Case Report

A PATIENT WITH TRAUMATIC BRAIN INJURY AND FULL BODY REFLEX SYMPATHETIC DYSTROPHY TREATED WITH CRANIAL ELECTROTHERAPY STIMULATION

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Abstract. *A 60-year-old male with an intracranial traumatic brain injury (TBI) and full body reflex sympathetic dystrophy (RSD) was treated 20 minutes daily with Alpha-Stim cranial electrotherapy stimulation (CES). The treatment provided satisfactory pain relief for the patient, allowing him to complete many tasks which were formally undoable. Post-treatment, the patient was able to enjoy a relatively higher quality of life than he was able to have with drug treatment alone.*

Descriptors. *cranial electrotherapy stimulation (CES), reflex sympathetic dystrophy (RSD), traumatic brain injury (TBI)*

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INTRODUCTION

In 1997, the Second World Brain Injury Congress in Seville, Spain, estimated the annual incidence of traumatic brain injury (TBI) to be 150 cases per 100,000 persons, with 10% of the incidences classified as severe (1). Most of these injuries related to automobile accidents, work involved incidences, violence, and injuries from war (2).

Reflex sympathetic dystrophy (RSD), a not infrequent consequence of TBI, is an idiopathic syndrome that involves severe pain, hyperesthesia, vasomotor instability, allodynia, dystrophic skin changes, and edema. It usually develops within one month after trauma, even if the trauma is mild. RSD is not limited to peripheral nerve routes and is often disproportionate to the initiating events. It is usually localized to the distal aspect of a single extremity, but it may spread to other extremities.

Although the exact incidence of RSD has not definitively been established, it has been reported to have a 2-5% incidence following peripheral nerve injury, 12-21% following stroke, 0.2-11% following Colles' fracture, 1-20% following coronary artery disease, and 0.05% from general trauma (3). It is bilateral in 18-50% of patients and has a relatively even distribution among all age groups, with the youngest child diagnosed at three years of age. The male to female ratio is estimated to be between 1:1 and 1:3.

The exact etiology of RSD following TBI is uncertain. Successful sympathectomy does not always provide pain relief, and excision of the injured region usually fails to relieve pain. Suffice it to say, better means to combat TBI with RSD are necessary. The purpose of the present report is to describe a severe case of TBI with RSD. The patient responded extremely well, experiencing a better quality of life, following use of cranial electrotherapy stimulation (CES).

Elliott J. Alpher, DDS, is Director of the Metropolitan Area Craniofacial Pain Center, in Washington DC. Daniel L. Kirsch, PhD, is a pain and stress management consultant, Mineral Wells, Texas. Address reprint requests to: Dr. Daniel L. Kirsch, PhD, 2201 Garrett Morris Parkway, Mineral Wells, TX 76067

CASE REPORT

Patient history. Following an automobile accident in 1986, WHH was diagnosed as having an intracranial closed-TBI with post-concussion syndrome, including 95% loss of vision in his left eye due to damage to the greater ocular nerve. He was 45% whole-body permanently impaired according to a board-certified neurologist in Bethesda, Maryland. After three years at the National Rehabilitation Hospital in Washington, DC, he returned with some limitations to sedentary work. WHH endured multiple ongoing problems including headaches, memory loss, neck pain, difficulty speaking and increased stuttering, difficulty thinking and perceiving, dizziness, and progressive decreases in concentration and attention. He also experienced pain and numbness in his left arm. Personality testing revealed evidence of mental confusion, unusual thinking, impulsivity, and insecurity.

Heavily medicated with 8-10 drugs, WHH frequently complained of a restricted life schedule. He would pause and wonder what he was doing, and he had several general seizures in which he would awake on the floor and not know how he had gotten there. He was unable to maintain any social relationships and barely found the strength to go to the National Institutes of Health Library to study TBI. His time was taken up primarily with rehabilitation, medical appointments, and medical consultations. He complained of severe pain focused around the greater occipital area and around the cervical area (C2, C3, and C4).

WHH married in June of 1991 and enjoyed about six months of normal married life until December 16, 1991, when he suffered a fractured left foot from an accident. He underwent surgery for the fracture at George Washington University Hospital where screws were placed in the first tarsometatarsal joint. Subsequently, the leg became swollen, erythematous, and he developed a burning pain in the entire lower extremity to the hip. A second surgery was performed to remove the screws. The neurologist opined that the screw impinged the nerve in the first web space or the onset of the injury significantly traumatized the sensory nerves resulting in RSD.

In 1992, a right side hernia repair exacerbated the RSD. A repeat hernia repair was necessary in 1996. The RSD expanded on his right side. Since then, the RSD has involved his entire right leg as well as his groin, and continued in the left leg and left groin as well. The RSD became classified as Stage III, acutely progressive from above the waistline down the length of his body. Eventually, the RSD spread and became global (generalized and centralized) to his entire body causing high levels of pain in his hands, torso, feet, face, and legs. He developed limb swelling, discoloration, and vasomotor instability as well as sympathetically induced pain throughout his body. He had pain to light touch (allodynia), burning pain, and sensations of primary and secondary hyperalgesia. Atrophy had reduced muscle leg strength 45% in the left leg, and 50% in the right leg; and arm and hand muscles by 50% left and 55% right. He was diagnosed with depression and placed under the care of a psychiatrist.

On June 4, 1997, the first tarsometatarsal joint of his left foot was reinjured when a scooter was dropped on the joint. As a result, he was declared 100% permanently disabled by an orthopedic surgeon in Washington, DC, and a neurologist in Bethesda, Maryland. The disability was confirmed by the Social Security Administration.

Over the years, WHH has been prescribed numerous medications including Prozac 20 mg four times daily, Catapres 20 mg daily, Effexor 100 mg morning and at bedtime, Levo-Dromoran 1 mg twice daily, Balofen 10 mg split AM and PM, Risperdal 7.5 mg at bedtime, Kolopin 0.5 mg three to four times per day as needed, C-Dextromethorphin 60 mg three times daily, and Fentanyl patches for four years. None of the medications reduced his whole body chronic intense critical pain and burning, nor did they relieve his difficulty in sleeping. Standard milliampere transcutaneous electrical nerve stimulation (TENS) did not help. The patient even claimed these treatments made him worse, and he became concerned about the short- and long-term side effects various drugs had on his ability to function. He occasionally received acupuncture, which provided some relief.

CES TREATMENT

WWH was eventually referred from the National Rehabilitation Hospital in Bethesda to the Metropolitan Area Craniofacial Pain Center in Washington, DC, for dentistry and treatment of temporomandibular joint disorder (TMD). In order to overcome anxiety from the dental procedures, he received cranial electrotherapy stimulation (CES). Alpha-Stim 100 (Electromedical Products International, Inc., Mineral Wells, Texas) CES had been shown to relax patients for dental treatment in a manner similar to nitrous oxide and was routinely employed at the Metropolitan Area Craniofacial Pain Center to control pain and anxiety. The Alpha-Stim was used at settings of 0.5 Hz at 100 μA for 20 minutes prior to administration of anesthetics. Post CES, WHH exhibited marked relaxation, with a reduced anxiety level and a significantly enhanced pain threshold. Based on these positive results, he was prescribed daily 20-minute treatments on the Alpha-Stim 100. After the initiation of CES treatment, the patient returned to work, and improved his family and social life. WHH estimated 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% from insomnia. Importantly, use of Alpha-Stim CES was associated with elimination of need for morphine and Fentanyl patches. His other medications were reduced to: Prozac 10 mg daily, Catapres 0.1 mg twice daily, Effexor 50 mg AM and 25 mg PM, Levo-Dromoran 1 mg twice daily, Restoril 7.5 mg at bedtime, Kolopin as needed, and Gabapentin (Neurontin®) 400 mg as needed.

DISCUSSION

CES was employed in WHH to relieve tension prior to dental procedures. Fortuitously, CES produced outstanding benefits on this patient's RSD associated with his previous TBI. Cra-

nial electrotherapy stimulation has been shown to help both closed-brain injuries (4,5) and reflex sympathetic dystrophy. Accordingly, there was ample reason to attempt this therapeutic modality on the patient, since WHH was not willing to accept medically forced retirement. While medications, physical therapy, and TENS failed to help him significantly, CES improved his condition sufficiently to WHH to return to work and maintain an endurable quality of life. Because Alpha-Stim CES has been shown to induce spectral smoothing in pain patients' electroencephalograms and has a parasympathetic-like anxiolytic effects, it seems a reasonable and safe treatment for both TBI and RSD (6-8).

For WHH, a single CES treatment lasts for 6-8 hours, allowing him to get through the day. Although the pain gradually returns, it never soon reaches the level experienced prior to CES treatment. CES reduces his pain to a point where he can perform his daily exercise routine. He is also able to rest better at night which he credits as creating a "positive emotional and physical self-environment." WHH now feels more rested in the morning and is able to work 30-40 hours per week, up from a maximum of 15 hours prior to CES.

CES is a simple treatment that can be easily administered by a physician or patient. Its use for therapeutic purposes is not new. At least two millennium ago, physicians used electric eels to relieve pain; and experimentation with low intensity electrical stimulation of the brain was first reported by Drs. Leduc and Rouxeau of France in 1902. The electrical current is characteristically applied by clip electrodes that attach on ear lobes or by stethoscope-type electrodes placed behind the ears. CES devices are generally limited to less than one milliampere (mA) of current.

Anxiolytic, anti-depressive, and pain relieving effects of CES are usually experienced during a treatment, but these effects may be seen hours later, or as late as one day after treatment. In some people, it may require a series of 5-10 daily treatments before a response is seen. One 20-minute session is usually all that is needed to control anxiety, depression, and pain effectively for at least a day or two, and the effects appear to be cumulative. CES may also be used as an adjunct to pharmacotherapy and other methods of treatment.

At present, there are over 100 research studies on CES in humans and 20 experimental animal studies (9). No significant serious adverse side effects have been reported. Occasional self-limiting headache, discomfort, or skin irritation under the electrodes, or light-headedness may occur. Patients with a history of vertigo may experience dizziness for hours or days after treatment.

The current state of knowledge of bioelectrical systems is limited. Currently, there is no uniform agreement on the mechanisms of action of CES. It is generally believed that the effects are primarily mediated through a direct action on the brain at the limbic system, the hypothalamus, and/or reticular activating system (10-12). The primary role of the reticular activating system is the regulation of electrocortical activity. Electrical stimulation of the periaqueductal gray matter has been shown to activate descending inhibitory pathways from the medial brainstem to the dorsal horn of the spinal cord, in a manner similar to β-endorphins (13-15). Cortical inhibition is a factor in the Melzack-Wall Gate Control theory (16). It is possible that CES may produce its effects through parasympathetic autonomic nervous system dominance via stimulation of the vagus nerve (CN X) (17). This may explain its efficacy in RSD, as the vagus is one of the most significant parasympathetic nerves, thus offsetting the sympathetic dominance of RSD. Other cranial nerves such as the trigeminal (CN V), facial (CN VII), and glossopharyngeal (CN IX), may also be involved (18). Electrocortical activity produced by stimulation of the trigeminal nerve has been implicated in the function of the limbic region of the midbrain affecting emotions (19). Substance P and enkephalin have been found in the trigeminal nucleus, and are postulated to be involved in limbic emotional brain factors (20). The auditory-vertigo nerve (CN VIII) must also be affected by CES, accounting for the dizziness experienced when the current is too high, and the positive effects of treating tinnitus and sensorineural hearing loss (21,22).

Animal studies of CES using monkeys reveal that 42-46% of the total applied current enters the brain, with the highest concentration in the thalamic region (23). Rat studies showed as much as a threefold increase in ß-endorphin concentration after just one CES treatment (24). Mongrel dog research suggests that CES releases dopamine in the basal ganglia, and that overall physiologic effect appears to be anticholinergic and catecholamine-like in action (25). The size, location, and distribution of synaptic vesicles were all within normal limits after a serious of ten, one-hour treatments in Rhesus monkeys (26).

Reviews of CES by Kirsch (9) including an annotated bibliography of 106 human studies involving 5,439 subjects, of which 4,058 received CES and the remainder served as sham-treated or controls revealed significant changes associated with anxiolytic relaxation responses, such as altered (lower levels) electromylograms (6,11,27,28), slowing on electroencephalograms (29-33), increased peripheral temperature (an indicator of vasodilation) (6,10), reductions in maximal acid output (34), and in blood pressure, pulse, respiration, and heart rate (6,18). Heffernan found CES to normalize electroencephalograms and produce spectral smoothing (7,8).

Smith and his cohorts conducted a double-blind study of 21 closed-head injured (CHI) patients with an average age of 30 and time since injury ranging from six months to 32 years (mean = 11 years) (4). They were randomly assigned to CES treatment (N = 10), sham treatment (N = 5) or control "wait-in-line" (N = 6) groups. The CES and sham groups had 12 treatments, given daily over a period of three weeks. The subjects were pre- and post-tested on the Profile of Mood States (POMS). The CES treated subjects, but not the sham treated subjects or controls, improved significantly on every POMS subscale: tension/anxiety, depression/dejection, anger/hostility, fatigue/inertia, confusion/bewilderment, and total mood disturbance. One patient on sham CES was seen to have a seizure. No negative effects from CES treatments was seen. The authors concluded that therapists of CHI patients may well try adding CES therapy, a prescription, but non-pharmacologic treatment, to the treatment of this currently heavily

medicated patient population.

Wilson and Childs reported on the CES treatment of four patients with measurable attention-to-task deficit (5). Two had severe pain problems (27 year old female and 30 year old male) but no brain injury, while two had suffered from post brain trauma (29 year old male, 25 year old female). One of the pain patients served as placebo control (the 30 year old male) for the other three, each of whom served as his or her own control. CES was given for 50 minutes per day, five days a week, for three weeks. Patients were pre- and post-tested on standardized cognitive measures (Trail Making Test, Digit Symbol Test, Porteus Mazes, Consonant Trigrams Test, Rey Auditory-Verbal Learning Test, Paced Serial Arithmetic Test) before and following CES, and again three weeks later. Patients were also tested on the Profile of Mood States. The results among the CES treated patients showed striking and significant improvement in the post treatment scores and in the associated extent of the neurological deficit. It was concluded that CES is an effective non-drug alternative in a cognitive rehabilitation model for treating attention-to-task deficit. No adverse side effects were reported.

CONCLUSION

Crainial electrotherapy stimulation (CES) proved to be an effective treatment for symptoms associated with intracranial traumatic brain injury (TBI) and full body reflex sympathetic dystrophy (RSD) in a 60-year-old male. The treatment provided satisfactory pain relief for the patient, allowing him to complete many tasks which were formally undoable. Post-treatment, the patient was able to enjoy a relatively higher quality of life than he was able to have with drug treatment alone. CES is worthy of therapeutic consideration in such cases; it appears to be a very safe alternative to pharmacotherapy and exhibits minimal side effects.

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