treatment of drug, alcohol and nicotine addiction by neuroelectric therapy: Analysis of results over 7 years. *Journal of Bioelectricity*. 1984. 3(1&2):193-221.
20. Matteson MT and Ivancevich JM. An exploratory investigation of CES as an employee stress management technique. *Journal of Health and Human Resource Administration*. 1986. 9:93-109.
21. Jemelka R. *Cerebral electrotherapy and anxiety reduction*. Master's Thesis, Stephen F. Austin State University. May, 1975.
22. Herst ED, Cloninger CR, Crews EL, and Cadoret RJ. Electro-sleep therapy: A double-blind trial. *Archives of General Psychiatry*. 1974. 30(4):463-66.
23. Feighner JP, Brown SL, and Olivier JE. Electro-sleep therapy: A controlled double-blind study. *Journal of Nervous and Mental Disease*. 1973. 157(2):121-128.
24. Koegler RR, Hick SM, and Barger JH. Medical and psychiatric use of electro-sleep (transcranial electrotherapy). *Diseases of the Nervous System*. 1971. 32(2):100-104.
25. Kirsch DL and Smith RB. The use of cranial electrotherapy stimulation in the management of chronic pain: A review. *NeuroRehabilitation*. 2000. 14:85-94.
26. Kirsch DL and Gilula MF. CES in the treatment of pain-related disorders. *Practical Pain Management*. 2008. 8(3):12-25.
27. Wharton GW, McCoy CE, and Cofer J. *Effect of CES therapy on spinal cord injured patients*. Presented at the American Spinal Injury Association. New York. April, 1982.
28. Logan MP. Improved mechanical efficiency in cerebral palsy patients treated with cranial electrotherapy stimulation (CES). 1988. In Kirsch, DL, *The Science Behind Cranial Electrotherapy Stimulation*. 2nd Ed. Medical Scope Publishing, Edmonton, Alberta, Canada. 2002.
29. Malden JW and Charash LI. Transcranial stimulation for the inhibition of primitive reflexes in children with cerebral palsy. *Neurology Report*. 1985. 9(2):33-38.
30. Okoye R and Malden JW. Use of neurotransmitter modulation to facilitate sensory integration. *Neurology Report*. 1986. 10(4):67-72.
31. Smith RB and Day E. The effects of cerebral electrotherapy on short-term memory impairment in alcoholic patients. *International Journal of the Addictions*. 1977. 12(4):575-562.
32. Smith RB. Confirming evidence of an effective treatment for brain dysfunction in alcoholic patients. *Journal of Nervous and Mental Disease*. 1982. 170(5):275-278.
33. Schmitt R, Capo T, Frazier H, and Boren D. Cranial electrotherapy stimulation treatment of cognitive brain dysfunction in chemical dependence. *Journal of Clinical Psychiatry*. 1984. 45(2):60-63.
34. Childs A. *Droperidol and CES in Organic Agitation*. Clinical Newsletter, Austin Rehabilitation Hospital. 1995.
35. Childs A and Price L. Cranial electrotherapy stimulation reduces aggression in violent neuropsychiatric patients. *Primary Psychiatry*. 2007. 14(3):50-56. Presented at the American Psychiatric Association 160th Annual Meeting: Addressing Patient Needs. San Diego, California. May 23, 2007.
36. Smith RB, Tiberi A, and Marshall J. The use of cranial electrotherapy stimulation in the treatment of closed-head-injured patients. *Brain Injury*. 1994. 8(4):357-361.
37. Jarzembki WB, Larson S J, and Sances Jr A. Evaluation of specific cerebral impedance and cerebral current density. *Annals of the New York Academy of Sciences*. 1970. 170:476-90.
38. Pozos RS, Strack LE, White RK, and Richardson AW. Electro-sleep versus electroconvulsive therapy. In Reynolds, David V. & Sjoberg, Anita E. (Eds). *Neuro-electric Research*. Charles Thomas. Springfield. 1971. 23:221-225.
39. Kennerly R. QEEG analysis of cranial electrotherapy: a pilot study. *Journal of Neurotherapy*. 2004. (8)2:112-113. Presented at the International Society for Neuronal Regulation Annual Conference. Houston, Texas. September 18-21, 2003.
40. Cox A and Heath RG. Neurotone therapy: A preliminary report of its effect on electrical activity of forebrain structures. *Diseases of the Nervous System*. 1975. 36(5):245-247.
41. Heffernan M. Comparative effects of microcurrent stimulation on EEG spectrum and correlation dimension. *Integrative Physiological and Behavioral Science*. 1996. 31(3):202-209.
42. Heffernan M. The effect of variable microcurrents on EEG spectrum and pain control. *Canadian Journal of Clinical Medicine*. 1997. 4(10):4-11.
43. McKenzie RE, Rosenthal SH, and Driessner JS. *Some psycho-physiological effects of electrical transcranial stimulation (electrosleep)*. American Psychiatric Association, Scientific Proceedings Summary. 1971.
44. Magora F, Beller A, Assael MI, and Askenazi A. some aspects of electrical sleep and its therapeutic value. In Wageneder, F.M. and St. Schuy (Eds). *Electrotherapeutic Sleep and electroanesthesia*. *Excerpta Medica Foundation, International Congress Series No. 136*. Amsterdam. 1967. pp 129-135.
45. Brotman P. Low-intensity transcranial electrostimulation improves the efficacy of thermal biofeedback and quieting reflex training in the treatment of classical migraine headache. *American Journal of Electromedicine*. 1989. 6(5):120-123.
46. Overcash SJ and Siebenthal A. The effects of cranial electrotherapy stimulation and multisensory cognitive therapy on the personality and anxiety levels of substance abuse patients. *American Journal of Electromedicine*. 1989. 6(2):105-111.
47. Kelley J, Whitney K, Irene H, and Kaiman C. Cerebral electric stimulation with thermal biomedical feedback. *Nebraska Medical Journal*. 1977. 62(9):322-26.
48. Barabasz AF. Hypnosis and cerebral electrotherapy in the treatment of sleep disturbances in mildly depressed patients. *Hypnosis Quarterly*. 1976. 19(2):321-34.
49. Stanley TH, Cazalaa JA, Atinault A, Coeytaux R, Limoge A, and Louville Y. Transcutaneous cranial electrical stimulation decreases narcotic requirements during neurolept anesthesia and operation in man. *Anesthesia and Analgesia*. 1982. 61(10):863-866.
50. Stanley TH, Cazalaa JA, Limoge A, and Louville Y. Transcutaneous cranial electrical stimulation increases the potency of nitrous oxide in humans. *Anesthesiology*. 1982. 57:293-97.
51. Childs A and Crismon ML. The use of cranial electrotherapy stimulation in post-traumatic amnesia: a report of two cases. *Brain Injury*. 1988. 2(3):243-47.
52. Alpher EJ and Kirsch DL. Traumatic brain injury and full body reflex sympathetic dystrophy patient treated with cranial electrotherapy stimulation. *American Journal of Pain Management*. 1998. 8(4):124-128. Presented at the Ninth Annual Clinical Meeting of the American Academy of Pain Management. Atlanta, Georgia. September 1998.
53. Bennett RM, Cook DM, Clark SR, Burckhardt CS, and Campbell SM. Hypothalamic-pituitary-insulin-like growth factor-1 axis dysfunction in patients with Fibromyalgia. *Journal of Rheumatology*. 1997. 24:384-389.
54. Cork RC, Wood P, Ming N, Clifton S, James E, and Price L. The effect of cranial electrotherapy stimulation (CES) on pain associated with fibromyalgia. *The Internet Journal of Anesthesiology*. 2004. 8(2).
55. Lichtbroun AS, Raicer MM, and Smith RB. The treatment of fibromyalgia with cranial electrotherapy stimulation. *Journal of Clinical Rheumatology*. 2001. 7(2):72-78.
56. Capel ID, Dorell HM, Spencer EP, and Davis MWL. The amelioration of the suffering associated with spinal cord injury with subperception transcranial electrical stimulation. *Spinal Cord*. 2003. 41:109-117.
57. Tan G, Rintala DH, Thornby J, Yang J, Wade W, and Vasilev C. Using cranial electrotherapy stimulation to treat pain associated with spinal cord injury. *Journal of Rehabilitation Research and Development*. 2006. 43(4):461-474. Presented at the South Central VA Health Care Network's Pain Management Initiative 2nd Annual Pain Management Symposium: Campaign Against Pain. Jackson, Mississippi. April 7, 2006.