

## The Use of CES in the Maintenance of Health and Wellbeing

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Cranial Electrotherapy Stimulation (CES) is the American FDA's term for what the rest of the world calls "electrosleep." Modern electrosleep devices originated in Russia in 1953, and arrived in the U.S. ten years later, in 1963, when they began to be researched with patients complaining of insomnia.

Various uses of small to moderate electrical currents had been researched since the early 1900s in Europe, in an attempt to see exactly what current intensity and pulse rate were required to put a patient to sleep when applied to the head. By that, they meant what treatment parameters were required to knock him out or force him to lose consciousness and maintain the patient in that state for a period of time. Researchers finally gave up on finding a specific type of current that would reliably put most patients to sleep. Unlike those earlier models, modern CES devices are typically pocket sized, run off of a 9 volt battery, and pulse from 100 up to 15,000 times per second. The current intensity usually is at or just below 1 mAmp, but can go up to 4 mAmp with higher pulse rates. Most CES devices, when fully on, would just light a flashlight bulb at best, and in the majority of clinical studies - which were double blind - patients have not felt the stimulation at all during treatment.

In the early 1950s Russian medical researchers were working with these very low levels of current, which they applied via two electrodes attached to the closed eyelids and two attached behind the head at the base of the skull. They were attempting to find a psychiatrically useful current, and while the current level was much too low to force a person into a sleep state, they found to their great interest that patients were claiming vastly improved sleep during nights following sessions when these very minor amounts of stimulation passed across the head. They then began studying this sleep inducing effect specifically, and in 1953 finally came out with the Somniatron electrosleep device.

Several similar devices were later manufactured in the U.S. for research purposes, and their clinical use began among inpatient and outpatient psychiatric patients, usually in University Teaching Hospitals. Several other Universities began research with animals in an effort to see if CES really did change how the brain functioned, if it was safe to use, and what the mechanism of action might be.

They found that the current traveled throughout the brain,<sup>1,2</sup> that it increased production and firing of neurotransmitters in neurons,<sup>3</sup> and that when researchers deliberately threw neurotransmitters out of balance in the brain, electrosleep would put them back into balance.<sup>4-6</sup> Other researchers found that electrosleep would apparently also put back into balance those neurotransmitters in patients whose neurotransmitters had been thrown out of balance by various addicting substances.<sup>7-10</sup>

Following the initial research in the U.S., several units began to be sold for clinical use before the 1976 Amendment gave the FDA control over medical devices. The Amendment gave the FDA power to determine the safety and effectiveness of medical devices prior to allowing them into the U.S. marketplace, and all electrosleep devices which were on the open market prior to the Amendment were grandfathered and left on the market, with a provision that the FDA could call them in later to have them show their safety and effectiveness, a process costing up to an estimated \$800 million.<sup>11</sup>

The FDA also decided to call electrosleep devices Cranial Electrotherapy Stimulation, since by then their clinical uses had expanded from sleep to include depression and anxiety. A preliminary look at CES by the FDA's Neurology Panel in 1978 suggested they should be accepted for the safe and effective treatment of addictions, and that the other treatment claims should be looked at again as more research became available.<sup>12</sup>

When one looks back at specific neurotransmitter systems that are influenced and possibly rebalanced by CES one finds himself confronted directly with the body's neurohormonal stress system.<sup>13</sup>

Stress is caused by a person entering a dangerous fight-or-flight situation, and is relieved when the person is no longer in that situation. To operate effectively in such a situation, the body has to dramatically shift its neurohormonal balance out of its normal homeostasis. Stress, in that situation, is very healthy and can even be life saving, such as when a person runs out of the path of an automobile that is swerving toward him out of control, or jumps away from a snake, poised to strike, suddenly encountered on a trail in the woods.

Chronic stress, however, is a different matter and occurs when a person is living in a threatening situation he can not escape... a job, an unfortunate relationship, driving

daily in dangerous commuter traffic, watching the evening news on T.V., with every “Oh, my god,” story the news producer can find to put on (“if it bleeds, it leads”<sup>14</sup>). When under chronic stress, the body’s neurohormonal system does not come back into its normal homeostatic balance, and the resulting imbalance is said to cause up to 90% of the physical illnesses brought to the attention of physicians.

Major symptoms of a system under chronic stress number among them, insomnia, depression, anxiety, posttraumatic stress disorder, various compulsive behavior disorders, not the least of which are the various addictions in which the person uses various drugs (or medications) in an effort to alter the neurohormonal system back to a more acceptable level. Physical problems also increase, such as heart attacks, strokes, diabetes, cancer, obesity, and infections such as colds and flue, among any number of others.<sup>15-16</sup>

And where does CES treatment interface with this syndrome? From the earlier animal and later human research,<sup>17</sup> CES can best be described as an adaptogen, in which CES acts to increase the body’s resistance to adverse influences by reestablishing the homeostatic balance between the body’s various neurotransmitters that have been thrown out of balance by chronic stress. In basically rebalancing the physiological system, CES influences a wide range of physical, chemical and biochemical factors that have a normalizing effect on the body. CES, then, acts to alleviate stress, and in the process improve all kinds of conditions that have been generated by that stress.<sup>18</sup>

For example in 18 studies of insomnia, the average improvement was 62%. In a similar number of depression studies, the average improvement was 47%, while in 38 studies of anxiety, the average improvement was 58%. Those were the average improvement scores. In 31 double blind studies of various psychological problems, while the average improvement was found to be 56%, the range of improvement went from a low of 23% to a high of 91% among study group patients, a treatment effect never seen in pharmaceutical treatment of those types of disorders.

Highly positive treatment effects have also been found in other areas of dysfunction, such as in persons recovering from the effects of addiction, in children and adults suffering from Attention Deficit Disorder, in persons suffering from stress related memory loss, and in patients suffering from tension headaches and other types of stress

related pain syndromes. And more importantly, no significant negative side effect has ever been reported in more than 47 years of CES research and treatment in the U.S

More recently, following the Vietnam War the Post Traumatic Stress Disorder or PTSD is being given much attention. During World Wars I and II, the disorder was known as shell shock and thought to be caused by the immediate stress of battle. The cure, at the time, was to let the men lie quietly in or just outside the medical tent away from the battle area, and rest until their nerves settled down.

Once the syndrome was described, it was discovered that perhaps 25% or more of persons who have never been in the military have experienced PTSD. It has been precipitated by such things as child abuse or other childhood trauma such as emotional abandonment by parents or parental surrogates. In adults it has been precipitated by serious car accidents, major surgery, rapes, muggings, and in general by any other event in which the person felt helpless during an event he/she perceived as life threatening. Nine times more females than males are now known to experience PTSD, and up to 75% of persons suffering from fibromyalgia have PTSD either currently or in their background.

It is now known that PTSD represents a basic split off of parts of the brain in which the emotional trauma was recorded, so that the waking brain remains unaware of it. The problem is that the part of the brain storing the memory often reactivates during sleep and the event is recalled in very stressful nightmares. Also, during the day, any number of small stimuli that occur can reactivate that section of the brain, and a flashback occurs. Accompanying a nightmare or flashback, the entire sympathetic nervous system is called into play and the resulting stress, both physical and emotional can be overwhelming.

Because so many things can trigger a flashback, the person slowly but surely begins to close off ever more areas of his past experience in order to not provoke an episode. The brain actually becomes phobic of those activities that can act as triggers, and closes them off from its daily awareness. The person, as a result, remains in hyper aroused alert status, with an ever narrower life view and experience. To those looking on, the person become quieter, less sociable, and tends to limit activities in all areas of his/her life more and more.

CES treatment in PTSD should have a pronounced effect in that PTSD symptoms always increase when the person is under stress of any kind. Also, the research with CES in phobic patients indicates that phobic fear can not be experienced while CES treatment is in progress, and at least for a time thereafter. It is the panic felt by patients when the phobic areas are roused, with the accompanying uncontrolled system-wide sympathetic physiological arousal, that gives them their greatest fear and dread. To have CES available during those times of panic should be very immediately helpful, and contribute markedly to a longer term cure as those feelings of helplessness dissipate or habituate via the continuing use of CES.

For this reason, it has been suggested that the use of CES during desensitization therapy, a therapy found very effective in treating PTSD, should allow desensitization therapy proceed at a much more rapid rate, and possibly be much more effective if it reduced or eliminated the fear while the desensitization was in process..

If nothing more, CES should reduce or eliminate many phobic areas within the personality, allowing the person to come down from his hyper aroused state and begin interacting in more areas of his life's normal experience once again. That would be a type of desensitization therapy process on its own.

Clinical experience has shown that PTSD patients initially never go out without their CES device handy for use at a moment's notice. The presence of the device gives them a needed feeling of security they can not get in any other way.

How much treatment is required to produce these effects with CES? Patients respond to differing amounts of CES treatment, depending on which of their neurohormonal systems CES is intended to rebalance. While effects begin to be felt from the first treatment, almost all patients are expected to come back within normal homeostatic limits with 30 minutes to 1 hour of treatments every day for 14 to 21 days, depending on the availability of any required neurohormonal precursors in their diet, their level of activity and so on.

References:

1. Jarzembski, W.B., S.J. Larson, and A. Sances Jr. (1970) "Evaluation of specific cerebral impedance and cerebral current density." *Annals of the New York Academy of Sciences*, 170:476-490.
2. Kennerly, Richard C. (2006) changes in quantitative EEG and low resolution tomography following cranial electrotherapy stimulation. Doctoral Dissertation, University of North Texas, Denton, Texas.
3. Siegesmund, K.A., A. Sances Jr., and S.J. Larson (1967) "The effects of electrical currents on synaptic vesicles in monkey cortex." In Wageneder, F.M. and St. Shuy (Eds.) Electrotherapeutic Sleep and electroanaesthesia. International Congress Series No. 136. New York: Excerpta Medica Foundation, pp 31-33.
4. Dougherty, P.M., W.Q. Hong, L.A. Faillace, N. Dafny (1990) "Trans-cranial electrical stimulation attenuates abrupt morphine withdrawal in rats assayed by remote computerized quantification of multiple motor behavior indices." *European Journal of Pharmacology* 175(2):187-95.
5. Dougherty, P.M., N. Dafny (1989) "Trans-cranial electrical stimulation attenuates the severity of naloxone-precipitated morphine withdrawal in rats." *Life Sciences*. 44(26):2051-6.
6. Pozos, R.S., L.E. Strack, R.K. White, and A.W. Richardson (1971) "Electrosleep versus electroconvulsive therapy." In Reynolds, D.V. and A.E. Sjöberg, (Eds) Neuroelectric Research. Springfield:Charles Thomas pp221-225.
7. Gold, M.S., A.L.C. Pottash, H. Sternback, J. Barbaban, and W. Annitto (1982) "Anti-withdrawal effects of Alpha Methyl Dopa and Cranial Electrotherapy." Paper presented at The Society of Neuroscience, 12<sup>th</sup> Annual Meeting, October
8. Gomez, E. and A.R. Mikhail (1978) "Treatment of methadone withdrawal with cerebral electrotherapy (electrosleep)" *British Journal of Psychiatry*. 134:111-113.
9. Smith, R.B. and L. O'Neill (1975) "Electrosleep in the management of alcoholism." *Biological Psychiatry*. 10(6):675-680.
10. Patterson, M.A., J. Firth, and R. Gardiner (1984) "Treatment of drug, alcohol and nicotine addiction by neuroelectric therapy: Analysis of results over 7 years." *Journal of Bioelectricity*. 3(1,2):193-221.
11. Goozner, M. (2004) The \$800 million pill; The truth behind the cost of new drugs. Berkley: University of California Press.
12. Federal Register, 1978.
13. Selye, H. (1978) The stress of life. New York: McGraw-Hill.
14. Kitty, A. (2005) Don't believe it! How lies become news. New York: Disinformation.
15. Black, P.H. (2006) "The inflammatory consequences of psychologic stress: Relationship to insulin resistance, obesity, atherosclerosis and diabetes mellitus, type II." *Medical Hypotheses* 67(4):879-891.
16. Ware, W.R. (2008) "Psychological stress, insulin resistance, inflammation and the assessment of heart disease risk. Time for a paradigm shift?" *Medical Hypotheses* 71(1):45-52.
17. Shealy, C.N., R.K. Cady, R.G. Wilkie, R. Cox, S. Liss, and W. Clossen (1989) "Depression: a diagnostic, neurochemicals profile and therapy with cranial electrical stimulation (CES)." *Journal of Neurological and Orthopaedic Medicine and Surgery*. 10(4):319-321.
18. Smith, R.B. (2007) Cranial Electrotherapy Stimulation; It's first fifty years, plus three: A monograph. Mustang, OK: Tate Publishing. Pp 83-87.

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